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Thank you again for supporting the OVMA Conference and Trade Show, and the continuing education of veterinary teams.

Matthew Richardson, DVM
2023 Conference Chair
Ontario Veterinary Medical Association
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VetDentEdu, Inc.

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**J. CATHERINE SCOTT-MONCRIEFF, VET MB, MA, MS, DACVIM-SAIM, DECVIM-CA**  
Professor, Department Head, Purdue University

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**DENNILYN PARKER, BSC, DVM, M.VET.SC, DABVP(AVIAN)**  
Associate Professor – Zoo, Exotic and Wildlife Medicine, Western College of Veterinary Medicine, University of Saskatchewan

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Small Animal Program
WHAT IS PERIODONTAL DISEASE, REALLY?
Fraser A. Hale, DVM, Dipl AVDC

Introduction:
Given that the human dental profession has spawned a specialist collage based solely on the study and treatment of periodontal disease, we must accept that this 50-minute presentation is just dipping a toe in a small puddle at the edge of this vast ocean of knowledge. And space limitations allow me to share only a bit with you here. I have other resources available through http://www.toothvet.ca/Old%20CUSP%20Articles.htm such as…
- the impact of crowding on periodontal disease - http://www.toothvet.ca/PDFfiles/Crowding.pdf
And more links will be found below.

The Short(ish) Answer:
I bet you are expecting me to say that periodontal disease is bacterial infection of the tissues around the tooth. Well, that is not only a gross oversimplification, it is also probably patently incorrect.

A more accurate statement (though still a serious over-simplification) would be that periodontal disease is the up-regulation of inflammation leading to progressive destruction of the tissues of the periodontium. The inducement is a dysbiosis between the various and complex oral microbiomes and the host’s defense mechanisms.

For an excellent review of some of the new findings and thinking on this you absolutely must find time to review this excellent paper – The Oral Microbiome in Dogs and Cats: Dysbiosis and the Utility of Antimicrobial Therapy in the Treatment of Periodontal Disease by Davis and Weese (Vet Clinics: Sap. 2022; 52(1). Among the new findings are that there are far more types and species of micro-organism within each biome than previously identified. While there are many (hundreds if not thousands) of genetically-distinct bacterial species, there are also diverse populations of viruses, molds/fungi/yeasts, protozoa and more and we are only just starting to contemplate how these other micro-organisms might factor into the equations.

And the oral cavity has a number of distinct biomes. The biome on the tooth surface differs from that in the gingival sulcus, which is different from the one on the tongue, on the palate, on the tonsils and then the one within a periodontal pocket (a site of active disease) is different again.

The biomes on the dental surfaces live within a biofilm known as dental plaque. Biofilms are highly complex societies of micro-organisms living in a protective matrix of polymers of bacterial and salivary origin. It has been shown that this slime increases resistance to antimicrobials by as much as 1500 times. Also, it has been shown that bacteria in plaque express very different physiologies/behaviours than what those same organisms would display as a monoculture in/on a culture medium. And, the vast majority of bacterial species in the biofilm have never been grown/cultured in the lab but have been discovered using newer DNA probe technologies. The thinking is that they have very specific growth requirements that are only met within the plaque film and which have never been replicated in the lab. For these and other reasons, culture and sensitivity testing is almost never going to provide clinically relevant information. It will only tell you which of all the hundreds of bugs in the sample grow best in the artificial environment of the laboratory and which drugs they are sensitive to in that very artificial situation.

And then there is the host – the animal who owns the mouth in which all of these critters reside. Some animals seem able to live in relative harmony with their oral flora and others most definitely cannot. What are the local and systemic immune-system factors that determine and...
individuals susceptibility/resistance to periodontal disease? That is an issue with far more questions than answers at this time.

By the way, the four periodontal tissues are the gingiva, the alveolar bone, the periodontal ligament and the cementum covering the tooth root (a paper on periodontal anatomy - http://www.toothvet.ca/PDFfiles/PerioAnat&Physio.pdf).

Here is my 26-minute video on periodontal anatomy and progression of disease – https://youtu.be/uRExyTGheAM.

Some Other Things Periodontal Disease Is and Is Not:

**Periodontal disease IS the most common health concern in domestic dogs and cats:**

You will see various numbers thrown about (usually in product promotional materials) indicating the incidence of periodontal disease and in many cases there are no valid studies to back the numbers. That said, it is widely acknowledged that most adult pets have at least some degree of periodontal disease making it the most commonly diagnosed disease entity in small animal general practice. And chances are, more of your patients have significant periodontal disease that you suspect because…Meta-studies based on insurance claims may not reflect this but that is because many insurance policies do not cover periodontal care and so no claims are generated.

**Periodontal disease IS a (largely) hidden problem:**

It would be wonderful if we could accurately assess the periodontal health of a patient, or a tooth based on visual inspection of a conscious patient. It is most definitely not possible to do this except in extreme disease. The reason is that periodontal disease is hidden disease. That is to say, it most commonly is going on below the gum line, hidden from view in the conscious patient.

The only way to assess the periodontal health of a tooth (and we must assess the health of every tooth on its own merits) is to examine the mouth with the patient under general anesthetic. Every tooth should be explored and probing depths measured at several locations around each tooth. And, whole-mouth intra-oral dental radiographs must be obtained.

Having raised the subject of probing depths, let’s dive in to that deeper. It has been commonly taught that the “normal” probing depth for dogs is 1-3 mms. This is like saying that the “normal” body weight for a dog is 15-30 kg. Our canine patients come in such wide range of sizes and so do their teeth. For the canine tooth of a 45 kg Mastiff, I would not worry about a 4 mm probing depth if all other parameters were fine. For the upper second molar in a 1.5 kg Chihuahua, a probing depth of 2 mm would represent 100% attachment loss (a pocket involving the entire length of the root). I discuss this more in this paper - http://www.toothvet.ca/PDFfiles/probing.pdf.

I have papers in these proceedings on Intra-oral dental radiology, so refer to that for more on the importance of this diagnostic tool.

**Periodontal disease IS caused by plaque:**

Dental plaque is a biofilm as mentioned above. In animals susceptible to gingivitis/periodontitis it is dental plaque that is the trigger that starts the inflammatory response that leads to eventual tissue destruction. Plaque is the enemy and the body wages a destructive war against it. And it is the plaque at the gingival margin and below the gum line that causes the problems. Plaque on the crowns, away from the gingiva really is not a player in this at all.
**Periodontal disease is NOT caused by calculus on the crowns:**

Calculus is just mineral deposits, mostly calcium carbonate (same stuff that eggshells are made of). If you were to sterilize calculus, you could almost use it as a bone grafting substrate. Calculus is an oral-health liability because it is porous and harbours dental plaque (the real culprit). But calculus itself is not pathogenic. And, as mentioned above, periodontal disease is going on below the gum line, not on the crowns of the teeth. Therefore, the presence or absence of calculus on the crowns of the teeth is a terrible predictor or periodontal health. There is almost no reliable connection between the amount of calculus on the crowns and the amount of periodontal disease. I routinely see teeth with no coronal calculus that have end-stage periodontal disease. Occasionally I will see a patient with loads of coronal calculus and nothing more than mild, reversible gingivitis. So, if you are just using the amount of coronal calculus visible on conscious examination to determine a patient’s periodontal status, you will likely be wrong more often as you are right.

**Periodontal disease IS largely preventable:**

For the majority of the pet population, periodontal disease is quite preventable, but it takes work, on our part and on the owner’s. It involves addressing developmental/anatomic oral liabilities at 6-7 months of age (see my paper on pro-active dental care in these proceedings), establishing a safe, effective daily home plaque-control program [http://www.toothvet.ca/PDFfiles/HomeCarePack.pdf](http://www.toothvet.ca/PDFfiles/HomeCarePack.pdf) and a schedule of annual professional care under general anesthetic. This is similar to what we do for our own oral health so it should really not surprise clients when they are thus informed.

**Periodontal disease IS dramatically impacted by oral anatomy such as dental crowding:**

It has been shows that the smaller the dog, the larger the teeth (relative to the size of the mouth). So small dogs tend to have significant dental crowding. And all brachycephalic dogs are going to have crowding issues because of their craniofacial deformities. Referring back to the paper and video on periodontal anatomy, you know that each tooth should have a collar gingival attachment around its entire circumference as the physical barrier keeping the oral microbiota away from the three other periodontal tissues (alveolar bone, periodontal ligament, cementum). When teeth are crowded, there is typically a discontinuity in that collar of gingival attachment allowing food, fur, debris and microbiota to get up into the periodontal space. More on that can be found in this paper - [http://www.toothvet.ca/PDFfiles/Crowding.pdf](http://www.toothvet.ca/PDFfiles/Crowding.pdf). And there is a bit more in these proceedings in my paper on pro-active dental care for micro-dogs and brachycephalic breed.

Management of this issue involves early (like at 6 to 7 months of age) extraction of smaller, less important teeth to improve the prognosis for larger, more important teeth.

**Periodontal disease is NOT an antibiotic-deficiency:**

My session in the Pearls section of these proceedings outlines how antibiotics are not only largely useless in the management of periodontal disease but in many cases are worse than useless. I treat horrific dental disease day and day out. I use antibiotics (as a single, IV dose at induction) maybe 4 to 6 times a year – that is all.

**Periodontal disease is NOT treatable with preventative measures:**

We must accept that you cannot prevent something that has already happened [http://www.toothvet.ca/PDFfiles/PerioAgain.pdf](http://www.toothvet.ca/PDFfiles/PerioAgain.pdf). Similarly, you cannot treat a disease with preventative measure. When do you vaccinate a puppy vs parvo virus? When it is healthy or when it shows up with bloody diarrhea? It is really common to see histories that indicate an owner was advised to start a dental diet or to start a tooth-brushing program AFTER it was
noted that the patient had gingivitis. Too late. If there are visible signs of disease starting a plaque-control program will provide no benefit and if it is a mechanical strategy (diet, chews, brushing…) it could be worse than useless. I explain the reasons in this video - https://youtu.be/3QkRbuPxB7M.

And as I have already mentioned, periodontal disease is a largely hidden problem, so even if the mouth looks great on conscious examination, you cannot be sure there is no hidden/painful pathology somewhere. Therefore, you really should not recommend any mechanical home dental care for any patient without first doing a detailed dental examination under general anesthetic and including whole-mouth intra-oral dental radiographs.

**Periodontal disease IS a managed issued, NOT a curable one (expect by extraction):**

When dealing with periodontal disease you (and the owners) should be thinking of management, not cure. For patients predisposed to or afflicted with periodontal disease, the only way to put an end to it for certain is to extract all of the teeth, and this is often the best option for the patient and their owners - http://www.toothvet.ca/PDFfiles/CleaningHouse.pdf. Short of that, all periodontal patients will require ongoing home and professional care for as long as they have teeth.

**Periodontal disease IS just one of many painful dental conditions:**

There is a lot of messaging that suggests that proper use of diets/chews/brushing…is all that is needed to maintain good oral health. Wrong. Managing/preventing periodontal disease does nothing to prevent tooth resorption in either dogs or cats (http://www.toothvet.ca/PDFfiles/Tooth_resorption_in_cats.pdf. http://www.toothvet.ca/PDFfiles/RLs_in_Dogs.pdf). It does nothing to prevent endodontic disease (https://youtu.be/NgR2aaoC2ME. http://www.toothvet.ca/PDFfiles/endo.pdf). It does nothing to address malocclusions with traumatic tooth-to-tooth or tooth-to-soft tissue contacts (http://www.toothvet.ca/PDFfiles/malocclusions.pdf). It does nothing to prevent dentigerous cyst formation (http://www.toothvet.ca/PDFfiles/dentigerouscysts.pdf) or the formation of other odontogenic cysts. It likely would have very little effect on preventing oral neoplasms. So even if you patient appears to have no periodontal disease, it would still be prudent to perform periodic detailed assessments under general anesthetic to determine if there are any other issues in need of attention.
PRO-ACTIVE DENTAL CARE FOR MICRO-DOGS AND BRACHYCEPHALIC BREEDS

Fraser A. Hale, DVM, DiplAVDC

Many breeds are intentionally bred to have serious cranio-facial deformities or to be so miniaturized that the proportions in their mouths simply do not work. These imposed structural features have a serious negative impact on oral health and therefore quality of life. However, with early, pro-active intervention, we can make the best of some very bad designs and significantly improve the oral health of these innocent victims of ‘fashion’. Of course, any breed of dog or cat may have an accidental developmental abnormality and these require attention as well. Therefore, I recommend that ALL dogs and cats have a thorough oral inventory done, with whole-mouth intra-oral dental radiographs, by seven months (yes, MONTHS, not years) of age to find and address any developmental and anatomic oral liabilities.

Space limitations do not allow for a complete, illustrated discussion of this topic but there are several resources at http://www.toothvet.ca/Old%20CUSP%20Articles.htm that add to the discussion:
- regarding brachycephalic animals in general - http://www.toothvet.ca/PDFfiles/Stop_Brachy_2.pdf

There are at least seven distinct developmental/anatomic problems that should be either ruled-out or identified and addressed in all pets but which are particularly common in micro-dogs and brachycephalic breeds. This assessment should be done at about six to seven months of age (or sooner depending on the problem) and the problems addressed proactively to optimize oral health.

The problems are:
1. persistent deciduous teeth
2. impacted/unerupted permanent teeth
3. under-erupted permanent teeth
4. missing permanent teeth
5. deformed permanent teeth
6. crowded permanent teeth
7. malocclusions.

Some animals have all of these problems, some select only a few from the menu and so the combinations and permutations are numerous. For simplicity, I will discuss each as a separate entity, though they may interact.

Persistent Deciduous Teeth

It seems paradoxical that the little mouths, that have so little room to spare, are the ones that seem most likely to hold onto the deciduous teeth as the permanent teeth erupt but this is a problem seen more in micro-dogs than in larger dogs. It can, of course, be seen in any breed.

First, it must be understood that the mechanism that triggers the absorption of the deciduous tooth roots, allowing the crowns to exfoliate, is not known. Nor is the reason for persistence of some deciduous teeth understood.
Regardless of why the deciduous teeth fail to leave on schedule, when this happens, we need to intervene. The rule is that there should never be two teeth trying to occupy the space meant for one tooth. If the adult tooth has started to show itself through the gingiva, then the deciduous tooth needs to be removed to allow room for the adult to erupt into its proper position. Failure to do this in a timely manner can result in the adult tooth not erupting fully or erupting into an undesirable position.

The persistence of this maxillary canine tooth in a 6-month-old JRT (photo below left) was forcing the permanent canine tooth to erupt too far forward, obstructing the eruption of the lower permanent canine tooth.

Sometimes the deciduous tooth is still in place and there is no permanent tooth trying to erupt because the permanent tooth is absent. I have found that deciduous teeth do not do well long-term in an adult mouth. Often one root resorbs but the other one holds on (photo above right). In those cases, there is typically periodontal infection around the resorbed root as shown above.

**Impacted/Unerupted Permanent Teeth**

When permanent teeth are developing, there is a sack of tissue that surrounds the developing crown. This “enamel organ” is composed of an inner enamel epithelium, an outer enamel epithelium and a stellate reticulum between. The inner enamel epithelium is responsible for the mineralization of the enamel that covers the crown of the tooth. Once this is finished, the stellate reticulum atrophies and the two epithelial layers collapse onto each other to form the reduced enamel epithelium. When the tooth breaks through the gingiva into the oral cavity, most of the reduced enamel epithelium is torn away and lost, though a ring of it remains around the base of the crown to form the junctional epithelial attachment of the gingiva.

When a permanent tooth fails to erupt, the reduced enamel epithelium remains intact. As the epithelial cells exfoliate into the space between the reduced enamel epithelium and the crown of the tooth, they sets up an osmotic gradient that pulls fluid into the space. As the fluid accumulates, the sack expands. This is a dentigerous cyst. Left to its own, the cyst just keeps inflating and growing, while causing the destruction of the surrounding bone. Eventually the cyst can break through the bone to cause a visible oral or facial swelling, but by this time a lot of damage has been done. The image shows a very large cyst in the right mandible of a three-year-old golden retriever that developed around an un-erupted lower 1st premolar tooth. Large cysts can form around even the smallest teeth.
To prevent dentigerous cyst formation, always radiograph areas of apparently missing teeth. If the permanent tooth is present but unerupted and is not likely to erupt, remove it surgically ASAP. For boxers, always radiograph even if it seems all the teeth are there because boxers commonly have supernumerary 1st premolars and one of the extra ones may be un-erupted.

**Under-Erupted Teeth - Pericoronitis**

- gingiva and periodontal ligament will only attach properly to bone and cementum
- the gingival should be firmly attached to the cementum in the area between the top of the bony socket and the cementoenamel junction
- the free gingival margin is normally just slightly coronal to the cementoenamel junction
- the free gingiva lies against but does not attach to the enamel resulting in a space between gingival and enamel known as the gingival sulcus. The normal depth of the gingival sulcus will depend on the size of the patient and the size of the tooth. For a canine tooth in a large dog, the gingival sulcus may be 3 or more millimeters. For the second maxillary molar in a 1.5kg Yorkie, anything over 0.5 millimeters would have me worried (http://www.toothvet.ca/PDFfiles/probing.pdf).

Now we need to consider the situation that may occur if a tooth fails to erupt fully. This is a very common problem for the canine teeth in small, brachycephalic dogs (Boston terriers, pugs, shih tzus...). Sometimes the tooth is prevented from erupting properly by a malocclusion or crowding issue. Sometimes it remains under-erupted for no apparent reason (just another cost to the animal of selective breeding based on "cute" rather than on "healthy"). Another common location for under-eruption is the mesial portion of the mandibular 1st molar tooth in micro-dogs.

When a tooth fails to erupt properly, too much of the enamel-covered crown remains below the gum line but since the gingiva and periodontal ligament do not attach to this buried enamel, there is a very deep gingival sulcus which quickly becomes contaminated with oral bacteria giving rise to chronic (hidden) inflammation around the buried crown (pericoronitis). This will then lead to periodontal disease.

If a tooth is under-erupted because its eruption path is being physically obstructed, then timely removal of the obstruction (usually selective extraction of a less important tooth to allow a more important tooth to erupt) may allow the important tooth to erupt adequately. This should to be done by six months of age. If the tooth's eruption is not being prevented by something you can remove (to encourage eruption), then options would include a crown lengthening procedure (surgical crown lengthening or orthodontic forced eruption - way beyond the scope of this paper) or extraction of the under-erupted tooth.

In the next images, we see a seriously under-erupted lower right canine tooth crowded up against the back of the 3rd incisor tooth. In the radiograph, we can see the cementoenamel junction (green line) is well below the level of the alveolar crest (blue line) and much too far below the free gingival margin (red line). The dog is also missing the adult lower 1st and 2nd premolar and has a persistent primary 2nd premolar (the rest of the mouth was similarly messed-up).
Missing Permanent Teeth

Absence of a strategically important tooth might be a concern, but there is nothing that can be done to correct the problem. On the other hand, missing some teeth can be a distinct advantage. Most little dogs and brachcephalic animals just do not have room for all their teeth, so to be missing a few leaves more room for the rest. Just be darned sure you radiograph to ensure the tooth is missing, not hiding. Then record it in the permanent record as being radiographically confirmed to be absent.

Deformed Permanent Teeth

Any tooth can be deformed and in a variety of ways, but there is one form of deformity to which micro-dogs seem particularly prone (though as with all of these issues, I have seen it in all sizes of dog). At present, we are calling this Mandibular Carnassial Tooth Malformation (MCTM) - https://pubmed.ncbi.nlm.nih.gov/31956654/.

Normally the roots of the mandibular first molar are divergent as in the radiograph below left. In the normal tooth, the only way into or out of the endodontic chamber is through the apical delta (a collection of tiny channels at the tip of the roots - http://www.toothvet.ca/PDFfiles/endo.pdf).

With MCTM, the roots are more parallel or convergent (sometimes the tips even cross over each other). More importantly, there are typically one or more openings directly into the pulp chamber located near the base of the crown of the tooth or in the furcation. This allows oral bacteria to gain access to the pulp, causing a septic pulp necrosis. The infection then oozes out through the apical deltas to cause infection in the surrounding bone. The middle radiograph is of the lower right 1st molar of a large Lab cross and the image to the right is of a tiny toy poodle. Both of these deformed teeth have serious, chronic lesions of endodontic origin.

In most of the cases I have seen either personally or reported by others, the condition is bilaterally symmetrical. Therefore, if you find it on one side expect to find it on the other and look for it. I have never seen one of these dilacerated molars that did not require extraction. So, look for them and when you find them, get them out.

Crowded Permanent Teeth

Micro-dogs have micro heads and micro mouths. Unfortunately, they usually have macro teeth. It turns out that tooth size is very resistant to breeding influences and so on a pound for pound basis, little dogs have very big teeth compared to big dogs. If all the teeth (or even most of them) develop, there is rarely room for them to all fit in the mouth properly. This can cause various occlusal problems (see next section) but here I want to discuss the periodontal ramification. Of course all brachycephalic breeds, regardless of size, have insufficient room for their maxillary teeth.

An intact collar of attached gingiva around the entire circumference of the tooth is the barrier to periodontal disease. When teeth are severely crowded, there is no room for gingiva or even alveolar bone between the teeth and so periodontal disease starts early and progresses rapidly.
One common place for this is between the maxillary molars. In micro-dogs, the roots of the second maxillary molar may only be 2 to 4 millimeters long and so a periodontal pocket of 2 millimeters constitutes 50%-100% attachment loss which is end-stage periodontal disease and indication for extraction. In a large dog, there may be nice spacing between the maxillary molars with good gingiva filling this space. Micro-dogs have the second molar jammed so tight against the back of the first molar that there is no gingiva and no bone between them. My approach is to sacrifice the second molar and suture the gingiva around the back of the first molar. This gives the larger and more important first molar a fighting chance. Failure to do this means (in most cases) early onset of periodontal disease in an area that is virtually impossible to reach for proper treatment and home care, so you end up losing both teeth (or more).

Another serious crowding concern is between the maxillary third and fourth premolars. Recall that the upper fourth premolar has three roots – one distal, one mesiobuccal and one mesiopalatal. Where two roots come together to join the crown is a furcation, so there is a mesial furcation between the mesiobuccal and mesiopalatal roots of the fourth upper premolar.

Recall also that gingiva and periodontal ligament will attach only to cementum and alveolar bone – they do not attach to enamel.

If the third and fourth premolars are crowded together, the distal shoulder of the crown of the third can get trapped in the mesial furcation of the fourth. This traps a lot of the enamel-covered crown below the gum line and in the periodontal space of the mesial roots of the important fourth premolar tooth.

The third common place for me to selectively extract to alleviate crowding and to improve the periodontal prognosis for the remaining teeth is the mandibular third incisors. Crowding between the corner incisor and the canine tooth puts both at risk. Removal of the incisor improves the outlook for the much more important canine tooth.

**Malocclusions**

Brachycephalic breeds are designed to have a Class III malocclusion (upper jaw too short compared to the lower) Many non-brachy micro dogs also have malocclusions. These are serious craniofacial deformities with negative consequences for the animal. Basically, any malocclusion that causes abnormal tooth-to-tooth or tooth-to-soft tissue contact needs to be alleviated. In a dog’s mouth, the teeth are not supposed to touch soft tissue at all, so any tooth-to-soft tissue contact is abnormal. The only place there is supposed to be any tooth-to-tooth contact is between the upper and lower molars. Any other tooth-to-tooth contact is abnormal.

Some malocclusions result in teeth crashing into each other leading to abrasive wear and/or traumatic pulp necrosis. Some malocclusions result in some teeth blocking the eruption path of other teeth. Some malocclusions result in teeth causing serious (and painful) trauma to the oral soft tissues.

No dog or cat needs a perfect bite but every one of them deserves a comfortable bite. If achieving this objective means selective extraction to alleviate abnormal contacts, then that is a medically appropriate approach.

**Conclusion**

This has been but an introduction. The “take home” message from this is that many dogs (and cats) have serious developmental and anatomic problems that can have a serious negative impact on oral health. These problems need to be identified and managed very early in life to prevent much pain and suffering. Then after that treatment, get the animal on a safe, effective daily home plaque control program and schedule of annual professional oral care (http://www.toothvet.ca/PDFfiles/HomeCarePack.pdf).
INTRA-ORAL DENTAL RADIOLOGY

Fraser Hale, DVM, FAVD, DiplAVDC

Due to space limitations and the very visual nature of this presentation, these notes are just an outline. I have included a number of references, many of which are links to papers on my website that are only ever just a click away. Many of the other resources (papers and videos) at http://www.toothvet.ca/Old%20CUSP%20Articles.htm, though not specifically about radiography, are richly illustrated with intraoral dental radiographs and these papers can be helpful in seeing the value of this diagnostic tool and learning more about how to interpret the images accurately.

The Importance of Doing Whole-mouth Intra-oral dental Radiographs in all Dental Patients. (a 43 minute video presentation on this is available at https://youtu.be/ZBc4bQ-HbFA)

I will start with the bold and provocative statement that if your practice has not incorporated intra-oral dental radiography into the assessment and management of every dental patient, you should cease to offer dental services at all. I say this not only for your clients’ and patients’ protection but also for yours and your practices’. For fun, contact the CVO and ask them what percentage of complaints submitted to them pertain to dental procedures or mismanagement of dental disease. The AVMA’s Professional Liability Insurance Trust newsletter from the summer of 2015 outlined a number of cases related to retained roots and iatrogenic jaw fractures. A paper by Moore and Neimeic reported that 86% of patients who had extractions without radiographs had retained roots. And there are several other references demonstrating the value of whole-mouth intra-oral dental radiographs.

Aside from the legal compulsion to obtain whole-mouth intra-oral dental radiographs, it is just the right thing to do. The vast majority of dental pathology is completely hidden from view as it is inside the tooth, inside the bone, below the gum line. We are not just talking about periodontal disease. Among the things that might be found with radiographs are:

- tooth resorption in both cats and dogs (yes, tooth resorption is common in dogs too)
- subgingival root fractures
- evidence of endodontic (pulp) disease
- abnormal anatomy
- retained root remnants
- unerupted teeth with or without cyst formation.

Even if your radiographic study reveals no abnormal findings, that too is very valuable information. It documents the current healthy status and establishes a baseline for comparison to future radiographs. You will never be sorry that you have a complete set of radiographs on-hand, but you may often regret not having this information.

And post-extraction images are also a must as they document that the tooth was removed completely or will reveal root tips still in need of removal so that you do not inadvertently leave some in place.

The Pros and Cons of CR versus DR Systems

I am not going to talk about analog film as it is essentially a dead technology, though I do have a paper on my website that does discuss film. I am going to discuss the differences between DR (digital radiography) and CR (computed radiography). I have a strong bias here based on my years of experience with both systems. I receive no compensation for sharing this opinion. Personally, I have both systems (a Sopix2 DR sensor and a Scan X CR system). I use and like
both systems but if I could only have one, it would be my Scan X system without a moment of hesitation.

DR systems consist of a relatively small (size 2) but thick, solid, rigid sensor that is attached by a wire to your computer (wifi versions are available too). The sensor is placed in the mouth, and exposed to radiation and within seconds, a digital image appears on your computer screen. For a single image, this system is the fastest with respect to how fast the image appears on your screen. Another advantage is that you can leave the sensor and tube head untouched until the image is on the screen, make any appropriate adjustments to their positioning and shoot again to see if the result is better or worse. This speeds the learning process for the beginner. Another neat feature of some (not all) DR sensors is that they cannot be over-exposed. There is even one system that is paired with the x-ray generator to provide automatic exposure.

The downsides to a DR system (in my opinion) are many. The size 2 sensor is too small for radiographing a large structure or a large dog and too big/thick/rigid for a micro dog (say, under 3 kgs). To do a whole-mouth series in a large dog would take dozens of images and even though each image may be fast, when you have to do dozens, it takes a long time and then you have a large number of small images and to my eye, this is harder to interpret than a few larger images of the same thing. With DR systems, you are putting the expensive part (the sensor) in the patient’s mouth, where it is at risk of being chewed upon (and destroyed).

CR systems include the Scan X system, which is the one I have. With CR systems, the expensive part is the scanner which sits safely on the countertop and sends the digital image file to your computer. Of all of the systems on the market, only the Scan X has more than one reading slot (my machine has four slots, but the more common Scan X Duo has two). I will explain why this is important in a moment.

The other part(s) of the CR system are the Phosphor Sensor Plates (PSPs). These are thin, flexible plastic sheets with a radiosensitive phosphor coating on one side. The plates are typically available in the standard film sizes of 0, 1, 2, 3, 4. Some companies offer other sizes (like one designed specifically for rabbits). One company (MidMark) offers what they call a size 4c, which is actually smaller than a size 4 and so I would not be attracted to that system. I love my full size 4 plates.

The plate is placed in a sealed, disposable plastic envelop, placed in the mouth and exposed, removed from the envelop and placed into the scanner to be read. It takes longer for the image to show up on the screen (compared to a DR sensor), but while the scanner is reading one plate you can be setting up and exposing the next plate. My team and I are so fast at doing this that we have the second plate ready to go into the scanner before the first one is out, and this is why I recommend the Scan X Duo with two slots. If the first slot is still busy, we can stick the next plate in the other, vacant slot. For me, a scanner with just one slot would be a real bottleneck in the system. This might not be an issue for beginners who are slower but eventually you might regret getting a single-slot machine.

The PSP plates are flexible and so can be bent to help fit them into tight spaces. The small size 0 plates are great for micro dogs and the large plates are great for medium and large dogs, nasal shots, paw-o-grams, budgie and hamster-grams and more. Using a combination of size 4 and size 2 PSPs, I can do a complete and beautiful whole-mouth study in a large dog with 12 exposures.14

There is a selfish reason for me recommending CR systems to you. Many veterinarians send me radiographs for assessment and I look at many more on VIN. If the patient is a medium to large dog, I really prefer CR studies (fewer, larger images) over DR studies. It is hard to
appreciate the beauty of a mountain vista when looking through a small window. A large, picture window makes it so much better.

**How to Get Set-Up to Quickly and Efficiently Obtain the Images**

While it may sound odd, you can speed up your radiograph acquisition by just making it policy that all patients get whole-mouth radiographs at the very beginning of the procedure. This saves the time it would take the team to decide which images to take if you were only doing some rather than all. Also, obtaining and interpreting intra-oral dental radiographs is a learned skill and as such, you need practice, practice, practice. You will get good at obtaining and interpreting diagnostic images faster if you practice at every opportunity.

The next thing is to develop a routine on which shots you take and in what order. There is no single right way to do this, but I tend to start with patients lying on their left side, so I radiograph the right side first, starting at the back of the maxilla, move forward to the rostral maxilla, then do the same for the mandible. Then I turn the patient over and repeat for the left side.

When using the Scan X system, I set up plate one and expose it. I hand that plate to an RVT who feeds it into the scanner while I set up the next plate and expose it. I pass plate two to the RVT who feeds it into a vacant slot while I set up plate three and we repeat this until I have exposed all the plates from one side. Then I do a visual examination of the mouth while the scanner finishes reading the first set of plates. Then I flip and repeat.

**Positioning Tips**

There are many resources showing the positioning for the standard views in dogs and cats including the parallel and bisecting angle technique. I have a paper in the CVJ that shows some other helpful views and tips and I recommend you get a copy of that paper.

One very helpful projection is known as the occlusal view. I use it most often as a post-operative view to confirm complete extraction of the caudal maxillary teeth or to look for retained roots in that area. I place the sensor flat against the arcade parallel to the palate and shoot dorsoventral through the sockets. Not only can this help you see retained maxillary premolar and molar roots more clearly, it can also help you determine where the root is in three dimensions.

When using the bisecting angle technique for the maxillary 4\textsuperscript{th} premolar teeth you can get a great view of the distal root but often the mesial and palatal roots are superimposed over each other. I have a short paper on my website that talks about how to address this using parallax shift.

There are a number of accessories marketed to help you hold your sensors in place in the mouth. Forget these. The best thing is just a wad of paper towel. It is clean, takes whatever shape you want, is disposable and inexpensive.

**Care and Maintenance of the Equipment.**

For DR systems, there is virtually no maintenance. To keep the sensor clean, we place it in a disposable clear plastic contamination-barrier sleeve. These are available from any dental supply company and are sold as Air-Water Syringe covers (they are intended as contamination barriers for the air-water syringe on your dental cart, so you should have these on hand anyway). Then, because the DR sensors are almost all black and the mouth tends to be a dark place, we put a latex finger cot over the sensor too. This holds the other barrier in place and makes it easier to see the sensor in the mouth (you need to be able to see your target when taking aim at it). At the end of the procedure, just remove and dispose of these coverings and wipe the wire down with a disinfectant to remove any blood or other contamination.
The main thing with the DR sensors is to ensure that your patient is well anesthetized and will not reflexly bite down on the sensor when it is placed in the mouth. One bite can ruin a sensor and they cost upwards of $10,000 so that hurts. Your practice insurance policy might cover damage to one sensor but if you make a habit of this, they will cut you off. Always check jaw tone and reflexes before placing the sensor.

For DR systems, there is more to maintenance. If any blood or schmutz gets on the phosphor side of a plate, it will result in a white mark on them image. Most such debris can be removed by wiping with 99% isopropyl alcohol. To avoid contaminating the plates, always use a fresh, sealed contamination barrier for each exposure and handle the plates by their edges.

PSPs can also be damaged by being chewed upon but at least they are a lot less expensive to replace. I have found that they do not do well with being laundered (they start to delaminate) so always check your pockets before putting your clothes in the laundry. Badly damaged plates should be replaced.

If dust or fur gets inside the scanner, it will block the laser beam and there will be a white line down the length of the image. Sometimes such debris can be removed using the adhesive cleaning sheets from the manufacturer. Sometimes that dose not work and the transport mechanism may need to be removed to clear the debris. Contact your vendor for specific instructions for your machine.

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TOOTH RESORPTION IN DOGS AND CATS
Fraser A. Hale, DVM, Dipl AVDC

Introduction:
Anyone who spends any time in the mouths of pet dogs and cats has come across tooth resorption in its various forms. But what is tooth resorption, what causes it, how do we manage it and is there anything we can do to prevent it? Those are all great questions, but the answers remain elusive.

Space limitations in these proceedings do not allow for a comprehensive discussion of any of those issues here, but I have a number of resources at www.toothvet.ca and I will provide you with links to the most relevant ones. To start with, review these two:

Reminder – to diagnose, monitor and manage tooth resorption in any patient, you absolutely must have diagnostic intra-oral dental radiographs. If you cannot obtain and interpret intra-oral dental radiographs, you must refer these cases to someone who can.

What we are not talking about:
A tooth with chronic septic pulp necrosis (whether there is direct pulp exposure or not) will go on to develop chronic apical periodontitis. This inflammation around the tip of the root can cause inflammatory root resorption from the apex up as well as destruction of the surrounding bone. External inflammatory root resorption from chronic septic apical periodontitis is not the subject of this presentation.

Another form of endodontically-induced resorption is known as internal resorption. In this, case, live but inflamed dental pulp resorbs dentin on the inside of the pulp chamber. We will not be discussing this either.

Anatomy Review:
The crown of the tooth is that part covered by enamel and the root is covered by cementum. Where the crown and root meet is the neck, cervix or cementoenamel junction (CEJ). The bulk of the tooth is composed of dentin and inside the tooth is a hollow chamber containing the dental pulp.

Most of the root is situated in a depression in the bone known as the alveolus. The most coronal portion of the root extends above the margin of the alveolar bone. Gingiva is the tough soft tissue that lies against the tooth and alveolar bone. The free-gingival margin is above the CEJ. As the gingiva does not attach to enamel, the gingiva above the CEJ is known as the free gingiva and the space between the free gingiva and the enamel is known as the gingival sulcus. From the CEJ to the crest of the alveolar bone, the gingiva should be firmly attached to the root cementum in a tight collar completely encircling the tooth. It is this collar of gingival attachment that isolates the periodontal ligament space from oral bacteria and acts as the barrier to periodontal disease. This is important to remember when determining if a resorbing tooth requires extraction or not.
Within the alveolus, there is a space between the root and bone (the periodontal ligament space), occupied by the periodontal ligament, which attaches to the bone and cementum. The periodontal ligament space appears radiographically as a fine radiolucent line following the contour of the root.

**Dogs Vs Cats:**

While tooth resorption has been a common and recognized problem in domestic cats for many decades, we are now seeing it quite commonly in dogs as well. Does this represent the emergence of a new problem in dogs (increase in incidence) or an increase in diagnostic rate as whole-mouth intra-oral dental radiographs have become commonplace in general practice? I believe that is a question we will never be able to answer. Regardless, the old days of thinking of tooth resorption as a feline problem are gone forever. It is now common enough in dogs that you should look for it in every dog you see.

Tooth resorption in both dogs and cats can take various forms. Some forms of resorption look clinically and radiographically very similar in both species whereas there is a form seen in some dogs that I have never seen in a cat mouth. The paper on feline tooth resorption describes type 1 and type 2 tooth resorption and here is a brief review of that.

**Types of Tooth Resorption in Cats:**

From the Nomenclature page at https://avdc.org/avdc-nomenclature/.

On a radiograph of a tooth with type 1 (T1) appearance, a focal or multifocal radiolucency is present in the tooth with otherwise normal radiopacity and normal periodontal ligament space.

On a radiograph of a tooth with type 2 (T2) appearance, there is narrowing or disappearance of the periodontal ligament space in at least some areas and decreased radiopacity of part of the tooth.

On a radiograph of a tooth with type 3 (T3) appearance, features of both type 1 and type 2 are present in the same tooth.

To the right is a radiograph of the left mandible of a cat with type 1 tooth resorption. There is loss of bone and tooth structure, no new hard tissue is filling the defects and beyond the areas of tissue loss, the bone, roots and periodontal ligament space all look normal. The mesial root of the 4th premolar has been transected allowing bacteria into the endodontic system of this tooth resulting in septic pulp necrosis. The infection has then oozed out through the tip of the distal root resulting in apical periodontitis and bone loss (arrow).

Type 1 lesions are associated with moderate to severe gingivitis and/or periodontitis. They typically start at the CEJ and can extend in all directions from that starting point. The cause of these lesions might well be local inflammatory reaction due to gingivitis or periodontitis. It is hard to say if the resorptive lesions are the cause of the periodontitis or the result of it.

Type 2 lesions start on the root surface within the socket and below the gingival attachment, isolated from the oral bacteria and are believed to be asymptomatic until the lesion breaks through the gingival attachment. Once the lesion extends coronal to the gingival sulcus, it becomes
visually apparent as soft tissue where you should be seeing enamel as in the photograph of a lower left canine (previous page).

Type 2 lesions are associated with extensive loss of root structure, loss of periodontal ligament and ankylosis. Where tooth tissue is being removed, the lesions are filled in with new hard tissue known as bone-like cementum-like tissue.

The radiograph (which is of the cat in the above photograph) shows advanced type 2 tooth resorption affecting both lower canine teeth. The right canine lesion (to your left) is all within the confines of the alveolus but the one affecting the left canine tooth has extended above the alveolus and onto the crown, as shown in the photograph.

Types of tooth resorption in dogs:

I have not seen published definitions of the different types of tooth resorption in dogs but some of the lesions I see are similar to feline Type 1 lesions while some are very similar to feline Type 2 lesions. Another form I have seen in dogs but not in cats is illustrated in the radiograph to the right. While tooth structure is being resorbed, radiographically-normal looking bone fills in where root tissue once was and the periodontal ligament space remains radiographically visible.

Causes of Tooth Resorption:

This will be a short and disappointing section because I believe we just do not know what causes of tooth resorption.

The etiology of tooth resorption in cats has been pondered and investigated more than it has in dogs. There have been a lot of theories put forward and then found wanting. One theory that still has some adherents is that it is caused by hypervitaminosis D. There is some credible evidence to support this notion, but a follow-up study refuted the findings and conclusion. I have seen no published theories on the etiology in dogs. So, I think the safest stance currently is we just do not know what causes tooth resorption. My discharge statement for any patient who has been diagnosed with tooth resorption bears the following statement:

*We do not know the cause of tooth resorption, but we know that dogs/cats who have had some are likely to have more. There is no way to predict which teeth will be affected next or when and we currently have no recommendations for slowing progression or preventing new lesions. All we can do is monitor and extract teeth as they become clinical problems.*

That said, I suspect that type 1 tooth resorption in dogs or cats may be caused by periodontal disease in the same way that periodontitis results in resorption of the alveolar bone.

Treatment for Tooth Resorption:

Not every tooth undergoing resorption requires treatment. For instance, the 2nd and 3rd premolars in that dog premolar radiograph above are certainly undergoing resorption, but the lesions are deep in the socket, well below intact gingival attachment. There is no communication between the resorbing areas and the bacteria-laden environment of the oral cavity. These teeth
are believed to be completely asymptomatic and it would be quite acceptable to simply
document their current status, leave them in place and radiograph them again in a year. The
first premolar, on the other hand has virtually no root left and should be ‘popped out’ now. For
more things to consider when trying to decide whether to extract today or not, please review this

Type 1 tooth resorption in cats and in dogs is associated with gingival inflammation and
periodontal disease and can be probed so are in communication with the oral environment.
Therefore, type 1 tooth resorption requires complete removal of the entire tooth (no root
remnants can be left in place). Sometimes, because of the loss of tooth structure, these teeth
fracture during extraction. Removal of the roots requires technique that cannot be taught here
but in short, one would reflect a flap labial and lingual to the tooth, smooth down the bone/root
with a #4 round bur, then cut a moat between the root and bone with a #1/2 round bur. Once
there is a moat around the root, a very fine elevator, such as a Cislak 1.3S can be used to
loosen the root and tease it from the socket. I have some YouTube videos that include this. In
this one, I use the moat technique to remove the palatal root of an upper 4th premolar in a dog
https://youtu.be/hwWjLGE6hqs. And in this one, I demonstrate the technique for removing an
uncooperative lower canine tooth from a cat - https://www.youtube.com/watch?v=DrjeimmhUig.

With type 2 tooth resorption, there is at least some ankylosis (fusion of root to bone with
calcification of the periodontal ligament) as well as loss of the structural integrity of root +/-
crown and the tooth is pretty much certain to crumble during extraction. This is where a great
deal of controversy arises. Given the difficulty in identifying, let alone removing these resorbing
roots, some advocate intentional root retention (more commonly known as ‘crown amputation’).
I do not practice, endorse or teach intentional root remnant retention. On my table, if a tooth
needs to come out, it ALL comes out. I remove all clinically/radiographically identifiable dental
tissue. However, I do not feel the need to remove the new bone that has replaced the lost root
structure.

Other considerations:
For cats with Feline Chronic Gingivostomatitis (http://www.toothvet.ca/PDFfiles/fcgs.pdf), you
must remove every scrap of every root of every tooth – no intentional root retention allowed.

In cats, there are some domino effects to be aware of when extracting teeth. For instance, if a
cat loses and upper canine tooth, there is a tendency for the upper lip to sag in and then the
lower canine traumatizes the upper lip as described in this paper -
http://www.toothvet.ca/PDFfiles/lip_entrapment.pdf. Therefore, if an upper canine tooth in a cat
mouth requires extraction, you may also be compelled to remove the lower canine, even if it is
healthy.

The other domino issue in cats arises when the lower molar needs to go. After loss of the lower
molar, the upper 4th premolar becomes functionally useless and is at risk of traumatizing the
mandible (http://www.toothvet.ca/PDFfiles/Feline_Gum_Chewer.pdf). Therefore, if I have to
remove a cat’s lower molar, I will also remove the upper 4th premolar and the upper molar.

Conclusion:
Tooth resorption is very common in cats and quite common in dogs. So common, in fact, that
you should assume that every patient has at least some tooth resorption until proven otherwise
by detailed clinical and radiographic examination under general anesthetic.

Teeth with lesions that are in direct communication with the oral cavity or very close to it should
be extracted. While some contend that there are circumstances in which intentional root
remnant retention is allowable, I continue to believe that when a tooth needs to come out, it all needs to come out.

At present, we do not have a solid sense of the cause(s) of tooth resorption, but it seems that animals who have had some resorption are likely to develop more. We have nothing to offer in the way of preventative measures or ways to predict rate of progression or which other teeth might be affected next or when. All we can do is monitor and extract teeth as they become clinical problems.
SURGICAL MANAGEMENT OF GINGIVAL HYPERPLASIA

Fraser A. Hale, DVM, Dipl AVDC

Due to space limitations, this paper has been edited and images deleted. For the fully-illustrated version visit www.toothvet.ca/PDFfiles/gingival_hyperplasia.pdf. I would also encourage you review this paper on periodontal anatomy and physiology - http://www.toothvet.ca/PDFfiles/PerioAnat&Physio.pdf. And watch my video on periodontal anatomy and disease progression at https://youtu.be/uRExyTGheAM for background.

Gingival hyperplasia refers to the pathological growth of excess or redundant gingival tissue as a result of an increase in the number of cells. By contrast, gingival overgrowth refers to an increase in gingival volume due to an excessive accumulation of extracellular matrix proteins.

In gingiva hyperplasia, the tissue is basically histologically normal (other than usually being inflamed) or may contain islands of dystrophic calcification or bony metaplasia but it is not considered a neoplasia. Of course, the distinction between a benign neoplasia of gingival origin and hyperplasia of gingiva is at times fuzzy and arbitrary. Some pathologists (Brian Wilcock, notably) feel that gingival hyperplasia and peripheral odontogenic fibroma (fibromatous epulis) are just different addresses along a single continuum, but that is a discussion for another day.

There are some breeds that are reported to be predisposed to idiopathic, generalized gingival hyperplasia including the collie, Great Dane, Dalmation and Doberman. In my practice, boxers are by far the most commonly affected, though one of my most dramatic cases was in a wheaton terrier. We do not know why boxers (or others) develop this exuberant gingival over-growth, but one theory is that it is their way of reacting to gingival inflammation. Rather than getting gingival recession, they get hyperplasia.

There are several drugs that have been associated with gingival overgrowth (the increase in matrix protein form of enlargement) including phenytoin derivatives, calcium channel blockers and cyclosporine. Since gingival overgrowth is a drug-induced increase in extra-cellular matrix proteins, the treatment is based on discontinuing the offending medication if at all possible. Once the drug is withdrawn, the gingival overgrowth will typically regress. In some cases, the gingiva will return to normal height and contour with no further treatment required. In others, some enlargement may persist and surgical gingivoplasty may still be indicated.

If you have done as I suggested and reviewed the video on periodontal anatomy and physiology, you will recall that the free gingival margin should taper to a knife-sharp edge and that it should be about 0.5 to 3 millimeters coronal to the cemento-enamel junction. Since the gingiva does not attach to the enamel but lies against, there is a potential space between the gingiva and the enamel known as the gingival sulcus. If the gingiva is healthy, the free gingival margin is not only very thin, it is also firmly braced against the enamel by dense collagen fibers and it is tucked under the cervical bulge. In this situation, debris (hair, food, woodchips…) coming along the crown will be deflected away from the sulcus rather than being forced into it.
At the bottom of the gingival sulcus, the (relatively weak) junctional epithelium is attached to the root cementum and then this gives way to the (much firmer) connective tissue attachment of the gingiva to the cementum and the outer surface of the alveolar bone. The body wants to maintain these three zones (sulcus, junctional epithelium, connective tissue attachment) in a relationship termed biologic width.

As the free gingiva becomes inflamed (reaction to dental plaque on the crown of the tooth), it starts to become edematous and collagenase starts to break down the stiff collagen fibers in the free gingiva. The rounded free-gingival margin starts to fall out of contact with the enamel opening up the potential space of the gingival sulcus to create an actual space. This makes it easier for debris and bacteria to enter the sulcus, resulting in more inflammation...setting up a downward spiral.

In those predisposed to hyperplasia, the free gingiva starts to grow up the crown of the tooth (coronally) as well as growing in thickness. Even if the attached gingiva remains attached right at the cemento-enamel junction, the sulcus becomes much deeper and so it becomes a periodontal pocket. In this case, as the increase in pocket depth is due to the coronal migration of the free gingival margin (rather than the apical migration of the floor of the pocket) it is referred to as a false pocket or a pseudopocket.

Diagrammatically, it might look like this:

This pseudopocket is a wonderful trap for hair, food, and of course, microbia. Chewing and brushing cannot reach into these deep spaces and so the advantage is given to the periodontal pathogens and periodontal disease is likely to ensue. Inflammation in the depths of the pseudopocket leads to loss of gingival attachment, destruction of alveolar bone and periodontal ligament (so the addition of a true periodontal pocket as the level of attachment migrates toward the apex of the root). This increased risk of developing periodontal infection and irreversible tissue loss is one of the reasons that gingival hyperplasia and gingival overgrowth needs to be addressed.

Another way in which gingival hyperplasia or overgrowth becomes clinically significant to the patient is seen when the enlargement becomes extreme. In advanced cases, the patient cannot close his/her mouth without chewing on the redundant/hyperplastic tissue. Even before this occurs, chewing on food and treats may cause the loose flaps of gingiva to be traumatized. The result of this trauma = pain!

As the ever-expanding gingiva is present as a constant force, it can actually result in the movement of teeth, sometimes all but pulling teeth out of their sockets.

With the problems arising from the redundant gingiva (false pockets, trauma, orthodontic tooth movements), the condition definitely needs treatment. The first step is to determine if the patient is on any medications that might be causing a drug-induced gingival enlargement. If the patient is on one of those drugs, get it off. If there is no history of being on any of the suspect drugs, and the problem is generalized
(surrounding many if not all teeth in a fairly symmetrical fashion), then you are likely dealing with idiopathic gingival hyperplasia and surgery will be required. Treatment is aimed at reshaping the gingiva back to normal height and contour. We are trying to re-establish normal and healthy gingival architecture.

First steps include whole-mouth intra-oral dental radiographs combined with probing and exploration to assess the status of the teeth and bones hiding below all this excess tissue. Especially in boxers, with their brachycephalic dental crowding, it is common to find extensive periodontal disease, particularly around the premolars. Teeth with advanced disease will need to be extracted.

The next step is to determine the appropriate level for the incision around the teeth that are going to stay in the mouth. We want to remove enough gingiva without removing too much. The literature describes probing to the bottom of the pocket to note its depth, then removing the probe and laying it on the outside of the gingiva with the tip at the level of the bottom of the pocket. Then the tip of the probe is driven into the outer surface of the gingiva at that level to create a bleeding point. This process is repeated every 2-3 mms around the tooth to create a dotted line of bleeding points that outline the location of the pocket depth and then this can be used as a guide for the excision line. I do not like this method, because if there is a true pocket as well as a false pocket, the surgeon may end up removing too much gingiva and exposing furcations of multi-rooted teeth.

The goal in gingival recontouring is to shape the gingiva back to normal height and thickness. We want the new free-gingival margin (after healing) to be 1-3 mms coronal to the cementoenamel junction so that the body can re-establish the normal biologic width. Depending on the modality used for resection, the surgeon should anticipate a post-operative retraction or loss of tissue during healing. This may amount to as much as 1 millimeter. Therefore, the level of the incision should account for this.

In the diagram below, the red line indicates the beveled angle and location of the incision and the shaded tissue is that which is to be removed. This incision reduces the depth of the pseudo pocket and also recreates a thin free-gingival margin that is located beneath the cervical bulge but above the cementoenamel junction. During healing, the tissue will retract to the level of the green line leaving the patient with normal gingival architecture.

If the pocket extends beyond the cementoenamel junction, that is a true periodontal pocket and requires periodontal surgery that is aimed at preserving and regenerating periodontal tissues. We do **not** reduce the depth of true periodontal pockets by removing gingiva. Only the pathologically increased and redundant gingiva is removed.

There are four modalities used for the removal of the excess gingiva and all have their applications and limitations, fans and detractors.
The simplest and most available technology is cold-steel in the form of a scalpel blade or a gingivectomy knife (Orban or Kirkland). As the excess gingiva is typically quite firm and may contain islands of osseous metaplasia and since the blade will contact the tooth surface when it has cut through the hyperplastic gingiva, blades will dull quickly and require frequent replacement (for scalpels) or sharpening (for gingivectomy knives). As well, since the gingiva has an excellent blood supply, cold-steel excision tends to be a very bloody procedure. Digital pressure with saline-soaked gauze on fresh incisions will help establish hemostasis. While time-consuming and bloody, cold-steel technique is least likely to cause collateral damage to surrounding tissues. On the other hand, the pressure needed to force the blade through tough gingival tissue can make fine control of the shape and depth of incision difficult.

Another ‘cold’ technique involves the use of twelve-fluted carbide burs in a high-speed dental hand-piece with a cooling spray of water directed at the bur tip as it cuts through the tissue. This method is somewhat faster and can allow for more accurate sculpting of the tissues but does require more equipment. Also, if the surgeon is not very careful, as the bur penetrates through the soft tissue it may contact and damage the hard tissues (enamel, cementum or bone) beneath.

Those who own a laser (CO\textsubscript{2} Nd:YAG, diode) seem to think it is a good choice for gingivoplasty. Reported advantages of laser surgery include sealing of the blood vessels during incision to maintain hemostasis and a clear field of view. The incision is also sterilized by the action of the laser (though the cut surfaces in an oral environment will soon be contaminated regardless). However, laser gingivoplasty is more time consuming, can increase healing times through the creation of the char layer and there is a considerable risk of collateral damage to surrounding hard and soft tissues. Several human and veterinary references recommend against the use of laser for gingival surgery.

My preferred modality for gingivoplasty is radiosurgery. Radiosurgery is essentially electrosurgery but the frequency of the electrical current is within the radio frequency range (3.0 to 4.0 megahertz) which is higher than regular electrosurgical units. The higher frequency means that there is less lateral heat dissipation into the surrounding tissues and used at an appropriate power setting, there is less thermal necrosis along the incision than with lower-frequency units and maybe even less than with laser. Radiosurgery is reported to result in less scar tissue formation than with laser. It is also far less expensive.

The Ellman™ Surgitron™ provides various wave-form options (pure cutting, cutting with coagulation, straight coagulation and fulguration modes) at 3.8 MHz. I perform gingivoplasty using the filtered (pure cutting) or the rectified (cut/coagulation) wave form. This allows the electrode to pass through the tissue without any pressure applied to incise the tissue while cauterizing small blood vessels as it cuts. The result is a clean, smooth, easily controlled and bloodless incision. My initial incision is a debulking cut to coarsely remove much of the redundant tissue and increase visualization of the underlying tooth. Then I make refining incisions gently removing small amounts of tissue at a time to bring the gingival margin to within a millimeter of the desired target height.
While the incised tissues do blanch a bit immediately after the cut, they ‘pink-up’ again within minutes.

While the Ellman™ Surgitron™ is a powerful tool, like all powerful tools, it is not without its risks. Just as you can take your fingers off faster with a power saw than with a hand saw, so can you cause serious tissue damage by careless use of radiosurgery. The power setting should be adjusted to the lowest setting that will allow the electrode to pass through the tissue without ‘drag’. The electrode should be kept on the move rather than allowing it to rest in one spot while active, as this would allow greater dissipation of heat into the surrounding tissues. The incision should be kept well away from bone and contact with the tooth avoided.

Analgesics such as NSAIDs and narcotics should be dispensed for several days post-operatively. The patient should be fed a softened diet and hard toys and treats withheld for two weeks. The owners should suspend any home care (tooth brushing) during the healing period as well. Once the gingiva has re-epithelialized (10 to 14 days), owners should return to, or start working on a safe, effective daily plaque-control program (http://www.toothvet.ca/PDFfiles/HomeCarePack.pdf). The theory is that, in patients predisposed to generalized gingival hyperplasia, keeping the teeth clean and the gingiva healthy may delay the recurrence of the hyperplasia and increase the time before surgical debulking becomes necessary again.

It is important to set reasonable expectations for clients. Removing excess gingiva does not remove the underlying predisposition to develop more. It does not tend to grow back in areas where teeth have been extracted. Wherever there are teeth, you and the owners should anticipate that the hyperplasia will recur in time and so the procedure may need to be repeated periodically.
UPDATE ON ADDISON’S DISEASE IN DOGS: WHAT IF MY PATIENTS ELECTROLYTES ARE NORMAL?

J. Catharine Scott-Moncrieff, MA, Vet MB, MS, DACVIM DECVIM
Professor Internal Medicine, Dept Veterinary Clinical Sciences, School of Veterinary Medicine
Purdue University, West Lafayette, IN, USA

INTRODUCTION
Hypoadrenocorticism (Addison’s disease) results from failure of the adrenal glands to secrete glucocorticoids and mineralocorticoids. Most cases of hypoadrenocorticism are due to primary adrenal failure, resulting in deficiency of usually both cortisol and aldosterone from the adrenal cortex. More rarely, Addison’s disease may be due to pituitary dysfunction resulting in a failure of ACTH secretion and pure glucocorticoid deficiency (secondary adrenal failure). In secondary hypoadrenocorticism, mineralocorticoid secretion is expected to be normal.

CLINICAL SIGNS
Signalment: Seventy percent of dogs diagnosed with hypoadrenocorticism are female, and most are young to middle-aged dogs (mean 4 - 5 years). The disease is heritable in the Standard Poodle, Bearded Collie, Portuguese water dog and the Nova Scotia Duck Tolling Retriever (NSDTR) and in these breeds no obvious sex predisposition is evident. In the Standard Poodle, Portuguese Water dog, and NSDTR, the disease appears to be inherited as an autosomal recessive trait. Incidence of hypoadrenocorticism in the NSDTR is estimated to affect 1.4% of the population while in the Standard Poodle 8.6% of poodles in one study were affected.

History and Physical examination: Clinical signs may be either acute or gradual in onset and often wax and wane. Owners may not realize how long their dog has been ill until treatment results in a dramatic improvement in activity level. Since 85-90% of adrenal reserve must be depleted before clinical signs are observed, it may require a stressful event to trigger clinical illness. Clinical signs may be very vague. Anorexia, vomiting, lethargy/depression, weakness, weight loss, diarrhea, shaking/shivering, polyuria, polydipsia, and abdominal pain may be observed. Most of these clinical signs can occur due to glucocorticoid deficiency alone. If mineralocorticoids are also deficient, the clinical signs tend to be more severe and polyuria, polydipsia, hypovolemic shock, collapse and dehydration are often present. Less common clinical signs include acute gastrointestinal hemorrhage, and seizures due to hypoglycemia or electrolyte derangement. The physical examination may be normal or may reveal lethargy, weakness, dehydration, bradycardia, weak pulses, decreased capillary refill time, and other evidence of hypovolemic shock.

DIAGNOSTIC TESTING FOR HYPOADRENOCORTICISM
Clinical Pathology
A complete blood count may reveal a nonregenerative normocytic normochromic anemia; alternatively the hematocrit may be increased due to dehydration. Eosinophilia, neutrophilia or lymphocytosis are found in only 20-30% of dogs with hypoadrenocorticism, but lack of a stress leukogram in a dog with systemic illness is common. A chemistry profile may reveal hyponatremia, hypochloremia, hyperkalemia, hypercalcemia, and hyperphosphatemia. These changes occur due to aldosterone deficiency with a resultant failure of the kidneys to conserve sodium. Other possible serum biochemical abnormalities include hypoalbuminemia, hypercholesterolemia, hypoglycemia, and increased liver enzymes. Specific gravity of the urine is commonly less than 1.030. The changes on the minimum data base in dogs with hypoadrenocorticism may initially mimic other disorders such as renal failure, hepatic disease, gastrointestinal disease, or insulinoma.
Serum electrolyte abnormalities
The majority of dogs with hypoadrenocorticism have the classic electrolyte changes of hyponatremia and hyperkalemia due to aldosterone deficiency.

Na:K ratio
The Na:K ratio is usually low in dogs with hypoadrenocorticism, and this ratio may be useful to guide emergency diagnosis and treatment while waiting for definitive test results.

Imaging studies
Most untreated dogs with hypoadrenocorticism have one or more radiographic abnormalities on thoracic and abdominal radiographs including microcardia, small cranial lobar pulmonary artery, narrow posterior vena cava, or microhepatica. Occasional dogs may have evidence of megaesophagus. Most dogs with hypoadrenocorticism have a measurable reduction in size of the adrenal glands, and sometimes the adrenal glands cannot be identified on ultrasound.

Electrocardiogram
In dogs with hyperkalemia, abnormalities may be present on the electrocardiogram. These include a peaked T wave and shortening of the QT interval in mild hyperkalemia, widening of the QRS complex, decreased QRS amplitude, increased duration of the P wave, and increased P-R interval in moderate hyperkalemia, and loss of P waves and ventricular fibrillation or asystole in severe hyperkalemia.

Basal cortisol
Measurement of a basal cortisol of >55 nmol/l (2 microg/dl) is a useful test to exclude a diagnosis of hypoadrenocorticism. Measurement of a basal cortisol concentration is not adequate for confirmation of a diagnosis of hypoadrenocorticism however, because some dogs have a low basal cortisol concentration but have an appropriate response to ACTH administration.

ACTH stimulation test
An ACTH stimulation test is necessary to confirm a diagnosis of hypoadrenocorticism because not all dogs with hypoadrenocorticism have the expected electrolyte changes, and because many other disorders may mimic the characteristic findings of Addison’s disease. In dogs with hypoadrenocorticism, both the pre- and post- ACTH cortisol concentrations are usually less than 27 nmol/l, (1 microg/dl), and both values should be less than the reference range for basal cortisol (usually 55 nmol/l, 2 microg/dl) to confirm the diagnosis.

ATYPICAL ADDISON’S DISEASE
It is now recognized that although most dogs with hypoadrenocorticism have obvious electrolyte changes such as hyponatremia and hyperkalemia, a subset of dogs with hypoadrenocorticism lack these electrolyte changes. In a retrospective study of dogs with hypoadrenocorticism, 24% of dogs lacked hyponatremia and hyperkalemia. In a study of 25 NSDTR, 32% lacked electrolyte abnormalities at the time of diagnosis. Reasons for normal electrolytes include secondary hypoadrenocorticism due to decreased ACTH secretion, selective destruction of the zona fasciculata and reticularis, early stage disease in which there has not yet been complete destruction of the zona glomerulosa, or ability to compensate for sodium wasting by increasing sodium intake. Dogs with glucocorticoid deficient hypoadrenocorticism tend to be older, have a longer duration of clinical signs, and are more likely to be anemic, hypoalbuminemic and hypocholesterolemic than those with electrolyte changes.

HOW DOES RECOGNITION OF ATYPICAL ADDISON’S DISEASE CHANGE THE DIAGNOSTIC APPROACH?
It is important for clinicians to recognize that an absence of the characteristic electrolyte changes of hyponatremia and hyperkalemia does not exclude a diagnosis of hypoadrenocorticism. Clinicians should have a high index of suspicion for hypoadrenocorticism so that this very treatable disease is not missed. Conversely reliance on measurement of electrolytes alone for diagnosis of hypoadrenocorticism can be misleading, because there are
many other causes of hypernatremia and hyperkalemia; the diagnosis must be confirmed by endocrine testing.

When evaluating any systemically ill dog the possibility of hypoadrenocorticism should always be considered. Useful clues may be the presence of waxing and waning illness, improvement with conservative management, and improvement after administration of glucocorticoids. Lack of a stress leukogram, and identification of small adrenal glands on ultrasound, also increases suspicion for hypoadrenocorticism. In dogs with suspected hypoadrenocorticism measurement of a basal cortisol is useful to exclude the diagnosis without the expense of an ACTH stimulation test.

MAINTENANCE THERAPY

Options for long term mineralocorticoid treatment include fludrocortisone (starting dose 0.02 mg/kg/day as a single dose or divided) or desoxycorticosterone pivalate (starting dose 2.2 mg/kg q 25 days). For both of these mineralocorticoids the dose should be titrated to effect. The dose of fludrocortisone typically needs to be increased over time whereas in many cases the final individualized dose of DOCP is < 2.2 mg/kg. Prednisone is typically recommended for glucocorticoid replacement. The starting dose is 0.2 mg/kg per day and the dose should then be tapered to the lowest dose that will control the clinical signs. It is important to avoid excess prednisone supplementation because this may result in manifestations of iatrogenic hyperadrenocorticism. Only 50% of dogs treated with fludrocortisone require supplemental prednisone, whereas most dogs treated with DOCP require prednisone at least every other day.

HOW DOES DIAGNOSIS OF ATYPICAL ADDISON’S DISEASE CHANGE THE DIAGNOSTIC APPROACH?

The main difference between managing dogs with classic Addison’s and those with atypical Addison’s disease is that dogs without electrolyte derangements do not require mineralocorticoid treatment and respond well to treatment with glucocorticoids alone. Treatment costs are therefore much cheaper. Doses of prednisone required to control the clinical signs are typically <0.1 mg/kg/day and some dogs can be managed on every other day treatment. If significantly higher doses of prednisone are required to control the clinical signs the possibility of an alternative diagnosis should be considered.

Dogs with atypical Addison’s should be monitored using clinical signs reported by the owner, physical examination findings (especially weight) and measurement of electrolytes. Serum electrolyte concentrations should initially be monitored frequently because some dogs that present without electrolyte derangements will develop them later. The ACTH stimulation test is not part of the regular monitoring for animals with either classic or atypical Addison’s disease.

REFERENCES


INTRODUCTION

Currently in the United States, most dogs with canine hyperadrenocorticism (HAC) are treated medically with trilostane or oDDD (mitotane). Hypophysectomy is available at a limited number of referral centers in the US. Surgical adrenalectomy is the treatment of choice for adrenal tumors (AT); however, medical therapy with mitotane and trilostane can also be effective in managing clinical signs in dogs with AT. When formulating an approach to treatment, it is ideal to identify which form of HAC is present. Tests that are used to differentiate PDH from AT include the low and/or high dose dexamethasone suppression tests, measurement of endogenous ACTH concentration, and diagnostic imaging modalities such as ultrasound and computed tomography.

Both mitotane and trilostane may be effective in management of both pituitary dependent and adrenal dependent HAC; both drugs have advantages and disadvantages. In some cases, it may be necessary to transition a patient from trilostane to mitotane or vice versa. Reasons for changing from one drug to the other include adverse drug effects, disease relapse, owner’s preference or ability to monitor the patient, and medication costs. When planning the drug transition it is important to understand the different mechanisms by which these drugs control the clinical signs of HAC and the different protocols for administration of the drugs.

TRILOSTANE

Trilostane is a synthetic hormonally inactive steroid analogue, which is a competitive inhibitor of the 3 ß-hydroxysteroid dehydrogenase system. The drug blocks synthesis of adrenal steroids including cortisol and aldosterone. Trilostane is rapidly absorbed orally (peak concentrations within 1.5 hours) although suppression of plasma cortisol concentrations is short lived (< 20 hours). Current recommendations for use of trilostane are to start at a dose of 1-2 mg/kg q 12 - 24 hrs and then increase or decrease the dose based on evaluation of ACTH stimulation tests performed 4-6 hours after drug administration. Twice a day therapy at a starting dose of 1 mg/kg may result in good control of clinical signs at a lower total daily dose with less risk of adverse effects than higher doses q 24 hours. ACTH stimulation testing should be performed 10, 30 and 90 days after start of treatment and 30 days after each dose adjustment. The goal of treatment
is to have both the pre- and post-cortisol measurement in the normal resting range (2-6 microg/dl), however higher ACTH stimulation test results are acceptable if the clinical signs are controlled. Measurement of a pre-trilostane cortisol concentration may be useful in assessing the ideal frequency of trilostane administration. Measurement of basal cortisol concentration may also be useful to assess response in some cases. Trilostane is well tolerated in most dogs. Adverse effects that are usually mild and self-limiting include diarrhea, vomiting, and lethargy in up to 63% of treated dogs. Occasional dogs develop hypoadrenocorticism which is generally glucocorticoid deficient only, although dogs with evidence of mineralocorticoid deficiency have been reported. Hypoadrenocorticism induced by trilostane is generally reversible, although in rare cases this may take several months, likely because of adrenocortical necrosis. There have been anecdotal reports of acute death shortly after starting trilostane treatment. In a recent retrospective study of dogs treated with either mitotane (n= 25) or trilostane (n=123), long-term survival was not statistically different between the groups. Median survival in the mitotane group was 708 days (range 33-1399), and in the trilostane group was 662 days (range 8-1971). Of the dogs that died in this study, 11% died due to causes that were attributed to the underlying PDH, and a further 17% died of causes that could have been related to their underlying PDH. In 38% of cases, the cause of death could not be directly attributed to PDH, and in 34% the cause of death was unknown.

MITOTANE

Mitotane is derived from the insecticide DDT and is a potent adrenocorticolytic agent. The drug causes progressive necrosis of the zona fasciculata and zona reticularis of the adrenal gland and at higher doses causes necrosis of the zona glomerulosa. Other effects of mitotane include fatty degeneration, centrilobular atrophy and congestion of the liver. Normal dogs are clinically quite resistant to the effects of the drug. Mitotane should never be administered in animals that are not eating well. Treatment with mitotane is begun at a dose of 50 mg/kg divided q 12 hours. Glucocorticoids are not usually administered concurrently, but a small supply of prednisone should be made available to the owner for emergencies. Mitotane at induction doses is typically administered for 5-10 days, until water consumption decreases to < 100 ml/kg/day. Mitotane should be discontinued immediately if a decreased appetite, depression, diarrhea, or vomiting are observed. At this point, the dog is reevaluated and an ACTH stimulation test performed. Prednisone treatment (0.1 to 0.2 mg/kg) is initiated in patients that are showing clinical signs of hypocortisolemia, until the results of the ACTH stimulation test are known. In patients which are not polyuric or polydipsic, patients in which water consumption cannot be monitored, and patients in which polydipsia is due to another
underlying disease (e.g. diabetes mellitus), mitotane should be administered for a maximum of 5-7 days prior to ACTH stimulation testing. The goal of treatment is to have both the pre- and post-cortisol measurement in the normal resting range (2-6 microg/dl). Maintenance therapy (50 mg/kg q 7-10 days) is started once the ACTH stimulation test shows adequate suppression and prednisone therapy (if necessary) has been discontinued. Failure to use maintenance therapy will result in re-growth of the adrenal cortex and recurrence of clinical signs. Efficacy of maintenance therapy is monitored by an ACTH stimulation test in 1 month and thereafter every 3 - 4 months. The dose of mitotane required for long term maintenance in dogs with PDH is very variable (26-330 mg/kg/week). Reasons for treatment failure include incorrect diagnosis, the presence of an adrenal tumor (although some adrenal tumors will respond well), loss of drug potency due to poor storage or compounding of mitotane, or a need for a higher dose or duration of treatment in some dogs. Side effects of mitotane include gastric irritation, hypoadrenocorticism, hepatotoxicosis and very occasionally neurologic signs. Mean survival time of 200 dogs treated with mitotane was 2.2 years (range 10 days to 8.2 years).

TREATMENT OF ADRENOCORTICAL TUMORS

Treatment of adrenal neoplasia involves either surgical resection of the tumor or medical therapy with mitotane, or trilostane. Adrenalectomy should be reserved for patients that do not have evidence of extensive tumor invasion or metastasis. Abdominal ultrasound and computed tomography can assist in planning the surgical approach.

Dogs with inoperable adrenal tumors or metastatic disease may respond to medical treatment with op-DDD (mitotane) or trilostane. Mitotane is often successful in controlling clinical signs and in some cases may result in tumor shrinkage. In one study, the mean dose of mitotane required to control clinical signs in dogs with adrenal tumors ranged from 35 to 1275 mg/kg/week. Trilostane may also be used to control clinical signs of HAC in dogs with functional adrenal tumors. In one study there was no difference in survival times for dogs with adrenal tumors treated with either mitotane or trilostane.

TRANSITIONING FROM MITOTANE TO TRILOSTANE

The most common reasons to transition from mitotane to trilostane include adverse effects of mitotane and frequent relapse of clinical signs while on mitotane treatment. Important data to obtain prior to the transition include a complete history and physical examination, review of endocrine testing, and diagnosis of cause of hyperadrenocorticism (pituitary dependent versus functional adrenal tumor). Mitotane should be discontinued prior to starting trilostane therapy. It is recommended to wait at least one month after discontinuation of mitotane before starting trilostane. Trilostane should not be initiated until there is recurrence of clinical signs of
hyperadrenocorticism, and until the post-ACTH cortisol concentration is within the upper reference range or greater than the upper limit of the reference range. Close monitoring of adrenal function is advised, as dogs previously treated with mitotane seem to be more responsive to the effects of trilostane. Trilostane treatment should be initiated at the lower end of the dose and ACTH stimulation testing performed as recommended above.

**TRANSITIONING FROM MITOTANE TO TRILOSTANE**

The most common reasons to transition from trilostane to mitotane include adverse effects of trilostane (e.g. hyperkalemia) and treatment of dogs with adrenal tumors. The adrenal glands become enlarged in dogs treated with trilostane and trilostane has no inherent cytotoxicity for adrenal tumors. Mitotane on the other hand can result in shrinkage of adrenal tumors. Although this would suggest that mitotane is the preferable drug to use in dogs with functional adrenal tumors, survival studies of dogs with adrenal tumors have shown similar survival with both drugs. Important data to obtain prior to the transition include a complete history and physical examination, review of endocrine testing, and diagnosis of cause of hyperadrenocorticism (pituitary dependent versus functional adrenal tumor). Trilostane should be discontinued prior to starting mitotane therapy. Mitotane should not be initiated until there is recurrence of clinical signs of hyperadrenocorticism, and until the post-ACTH cortisol concentration is within the upper reference range or greater than the upper limit of the reference range. Because of the short half-life of trilostane, a long wash out period between the two drugs is usually not necessary, however close monitoring of adrenal function is advised, as dogs previously treated with trilostane may be more sensitive to the effects of mitotane because of the adrenal hyperplasia induced by trilostane. Mitotane should be initiated at the standard induction dose and the dog monitored carefully for signs of hypoadrenocorticism as described above. An ACTH stimulation test should be performed after induction and the after the first 30 days of maintenance therapy.

**REFERENCES**

PATHOPHYSIOLOGY OF DIABETES MELLITUS

Diabetes mellitus (DM) is a common endocrine disease in dogs and cats which is characterized by an absolute or relative deficiency of insulin. This results in a decreased ability of cells to take up and utilize glucose, amino acids, fatty acids, and electrolytes. Insulin deficiency results in increased gluconeogenesis, glycogenolysis, lipolysis, ketogenesis, and protein catabolism.

Two types of DM are recognized in dogs and cats. Type I DM is due to an absolute deficiency of insulin. This form of diabetes is characterized by minimal secretory response to β-cell secretagogues such as glucagon and is the most common form of DM in dogs. Type II DM is characterized by an abnormal pattern of insulin secretion in combination with peripheral insulin resistance and is the most common type of DM in cats. Type II diabetic cats may go into diabetic remission if they achieve good glycemic control however unfortunately there is no practical diagnostic test that can be used to distinguish between the two types of DM in cats. Factors that likely influence whether a diabetic cat goes into remission include the severity of pancreatic pathology, whether there is concurrent disease that causes insulin resistance, whether the cat is overweight, and whether the cat is fed a low carbohydrate diet.

DIAGNOSIS

The diagnosis of DM is made based on characteristic clinical signs of diabetes mellitus (polyuria, polydipsia, polyphagia, and weight loss), and documentation of hyperglycemia and glycosuria. In cats, the diagnosis is complicated by stress hyperglycemia. When making a diagnosis of DM in cats, it is therefore important not only to document persistent hyperglycemia and glucosuria, but also to rule out other diseases that may cause similar clinical signs such as hyperthyroidism and gastrointestinal disease. Measurement of fructosamine concentrations or urine glucose concentrations of samples collected in the home environment, allows the clinician to distinguish between stress induced hyperglycemia, and persistent hyperglycemia due to diabetes mellitus. Measurement of fructosamine is unreliable for cats with concurrent hyperthyroidism because increased protein turnover decreases fructosamine concentration. Glucosuria may also occur secondary to ketamine anesthesia, chronic renal failure, and post-obstructive diuresis so is not on its own diagnostic for diabetes mellitus. The presence of significant ketonuria or ketonemia and concurrent hyperglycemia is diagnostic for diabetes mellitus.

TREATMENT OF DIABETIC PATIENTS

Treatment of diabetes mellitus in dogs and cats relies on insulin therapy, dietary modification, management of concurrent illness and weight management.

INSULIN THERAPY

Table 1: Insulin products currently available commercially and used in cats and dogs in USA

Short duration:

Regular insulin (Zinc insulin crystals)

Products: Humulin R [Lilly], Novolin R [NovoNordisk] Both human recombinant. 100 U/ml
Moderate duration:
NPH insulin (neutral protamine hagedorn)
Products: (Humulin N [Lilly], Novolin N [NovoNordisk] Both human recombinant 100 U/ml
Lente insulin (65% crystalline and 35% amorphous)
Product Vetsulin (Merck) pork 40 U/ml

Long duration:
PZI insulin
Insulin complexed with protamine and zinc.
Product: ProZinc [Boehringer Ingelheim] human recombinant (40 U/ml)
Glargine
Insulin analogue
Product: Lantus [Sanofi-Aventis], human recombinant (100 U/ml)
Detemir
Insulin analogue
Product: Levemir [NovoNordisk], human recombinant (100 U/ml)

Insulin treatment in cats: There are three insulin products that are appropriate for first line treatment of diabetes mellitus in cats; Protamine zinc insulin, Lente insulin, and Glargine insulin. NPH insulin tends to have a very short duration of action in cats and is not recommended as first line insulin.
The starting dose for insulin in a new feline diabetic patient is 1-3 U/cat (0.25 – 0.5 Unit/kg). It is recommended that insulintreatment is started at the lower end of this dose. It is difficult to predict in advance which cats will do better with which insulin formulation. Cats should be carefully monitored for occurrence of hypoglycemia, because of the possibility of remission of diabetes mellitus. A blood glucose curve should be performed 7-14 days after making any change in insulin formulation. Whichever formulation is chosen, twice a day insulin therapy is more likely to result in good glycemic control than once a day therapy. If twice a day treatment is not possible once a day therapy with PZI or Glargine can result in effective control of clinical signs in some cats.

Insulin treatment in dogs:
The most effective insulin formulations in dogs are Lente Insulin (Vetsulin/Caninsulin) and human recombinant NPH (Humulin N) at a starting dose of 0.25 - 0.5 U/kg twice a day. Use of human recombinant insulin or pure pork insulin avoids the complications that can occur due to development of anti-insulin antibodies in dogs treated with beef/pork insulin. The long acting insulin PZI has also been approved for once a day use in dogs. The starting dose for PZI is 0.7 U/kg q 24 hours. In dogs with rapid metabolism of insulin PZI can also be administered twice a day. Insulin analogs such as Glargine and Detemir are not appropriate for initial management of canine diabetic patients, however treatment with these products may be helpful in dogs that have a very short duration of insulin action. Detemir has been evaluated in a small number of diabetic dogs. It is important to be aware that this insulin is much more potent than other insulin in the dog and the starting dose is lower. The dose needed for good glycemic control in one study ranged from 0.05-0.34 U/Kg.

Goals of insulin treatment: The primary goal of insulin therapy in diabetic patients is to control clinical signs of DM while avoiding hypoglycemia. Severe hypoglycemia can be life-threatening and even mild insulin- induced hypoglycemia can result in clinical signs of poor glycemic control due to the insulin resistance that results from secretion of anti-insulin hormones such as glucagon, growth hormone, cortisol, and epinephrine. Persistent severe hypoglycemia can lead to permanent neurologic damage. The long-term benefits of tight glycemic control, while well established in human diabetic patients have not been demonstrated in dogs and cats; although
Theoretically better glycemic control should result in fewer diabetic complications such as recurrent infection, proteinuria, and cataract formation. The likelihood of diabetic remission in cats is higher with tighter glycemic control. The goals of diabetic regulation should therefore take into account the lifestyle of the owner, the presence of concurrent illness, the age of the patient, and the practicality of tight glucose monitoring.

Ideally the blood glucose should be maintained between 80 and 200 mg/dl, however most patients will have some blood glucose concentrations that fall above this range and most patients are clinically well regulated if most of the blood glucose concentrations are less than 300 mg/dl. Occult hypoglycemia is an important cause of poor glycemic control and can lead to unnecessary visits to the emergency clinic. If the blood glucose falls below 80 mg/dl on the BG curve, the insulin dose should be decreased. It is important to remember that it is difficult to assess the duration of insulin action if the glucose nadir is in the hypoglycemic range because this can lead to release of counter-regulatory hormones such as glucagon which drives the blood glucose back up prematurely.

**MONITORING DIABETIC PATIENTS**

The ideal monitoring strategy should be multimodal and individualized for the patient and owner(s). Parameters that can aid in assessing the adequacy of diabetic control include clinical signs, bodyweight, serial blood glucose concentrations measured at home or in the clinic, fructosamine concentrations, glycosylated hemoglobin concentration (HbA1C), and urine glucose concentrations. The presence of ketones in the blood or urine can also be useful to indicate the presence of impending diabetic ketoacidosis. The most important factor in assessing diabetic control is whether clinical signs are well controlled. Blood glucose concentrations, urine glucose concentrations and glycated proteins should be interpreted in the light of the clinical signs. Monitoring should be individualized to meet the needs of the patient and owner.

**Blood glucose curves:**

Although blood glucose curves have long been considered to be the gold standard for evaluating glycemic control, they have significant limitations, in particular the effect of stress, and the presence of day-to-day variability. Blood glucose curves are expensive and require collection of multiple blood samples which can be stressful to the patient even when performed by the owner at home. Misinterpretation of blood glucose curves can lead to incorrect treatment decisions.

**Glycosylated proteins:** Measurement of glycosylated proteins allow assessment of longer-term glycemic control and can aid in interpretation of blood glucose curves. Glucose binds irreversibly to serum proteins and hemoglobin and these products persist for the life of the proteins. The resultant products can be measured in serum or whole blood respectively. Fructosamine indicates adequacy of glycemic control over the previous 2-3 weeks, while HbA1C reflects glycemic control for the previous 4-6 weeks.

**Urine glucose:** Urine glucose concentrations can also be used to assess glycemic control and are particularly helpful in cats to assess for the presence of diabetic remission as well as to detect relapse. Urine glucose should not be used to determine the daily dose of insulin but trends in urine glucose can be very helpful in assessing diabetic control especially if assessed on a consistent basis and recorded in a diary or log.

**Continuous glucose monitoring (CGM):** Newer continuous interstitial glucose monitoring (CGM) techniques are changing the approach to blood glucose monitoring. These systems allow continuous evaluation of interstitial blood glucose concentration for up to 14 days via a small flexible subcutaneous catheter, replacing the blood glucose curve. The newer systems are affordable, easy to use, and well tolerated by patients. The reports that are generated can be downloaded as a pdf or uploaded to a website and allow an integrated analysis of changes in blood glucose over a 14 day period.
The Freestyle Libre device is the most common CGM used in veterinary medicine. Purchase of the sensor and reader requires a prescription. The sensor is a one-time use disposable device while the reader is a onetime purchase and can be used multiple times with different sensors. The reader allows wireless monitoring of the interstitial glucose. Alternatively, the owner can download the Freestyle Libre app for Android or iPhone and download the data to their phone from the sensor. The reader and the app can be used together as long as the reader is initially used to set up the sensor.

Two different devices are available: the Libre 14 day device and the Freestyle Libre 2 device. The Freestyle Libre 14 day is the device most frequently used in cats and dogs, however currently the Freestyle Libre reader is hard to find.

A video demonstrating how to place a freestyle Libre 14 day sensor can be accessed using the QR code below.

![QR Code](image)

Depending upon the patient, the sensor can be left uncovered, or can be protected by an adherent patch or dressing, a pet sweater, or a thunder shirt. A covering is recommended in active patients or patients with house mates that might attempt to remove the sensor. The reader is able to read the sensor through a jacket or bandage. Although the sensor is waterproof, we do not recommend bathing the pet or allowing the pet to swim while the sensor is in place.

The Freestyle Libre sensor (FSL) measures the interstitial glucose every minute and stores this data every 15 minutes on the sensor disc. This disc can store up to 8 hours of data. Every time the sensor is scanned, the data is downloaded onto the reader. The sensor can be scanned at any time but it needs to be scanned at least once every 8 hours in order to obtain continuous readings. Data from the reader can be uploaded to a computer for viewing as a pdf file any time during the life of the sensor. Complications with both the device and the patient do sometimes occur. Patients can develop erythema at the placement site and occasionally abscesses at the insertion site develop.

The FSL generates 24 hour interstitial glucose curves for up to 14 days, although early detachment occurs in as many as 60-80% of dogs and cats. The FSL can measure glucose concentrations between 40 and 500 mg/dl, however the graphs that are generated in the FSL reports do not display glucose concentrations greater than 350 mg/dl. The FSL is an important tool in patients with diabetic ketoacidosis, in patients with newly diagnosed DM and in unstable diabetic patients, in which it can be used continuously until better glycemic control is achieved. The FSL is also very useful for routine intermittent monitoring of glycemic control in stable patients.

Interstitial glucose measured by the FSL, correlates well with both peripheral blood glucose measured by both point of care glucometers, and blood glucose measured by reference methods. Although overall there is a good correlation, the FSL usually slightly overestimates the blood glucose in the euglycemic and hyperglycemic ranges, while slightly underestimating the
blood glucose in the hypoglycemic range. It is important to understand that there is a lag of a few minutes between changes in the blood glucose and changes in the interstitial glucose, so these measurements may differ, especially when the blood glucose is changing quickly. When a consensus error grid method was used to analyze the performance of the FSL compared to a reference method, the difference between the two measurements had little or no effect on clinical decision making for more than 98% of the samples. The performance of the FSL device is not affected by ketosis, but it is slightly less accurate in dehydrated animals. This should be taken into account when using the FSL in patients with Diabetic ketoacidosis.

The data from the FSL, can be viewed in real time using either the FSL reader or a cell phone app. Data from the FSL reader can be uploaded to a computer using free software, or can be uploaded to the Libreview website. The Libreview website allows the data from multiple patients to be stored in the cloud, and accessed not only by the owner, but also by all the veterinary care providers in a practice. Data from the Librelink cell phone app can also be wirelessly uploaded to the Libreview website. Once the data is uploaded, the user can generate a summary report, which can be viewed on-line or downloaded as a pdf.

The FSL summary report has a number of different viewing options that display the 24 hour interstitial glucose either in a day-by-day format or an integrated format. The integrated format allows an assessment of daily trends in interstitial glucose while the day-by-day format allows better assessment of day to day variability.

A major advantage of the FSL is the ability to collect 24-hour data for a diabetic patient. This is very helpful for determining different insulin requirements between the daytime and nighttime hours as well as to evaluate response to long-acting insulins given once a day. The other important advantage of the FSL curves is the ability to evaluate day-to-day variability in insulin response.

Interpretation of individual curves is similar to interpretation of a traditional blood glucose curve but with the ability to better appreciate day-to-day variability. The glucose nadir, duration of insulin effect and average glucose can be easily determined. Ideally, the glucose nadir should fall between 80 and 150 mg/dl and the glucose concentration should be below 300 mg/dl for the majority of the day. Problems that can be detected using the FSL glucose reports include inadequate dose of insulin, inadequate duration of insulin action (rapid metabolism), insulin-induced hypoglycemia and lack of response to insulin. Lack of response to insulin suggests either poor client compliance or insulin resistance. Based on the assessment of the curve, a change in insulin dose or formulation can then be made and the response assessed while the sensor is still in place. Because the glucose measurements are available in real time, clinically relevant hypoglycemia can be diagnosed and treated immediately, and the insulin dose decreased. When using the FSL to adjust the insulin dose, it is still important to wait 5-7 days between dose increases. With a sensor life of 14 days, it is usually possible to make two adjustments to the insulin dose during the life of the sensor; of course, the dose can be decreased multiple times if necessary.

Although the correlation between the FSL and point of care (POC) glucometers is usually good, sensor failure or sensor errors can and do occur. If the FSL glucose measurements do not fit with the clinical picture, the blood glucose measurement should be measured using a POC glucometer or another trusted method. Indications of sensor failure include an error message, a message indicating that the sensor should be scanned again at a later time, gaps in data, and unexpectedly wide swings in the blood glucose that do not fit with clinical signs. In these situations, if the results of the FSL do not correlate with the measurement from the POC device, the FSL sensor should be replaced.

In summary the FSL is a device that can be very valuable in assessment of glycemic control in dogs and cats. Having a good understanding of this technology allows maximum utilization of the advantages of this monitoring system and can improve the ability to accurately monitor diabetic patients.
REFERENCES


ACROMEGALY

Pathophysiology: Acromegaly is a syndrome in cats caused by excess secretion of growth hormone from a pituitary adenoma. Excess growth hormone (GH) secretion results in increased secretion of Insulin growth factor I (IGF-1) from the liver and peripheral tissues. Cats with acromegaly have profound insulin resistance and overgrowth of soft tissues, membranous bone and viscera. The anabolic effects of IGF-1 result in proliferation of bone, cartilage, and soft tissues, with organomegaly. The excess growth hormone secretion causes insulin resistance, carbohydrate intolerance, hyperglycemia and diabetes mellitus. Growth hormone is believed to cause a post receptor defect in insulin action at the level of the peripheral tissues. Feline acromegaly was until recently believed to be a rare condition in cats, however recent studies suggest that it may be a more common cause of insulin resistance in diabetic cats than has been previously recognized. In one study in which diabetic cats were screened for acromegaly by measurement of Insulin growth factor I (IGF-1), 32% had increased IGF-1 concentrations and acromegaly was confirmed in 10% of those cats.

Signalment and clinical signs: Most cats with acromegaly are middle-aged or older (median 10 years of age, range 4-17 years) and 90% are male (intact or castrated). Clinical signs in cats with overt acromegaly include polyuria, polydipsia, polyphagia (due to insulin resistant diabetes mellitus), large body size, weight gain despite poor glycemic control, and enlargement of the head and extremities. Median insulin dose in a group of 17 cats with acromegaly was 10 U q 12 hours (range 2-35 U). Physical examination may reveal abdominal organomegaly, inferior prognathia, cataracts, clubbed paws, broad facial features, cardiac murmurs or arrhythmias, respiratory stridor, lameness, peripheral neuropathy, and central neurologic signs attributable to an enlarging pituitary mass. Cardiomegaly and renomegaly may be evident on imaging studies. Although weight loss due to poorly regulated diabetes mellitus may occur initially, a key finding in acromegalic cats is weight gain or a stable weight (lack of weight loss) in a diabetic cat with other indications of poor glycemic control such as polyuria, polydipsia, and polyphagia. Median bodyweight in a report of 19 acromegalic cats was 6 kg (range 4-9 kg). Although acromegalic cats tend to gain weight rather than lose weight as the disease progresses, in the study discussed above, the body weights of the acromegalic cats were not significantly different than the body weights of well controlled or poorly controlled diabetic cats without acromegaly.

Acromegaly is a slowly progressive insidious disease. Some cats with acromegaly do not have the classic signs of weight gain and soft tissue enlargement at the time of diagnosis. It is likely that currently only the most severely affected acromegalic cats are diagnosed. In a recent study some acromegalic cats were phenotypically indistinguishable from normal cats and in other cats the changes were subtle and not obvious to the owners. Acromegaly should be considered in the differential diagnosis for any cat with insulin resistance especially if their body weight is stable to increasing, regardless of whether or not the more typical features of soft tissue enlargement are present. Some clinicians have even recommended evaluation for acromegaly in any cats that does not go into diabetic remission with appropriate diet and insulin therapy.

Diagnosis: A tentative diagnosis of acromegaly is based on the presence of consistent clinical signs, together with measurement of growth hormone and IGF-1 concentrations. Assays for
both IGF-1 and GH have been validated in the cat although measurement of IGF-1 is more widely available. Measurement of IGF-1 is a good screening test for acromegaly. The assay has been reported to have a specificity of 92% and sensitivity of 84%. Some poorly controlled diabetic cats have elevations in IGF-1 concentrations but in most cases these increases are not as high as in those cats with documented acromegaly. Occasional diabetic cats with no evidence of acromegaly have more substantial increases in IGF-1 so imaging of the brain should be performed to confirm the diagnosis. Most cats with acromegaly have increased serum GH concentration although the short half life and episodic secretion of GH may explain why GH is normal in some cases of acromegaly. The assay for GH is not widely available. A tentative diagnosis of acromegaly based on measurement of GH or IGF-1, should be confirmed by either contrast enhanced CT or MRI of the brain to document the presence of a pituitary mass. In one case of confirmed acromegaly, acidophil proliferation within the pituitary gland did not result in a detectable mass on CT or MRI. Thus even negative MRI findings do not preclude a diagnosis of acromegaly.

**Treatment:** Treatment modalities that have been evaluated for acromegaly in cats include radiation therapy, hypophysectomy, and medical therapy with somatostatin analogs such as octeotide, and pasireotide, and dopamine receptor agonists such as cabergoline. A long-acting somatostatin analogue pasireotide, has shown promise in the management of acromegalic cats. Treatment with cabergoline has shown promise in some studies and not others. Radiation therapy has been reported to result in improvement of neurologic signs in cats with pituitary tumors, and to result in decreased insulin requirements or diabetic remission in cats with acromegaly. Unfortunately, the cost and availability of radiation therapy often limit access to treatment. Hypophysectomy is effective for treatment of acromegaly in cats but is not widely available. In those cases in which treatment of the tumor is not possible due to financial or logistical concerns, long-term control of clinical signs of diabetes mellitus may be achieved using high doses of insulin. Because of the profound insulin resistance associated with acromegaly, hypoglycemic complications using this approach are unusual. Survival times of 4-42 months (median 20 months) were reported in 14 cats with acromegaly. Only two of these cats were treated with radiation therapy. Cause of death is most commonly due to renal failure and/or congestive heart failure.

**HYPERADRENOCORTICISM**

Hyperadrenocorticism (HAC) is another rare endocrine disease of cats associated with insulin resistance. Eighty percent of cats with hyperadrenocorticism are diabetic at the time of diagnosis of hyperadrenocorticism. Hyperadrenocorticism in cats is caused by either a functional pituitary tumor (PDH) or a functional tumor of the adrenal cortex.

**Signalment and clinical signs:** cats with hyperadrenocorticism are middle aged to older (median 10 years of age, range 5-16 years) and females are slightly over-represented. Clinical signs of hyperadrenocorticism in cats include signs of insulin resistant diabetes mellitus (polyuria, polydipsia, polyphagia, weight loss, and peripheral neuropathy), lethargy, abdominal enlargement or pot-bellied appearance, muscle atrophy, unkempt hair coat, bilaterally symmetrical alopecia, cutaneous fragility, and recurrent abscess formation. Cats with hyperadrenocorticism are also predisposed to bacterial infection so clinical signs of urinary tract infection, pyoderma and respiratory tract infection may also be observed. In addition to the findings already discussed above, the physical examination may reveal hepatomegaly, seborrhea, and cutaneous lacerations. Skin fragility may be so severe that tearing of the skin occurs during routine grooming of the hair coat by the cat. Virilization has been reported in cats with sex-hormone secreting adrenal carcinomas, and concurrent hyperaldosteronism has also been reported.

**Diagnosis:** The results of a CBC, biochemical panel, and urinalysis are typically consistent with those of diabetes mellitus. Increased alkaline phosphatase, alanine transferase, hypercholesterolemia, hyperglycemia, and low BUN are common. Cats do not have a steroid
induced isoenzyme of alkaline phosphatase, so changes in this enzyme are less prominent than seen in dogs, and occur secondary to poorly regulated DM. Endocrine tests that may be useful in substantiating a diagnosis of feline hyperadrenocorticism include the ACTH stimulation test, the low dose dexamethasone suppression test, and the cortisol:creatinine ratio. The urine cortisol:creatinine ratio is a useful screening test for hyperadrenocorticism. Urine for measurement of the C:Cr ratio should be collected at home to minimize the influence of stress. A normal value makes a diagnosis of hyperadrenocorticism unlikely. Increased values occur in cats with Cushing’s, however increases may also occur in cats with other concurrent illness so additional testing should be used for confirmation. The low dose dexamethasone suppression test is considered to be the test of choice for diagnosis of feline HAC and is performed using a higher dose of dexamethasone (0.1 mg/kg IV) than in the dog. A base-line blood sample is collected, and additional samples are collected at 4 and 8 hours after dexamethasone administration. Cortisol concentration will be suppressed (<1.5 µg/dl) at 8 hours in normal cats but not in cats with HAC. A few cats with HAC will suppress normally on the LDDS. If the index of suspicion for HAC is high a second test using the lower dose of dexamethasone (0.01 mg/kg) can be performed but interpretation is difficult because some normal cats will fail to suppress at this dose. The ACTH stimulation test is not a particularly sensitive test in cats because it is only abnormal in 50-60% of cats but it is useful in cases in which dexamethasone suppression testing is difficult to interpret and in cats with suspected iatrogenic HAC. The ACTH stimulation test is performed using a dose of 125 µg of Cortrosyn administered either IV or IM. Samples should be collected at baseline, and at 30 and 60 minutes after administration of ACTH. A post ACTH cortisol of >15 µg/dl at either 30 or 60 minutes after administration is consistent with a diagnosis of HAC.

Some adrenal carcinomas in cats have been associated with high circulating concentrations of other adrenal hormones such as androstenedione, progesterone, 17 hydroxy progesterone estradiol and testosterone. Cortisol concentrations in these cases are typically low with little response to ACTH stimulation. A sex hormone secreting tumor should be suspected in cats with clinical signs of hyperadrenocorticism, an adrenal mass on ultrasound and low cortisol response to ACTH. Confirmation is by a sex hormone profile with hormones measured before and after ACTH stimulation testing.

**Differentiation testing:** Either the high dose dexamethasone suppression test, endogenous ACTH stimulation or diagnostic imaging are used to differentiate pituitary dependent from adrenal dependant hyperadrenocorticism in cats. Eighty five percent of cats with HAC have the pituitary dependent form of the disease, while 15% are diagnosed with functional adrenocortical tumors. Adrenocortical carcinoma should be suspected in cats with sex hormone secreting adrenal tumors.

**Treatment:** Treatment options for cats with HAC depend upon whether the disease is pituitary dependent or adrenal dependent. Adrenalectomy is the treatment of choice in cats with adrenal tumors. In cats with PDH, the best outcomes have also been reported with bilateral adrenalectomy and trilostane treatment. Other drugs Drugs that have been used with varying success include mitotane, metyrapone, and aminoglutethimide. Other options in cats with PDH include hypophysectomy or radiation therapy of the pituitary tumor.

**HYPERALDOSTERONISM**

Primary hyperaldosteronism (Conn’s syndrome) is a disorder of middle-aged to geriatric cats due to autonomous secretion of aldosterone from the adrenal gland. Excess aldosterone concentrations result in hypertension due to both increased plasma volume and increased peripheral resistance. The disorder is considered rare in cats, but it is likely an underdiagnosed disorder because the hypertension and hypokalemia that are the hallmarks of this disorder may also occur in cats with CKD. Primary hyperaldosteronism should be suspected in any cat with hypertension and hypokalemia, especially if the hypertension or hypokalemia are relatively refractory to treatment. Causes of hyperaldosteronism include a mineralocorticoid secreting...
adrenal tumor (carcinomas are more common than adenomas) or idiopathic bilateral nodular hyperplasia of the zona glomerulosa. The most prominent clinical signs of hyperaldosteronism in cats include signs of target organ damage due to hypertension, and polymyopathy due to hypokalemia. Other clinical signs may include skin fragility in cats with concurrent hypersecretion of progesterone, inappetance, polyuria, polydipsia, and weight loss. Laboratory abnormalities may include hypokalemia, azotemia, and increased creatine kinase activity. Diagnosis of hyperaldosteronism is made by documentation of increased aldosterone and decreased renin concentrations which results in an increased aldosterone to renin ratio. Other diagnostic testing used to support the diagnosis includes measurement of an aldosterone:creatinine ratio, abdominal ultrasound that may demonstrate the presence of unilateral or bilateral adrenomegaly or the presence of an adrenal mass, or use of a fludrocortisone suppression test. The final diagnosis relies on histopathology of the adrenal gland.

TREATMENT OF HYPERALDOSTERONISM
When hyperaldosteronism is due to an adrenal tumor, adrenalectomy is the treatment of choice. In cats in which adrenalectomy is not possible due to bilateral tumors, bilateral nodular hyperplasia, presence of metastatic disease, or financial limitations, medical management includes treatment with the mineralocorticoid receptor inhibitor spironolactone, together with potassium supplementation and amlodipine to control hypertension.

SUMMARY
Feline acromegaly and feline hyperadrenocorticism are important causes of insulin resistance in cats. Although these disorders are rare, they are likely currently under diagnosed. Recent advances allow definitive treatment that may improve quality of life and long-term survival. Although the hallmark of both diseases is insulin resistance, other clinical features allow the clinician to distinguish the two disorders. It is important to be familiar with the clinical signs of both acromegaly and hyperadrenocorticism, so that early recognition can lead to early intervention. In addition if the clinician is familiar with the distinct clinical signs, then testing can be reserved for those cats that truly fit the clinical picture. Hyperaldosteronism is an important and under-diagnosed cause of hypertension and hyperkalemia. It is important to consider hyperaldosteronism in cats with CKD and hypertension especially if hypertension does not respond to routine management.

REFERENCES


DIAGNOSING AND TREATING GI STASIS IN PET RABBITS
Dennilyn Parker, DVM, MVetSc, ABVP (Avian)

GI stasis in rabbits is the slowing of the GI tract leading to decreased food and water intake and subsequent dehydration of the stomach contents. It is usually secondary to another condition – anything causing pain, stress, or decreased food intake, but needs to be addressed as a primary problem while also looking for the underlying cause. Common underlying causes: dental malocclusion – either from dental pain or inability to eat, urinary stones or sludge, urine scald, overweight rabbits, high carbohydrate diet, stressful surrounding, adhesions from previous abdominal, arthritis, etc.

These rabbits often present with decreased appetite and reduced fecal production. GI stasis caught early, especially if the underlying cause can be found and treated, usually respond very well to medical management. Prolonged GI stasis is another story and had a poor prognosis, and obstructions usually require intensive care and/or surgery. Sometimes these cases are easy to differentiate, sometimes the clinical signs overlap quite a bit.

Simple GI stasis
The owners report a gradual reduction in appetite and gradual reduction in fecal output and size of feces. These rabbits often present still bright and hoping around. Abdominal palpation will reveal a ‘doughy’ stomach that doesn’t appear painful, and when you palpate the stomach the contents will move back into shape – like a stress ball with some water in it. Often, once owners have been through a case of GI stasis, they recognize the signs early making treatment easier. These rabbits will have a normal temperature and emergency panel:

- Temperature 38.5-39.5°C
- Blood glucose 4-8 mmol/L
- PCV 34-43%
- TP 50-75 g/L
- Lactate 6.9 +/- 2.7 mmol/L

Simple GI stasis can be managed with:
- Fluids IV or SQ at 100-120ml/kg/day maintenance plus deficiencies
- Pain management – opioids e.g., buprenorphine 0.01-0.05 mg/kg SQ or IM q6-12 hours
- Assist feeding
- Exercise

Prolonged GI Stasis
If the GI stasis is prolonged, and the rabbit has been anorectic for several days, the rabbit will be in worse condition. They won’t be as active, their temperature may be decreased, and the lactate drops when their digestive system isn’t working properly. On palpation the doughy stomach is firmer and doesn’t return to its normal shape when indented – like a stress ball with no water in it. The longer the duration of GI stasis, the more critical the rabbits will be, and they can die if left untreated. These rabbits need more intensive treatment and will take longer to turn around. The prognosis is not as good. Treatment for these cases includes the same as above, but we will often add IV lidocaine at 2mg/kg IV loading dose over 5 minutes followed by a 100 micrograms/kg/min CRI.

GI Obstruction
Rabbits are unable to vomit, and this can sometimes make it difficult to differentiate GI stasis, especially advanced cases, from GI obstruction. Complete obstructions are emergencies. These
often present with a more acute onset, the owners will report they were fine a few hours before. The rabbit is painful and hunched or stretching out to get comfortable. These rabbits often have a low body temperature, high blood glucose from the pain and on palpation their stomach is firm like a blocked cat’s bladder. These rabbits are critical and will die quickly without treatment. If surgery isn’t an option for the owner, some have been managed with a lidocaine CRI, fluids and pain management. Passing a fenestrated feeding tube to relieve the pressure in the stomach can help. Many of these cases need to go to surgery.

Liver Lobe Torsion
Another cause of abdominal pain and anorexia in rabbits is a liver lobe torsion. These rabbits present in very critical condition. They have a low body temperature, high blood glucose and often have a very high lactate. On abdominal palpation, the torsed liver lobe may/may not be palpated and the stomach may feel like a GI stasis rabbit, which is secondary to the pain and inappetence. On further diagnostics, the liver values will be significantly increased. Imaging – either ultrasound or CT with contrast – should confirm the diagnosis. These rabbits should be stabilized as much as possible and then taken to surgery to remove the torsed lobe.

Prevention of GI Stasis:
Offer a good high fibre diet and lots of fresh water. Limit the amount of pellets offered, feed mostly hay and fresh leafy greens. Any diet changes should be done slowly. Lots of exercise. Minimize household stress. Good brushing – I like to pluck rabbits when they are shedding – especially the hair mats around the bum. Some prefer to shave rabbits to reduce ingestion of hair (even though this isn’t caused by a ‘hairball’).
BLOOD COLLECTION AND INTERPRETATION
Dennilyn Parker, DVM, MVetSc, ABVP (Avian)

General Tips:
We often use plasma instead of serum for our chemistries in exotic pets. One reason is that avian serum can turn into a gel, which can’t be used for testing. The other reason is we get a higher yield of fluid when we use an anticoagulant – which can make a difference in the number of tests run. In some avian and reptile species EDTA will lyse the cells. Many labs (but not all) will prefer reptile CBC submitted in heparin.

Birds:
Avian blood clots quickly. Some people will pre-heparinize the syringe to prevent this. We can take about 1% of body weight in a healthy bird – 10% of the body weight is blood and you can take about 10% of blood volume. This means you can take about 1ml in a 100 g cockatiel. Several sites can be used but in parrots we typically use the jugular vein. The right side is larger than the left, and you need to avoid the cervicocephalic air sac and the crop as these are potential sites for blood loss. The ulnar vein is small and can bleed quite a bit after venipuncture. The medial tibiotarsal vein is small in parrots but can be used in other species such as waterfowl. Birds have a poorly developed extrinsic clotting mechanism, so they need tissue factors to clot well. Liver disease, low calcium and other malnutrition factors can lead to excessive bleeding.

Interpretation of CBC:
We try to use established reference ranges for the species in question, remembering their limitations. As a very general and very rough guide:

- WBC count – usually < 12x10^9.
- WBC count over 40 x 10^9 is very significant and there are 4 major diseases we consider: Avian tuberculosis, chlamydiosis, aspergillosis, or an overwhelming bacterial infection
- Band cells – there should be <1% band cells – anything higher than this should make you pay attention, and if >3% especially with a high WBC
- Monocytes – increase with chronic disease

Interpretation of Plasma Chemistry:
Kidney evaluation:
- Uric acid is the most useful indicator of renal disease but not very sensitive or specific.
- Urea – low levels in birds – may increase in dehydration
- Creatinine – blood conc low in birds and do not correlate to kidney disease

Liver evaluation -
- Bile Acid – produced in liver and recycled – most useful test of liver function in birds
- AST increases with some liver disease, but also present in muscle, brain and kidney
- GGT – only low levels in liver – specific but not sensitive
- Bilirubin – not produced by birds
- Alk phos – not present in liver

Glucose is higher in birds than mammals
Calcium/Phosphorus is important esp in African greys. Calcium will increase in hormonally active birds

Ferrets:
Blood can be taken from several sites. The cranial vena cava is a common site. The jugular and ventral tail artery can also be used. A cephalic or toe pad poke can be used for a low stress
sample to determine a resting blood glucose. Simple fasting can increase the liver values in some ferrets. Isoflurane can significantly decrease PCV. Dexmedetomidine can increase blood glucose.

**Rabbits:**
Blood collection is often done from the lateral saphenous vein. The lateral ear vein can be used with caution. Blood collection and IV placement in the lateral ear vein has been reported to cause sloughing, although many experienced practitioners use it with good success. The jugular vein can be used, although the restraint can be stressful for a rabbit and when the neck is extended it can cause breathing difficulties (even leading to death).
Interpretation of rabbits CBC can be difficult as they don’t always increase the WBC count in the face of infection or inflammation but rather shift the heterophil:lymphocyte ratio.

**Reptiles:**
Lizards – ventral tail vein
Snakes – ventral tail vein, cardiac puncture
Turtles and tortoises – jugular, dorsal tail sinus
Geckos can drop their tail very easily – really need to consider how much you want the blood in these cases.
Reptile CBC are very useful. These species can be difficult to assess when they are sick and a CBC can help assess their immune system and monitor progression over the course of treatment. Calcium:phosphorus ration is also very important as many reptiles have a deficient diet and/or husbandry leading to hypocalcemia. Like birds, the most common cause of hypercalcemia would be a reproductively active female.
EGG RELATED REPRODUCTIVE ISSUES
Dennilyn Parker, DVM, MVetSc, ABVP (Avian)

Avian:
The most common reproductive issue is egg binding. This can be caused by many issues including an abnormally shaped egg, infection, or a calcium deficiency. Many species of parrots will lay an egg every other day. Some species will lay a set number of eggs per clutch while others, like cockatiels, will continue to lay until their nest is full. In species like cockatiels, they can keep laying eggs if they are taken away or if they break. Sometimes more than 50 eggs a year.

Husbandry changes can be made to reduce the number of eggs produced:
- Diet – reduce the amount of seeds/fat in the diet
- Remove any nesting areas
- Decrease the photoperiod
- Eliminate contact between the owner and bird that may be misleading – no petting down the back of the bird or under the wings
- Eliminate any perceived mate – another bird, mirror, toy – sometimes this is the owner which can be tricky

Deslorelin: Once all of the husbandry changes have been made deslorelin can be used to shut down egg production. It will increase production before it stops, so that is important to consider if there are concerns about the ability of the bird to pass an egg. The deslorelin implants have a relatively short duration and need to be used in conjunction with other changes.

Egg binding: This happens quickly, within a day. It may be the first egg of the clutch or the last egg. Early treatment can be successful with SQ fluids, sometimes with calcium added, heat and humidity. If that doesn’t work within 12-24 hours – or if the bird has already been straining for that long, then we consider collapsing the egg. Once egg is collapsed I usually wait up to 24 hours to see if the shell can be passed by the bird before I attempt to remove it.

Prolapsed cloaca: These cases often have the egg retained in the cloaca or adhered to the cloacal mucosa. Gently remove the egg and then replace the cloaca. If necessary place stitches across the cloaca.

Reptiles:
Reptiles present with two main conditions, preovulatory stasis and postovulatory stasis. Both conditions can present with decreased appetite and decreased activity. Follicles or eggs can be palpated or detected on radiographs and ultrasound. The conditions can be difficult to distinguish unless you can see egg shells present, in which case it is post-ovulatory which is usually a better prognosis.

Preovulatory stasis:
Multiple large follicles are present in the ovary. Some reptiles can resorb these follicles without incident but sometimes they do not and can get very sick. CBC – very inflammatory – high WBC count with toxic change and band cells. These lizards are very sick and should be stabilized as much as possible and then taken to surgery to remove the ovaries.
Post-ovulatory stasis
These lizards initially will have increased activity as they are looking for an appropriate place to lay their eggs. They are often anorexic because the eggs are a space occupying mass. There may be an issue where they can’t lay the eggs due to shell abnormalities, infection, calcium deficiency, abnormal shaped eggs. However, they can also hold the eggs for a long time – so it can be difficult to tell when they are egg bound vs gravid. If they are still in good condition and active, they may lay the eggs on their own if they are given proper substrate. If left too long, they may be too weak to pass the eggs or there may be an underlying problem and these lizards will need to go to surgery.
ROUTINE PROCEDURES IN EXOTIC PET MEDICINE
Dennilyn Parker, DVM, MVetSc, ABVP (Avian)

Rabbits:
History Questions
- Diet: Proper diet is mostly hay. Timothy hay is better than alfalfa hay, but any hay is better than none at all. Pellets aren’t necessary but can be offered in small amounts to easily assess if they are eating. Treats should consist of fresh green leafy vegetables. Any diet changes need to be made slowly.
- Other animals in the house: cats are dangerous to have in the house with rabbits due to Pasteurella. Ferrets can be dangerous and stressful.
- Spay/neuter status – important for females to be spayed, less important for males unless they have unwanted behaviours

Vaccination: RHDV vaccine is now available in Canada. Growing importance

Ferrets:
History questions:
- Diet – strict carnivores but are usually fed a kibble diet. Kibble is high in carbohydrates and is probably linked to the high level of insulinoma that we see in ferrets. Can increase animal protein by adding meals of canned cat or kitten food. Whole prey diets/raw food – can be good but difficult for average owner to do safely. Grain-free diets seem to be linked to urinary stones. Ferrets decide what is food early on and can be difficult to switch their diet as adults.
- Photoperiod: Prolonged exposure to long daylength is likely a predisposing factor in developing adrenal disease. Asking about lighting schedule in the room they are located and if the sleeping areas are dark.
- Ferret-proofing: Ask if the home is ferret-proofed to see if they owner is aware of the risks to ferrets. This means limiting access to foam/rubber objects. Some ferrets will go under the couch and rip foam. Ear plugs, foam insoles, doors, laundry baskets

Vaccination: Distemper and rabies vaccine. Most are sold at the pet store as ‘fully vaccinated’ but that just means one distemper at 6 weeks of age

Parrots:
History questions:
- Any other pets, especially cats
- Diet – what do you feed and what do they eat
- Do they fly
- Parrot proofing the house – this looks different if bird is flighted vs not
- How much sleep do they get and how is managed? Do they cover the cage vs have a sleeping room/closet for the bird

Commonly use sedation for physical exam, blood collection, beak and nail trims. Butorphanol and midazolam – 1-4 mg/kg of each IM. Smaller birds usually need a higher dose. Can be used intranasal as well but at a slightly higher dose. Midazolam easily reversed. Common procedures include beak and nail trims as well as trimming flight feathers when requested.

Reptiles (mostly Bearded Dragons):
History questions:
- Heat
- Humidity
• Diet
• Ultraviolet light
• What do feces/urate look like and how often

Common procedures include enemas, ultrasound.
COMMON EXOTIC PET EMERGENCIES
Dennilyn Parker, DVM, MVetSc, ABVP (Avian)

Rabbits:
When rabbits present on emergencies I will start with a brief history from the owner and then go back and get a full history once the rabbit has been assessed. Quick history questions:

- Presenting complaint
- How long has the rabbit been sick and how often is it handled
- Is it a female and if so is she spayed
- Diet
- Is there a cat in the house
- And recent medications including topicals – on the rabbit or anyone else the rabbit grooms

For the physical exam I will start with a distant exam and the prepare everything I need before I start. If there is respiratory distress I put it in an oxygen tank and consider adding midazolam. With respiratory distress check the nostrils and make sure they are clear – obligate nasal breathers. I also check thoracic compliance – this will be reduced with thymomas or eve heart disease.

Palpate abdomen – stomach can be palpated – check size and consistency of the stomach – large is a concern. Soft and doughy is normal, full like a blocked cat’s bladder – concerned about GI obstruction, painful – concerning. Palpate bladder – stones would be painful. Blood from the back end – female concerned about uterine cancer and/or bladder stone. Blood in the urine could mean stones in the ureters and/or urethra.

Emergency panel:
- Temperature 38.5-39.5°C
- Blood glucose 4-8 mmol/L
- PCV 34-43%
- TP 50-75 g/L
- Lactate 6.9 +/- 2.7 mml/L

Normal lactate runs higher in rabbits than in other mammals.

Ferrets:
Start the same way as with a rabbit – quick history from the owner about presenting complaint and duration. Ferrets have fewer true emergencies. Check mentation and get a blood glucose – foot pad prick is the easiest. 4-8mmol/L is normal. Run this before offering any food or giving any oral dextrose. If blood glucose is low, ideally start an IV or IO and give a bolus of 50% dextrose slowly to effect. Usually 1-2ml is enough and the ferret will magically wake up. If given too fast there may be a release of insulin and these cases are harder to manage. If you can’t get IV access you can apply corn syrup to the gums – or tell owners to do this if they are on their way to you, but only do this as a last resort. It will make a diagnosis harder if they are coming to you and usually too much is given.

If the blood glucose is fine, check respiration and chest compliance. Mediastinal masses can be seen with lymphoma in ferrets.

Abdominal palpation: looking for GI foreign body or enlarged bladder. Young ferrets are probe to GI foreign bodies. They will not vomit unless the foreign body moves into the pylorus. These ferrets are usually painful, and radiographs or ultrasound are indicated. If the bladder is enlarged, it is usually due to one of two reasons. Adrenal disease in a male ferret can cause an enlarged prostate which wraps around the urethra causing an obstruction. The other likely cause would be bladder stones, not common in ferrets unless they are on a grain-free (or high legume) diet. Ferrets can be difficult to pass a urinary catheter in, but not impossible. I will
usually perform a cystocentesis under sedation to empty the bladder and give the ferret more time to run diagnostics and place a urinary catheter.

**Birds:**
History is important but birds can hide illnesses. Not all owners will pick up on the subtle signs. It is difficult for owners to detect weight loss unless they are handling the bird regularly.

Questions to ask:
- Presenting complaint and duration
- Is there a cat in the house
- Any abnormal food offered – chocolate or avocado for example
- And nesting behaviour and/or eggs laid
- Any Teflon pans used in the house/self-cleaning oven

**PE:**
Birds that are sick in the exam room are usually very sick. Birds that are sick at home and acting normal in the exam room are often also very sick. Keep handling to a minimum. Start with a distant exam. If there are signs of respiratory distress – put those birds directly into oxygen.

When I have a bird I am concerned about, I will pick them up and feel the body condition and the coelomic space and then put then in the bird in the scale. This will tell me how chronic the illness is and how critical they are. If the coelomic space is enlarged and/or convex this would indicate and enlarged organ, egg, coelomic fluid – all of which will collapse the air sacs and decrease the amount of air the bird can move through their lungs. This also puts them at higher risk when handling.

Once I assess how stable the bird is I will give them breaks as needed – long enough breaks for them to fully recover from handling.
What are the most common clinical signs of lower urinary tract disease in cats?
Diagnosis of lower urinary tract disease must be founded in careful clinical examination to
differentiate lower urinary tract disease from disease originating in other body systems and to
attempt to refine the list of differential diagnoses. Cats with lower urinary tract diseases can
have pollakiuria (increased frequency of urination), stranguria (difficulty urinating), hematuria
(bloody urine), or inappropriate urination (urinating outside the litterbox). These are not
pathognomonic for lower urinary tract disease but provide a strong indication that investigation
of the lower urinary tract is required.

Urine sample collection and handling
Urine sample collection is a core component of the diagnosis of urinary tract disease. While
seemingly straightforward, care must be taken when collecting and handling urine samples, and
interpreting culture results.
The sample type depends on various factors, particularly the planned testing. If culture is
planned, cystocentesis should be collected unless there is a (very rare) medical
contraindication, because it reduces the risk of contamination. Free flow or catheterized
samples are higher risk for contamination with distal urinary tract inhabitants, as well as
contaminants from the perineum, vagina, and skin. As a result, false positive results from
contaminants are of greater concern. Cystocentesis is a viable option for the vast majority of
patients, but may not be a viable option for some. That creates a situation where a suboptimal
sample (e.g. free flow urine) has to be used, and care must be taken when interpreting results.
Free flow and cystocentesis samples can have good agreement under certain circumstances.(7)
If collected with care, processed by the laboratory within a few hours and when only a single
bacterial species is present at high (>100,000 CFU/ml) levels, agreement with cystocentesis is
excellent. The main practical limitation is the time from collection to processing, as that can be
quite prolonged in most veterinary practices. Less confidence in the relevance of culture results
is present with prolonged delays. When multiple bacteria are present and/or CFU/ml levels are
low, there is increasing likelihood that isolated bacteria are contaminants. More confidence can
be had in free flow and catheterized samples that yield no bacterial growth than those that do,
particularly when uncommon pathogens, common skin contaminants, mixed growth or low
colony counts are identified.
Because of the common delay from specimen collection to processing, specimen handling is
critical, as is consideration of potential impacts of delays on results. If specimens are to be
processed for culture immediately, they should be kept at room temperature. If a delay of
greater than 30 minutes is anticipated, specimens should be refrigerated. If at all possible,
specimens should be processed by the laboratory within 24 hours of collection. In human
medicine, specimens are generally discarded if they cannot be processed within 24 hours.
Unfortunately, it is likely that many veterinary specimens encounter long delays because of the
nature of the collection and submission process. Ways to improve culture results with delays
have been investigated but clear guidance is lacking. Urine preservative tubes are commercially
available. However, it is important to scrutinize the products, since some urine preservative
tubes are designed to kill bacteria (preserving cytology and biochemistry) while some are
designed to facilitate bacterial survival. Use of commercial tubes has been inadequately
investigated in veterinary medicine. Boric acid has been evaluated as a preservative for canine
and feline urine.(8) However, positive effects on bacterial recovery after shipping were not
identified. Collecting urine on swabs placed in Amies transport medium has also been
investigated, but no benefit over storage in a sterile tube was identified after 48 hours.(9) Use of
swabs also precludes quantitative culture, something that can be useful for interpretation of results. Given the limited evidence supporting ways to optimize stored specimens, care should go into the timing of collection (e.g. as close to lab pickup/submission time as possible). Delays, like specimen type (e.g. cystocentesis vs free flow) should be considered when interpreting results.

**Urine culture interpretation**

It is important to remember that culture results are a tool, not a definitive diagnostic test. Results need to be interpreted in the broader context of the patient and the relevance of results need to be considered. Positive urine culture results do not necessarily indicate clinical relevance or causation, so results must be scrutinized. Assessment of the level of bacterial growth (e.g. $10^5$ CFU/ml), whether a single or multiple organisms are detected and the specific organisms is required to determine likely clinical relevance. Low CFU/ml counts do not necessarily mean contamination, but they indicate a greater need to assess whether results might have been influenced by sample type (e.g. cystocentesis vs free flow) or sampling handling (e.g. time, temperature). True polymicrobial infections, where more than one bacterium is playing a primary clinical role, are probably uncommon to rare. Polymicrobial infections should also result in added scrutiny about the potential for contamination and consideration of whether one or all of the organisms that were isolated may be irrelevant. Assessment of the different species and their typical role in urinary tract disease is an important step. For example, if *E. coli* and *Enterococcus faecium* are isolated in a sample, it is most likely that *E. coli* is the causative agent and *Enterococcus* can be ignored.

Antimicrobial susceptibility data are important for determining treatment plans, but do not play a role in deciding the clinical relevance of an isolated bacterium. Susceptibility results only need to be considered once a bacterium is deemed clinically relevant and in need of systemic antimicrobial therapy. Antimicrobial resistance is not directly related to virulence. Resistant bacteria are simply more difficult to treat in situations where treatment is indicated. Therefore, the presence or absence of resistance does not impact determination of whether a bacterium is clinically relevant.

**Cytology**

Urine cytology is a core test that should be performed in every investigation of urinary tract disease. Cytology can be important for identifying a variety of abnormalities, including hematuria, pyuria, casts, crystals, abnormal cells and microorganisms (bacteria, fungi and rarely parasite ova).

**Urine dipstick and USG**

Cost effective, quick and easy to perform, standard dipstick analysis and determination of urine specific gravity are core components of urinary tract assessment. Leukocyte esterase or nitrate tests, present on some dipsticks marketed for use in humans, are not reliable in samples from animals and should not be used to detect pyuria. Cytological examination of the sediment should be used.

**Imaging**

Medical imaging remains the gold standard for urolith detection. Most uroliths are radiopaque and visible on survey radiographs. Ultrasound can detect less radiopaque uroliths (such as urate or blood solidified) but it is challenging to detect urethroliths. Detection of urolithiasis increases when radiography and ultrasonography are used together. Imaging can also be used to identify other acquired (e.g. bladder wall mass, abnormal renal architecture) or congenital (e.g. ectopic ureter) abnormalities.
Cystoscopy consists of inserting a rigid or flexible endoscopy in the urethra and bladder of a cat. It allows macroscopic observation of the mucosa and the luminal anatomy of the lower urinary tract as well as the performance of biopsies, or lasering (lithotripsy, ablation of ectopic ureter or polyps). This test can be performed in female cats only (male urethra too narrow and tortuous). Cystoscopy is indicated in cases of unexplained recurrent urinary tract infection, stranguria, hematuria or urinary incontinence. In male cats or when cystoscopy is not available, the injection of contrast agent in the urethra and bladder via a urethral catheter helps evaluate the urethra and bladder (urethral mucosa infiltration, uroliths, clots). In some referral centers, CT-Scanner is used to better appreciate the anatomy of the urinary tract. Intravenous injection of contrast agent can help determine the path of the ureters.

**Diagnosis of feline idiopathic cystitis (FIC)**
The diagnostic of FIC is performed by excluding other lower urinary tract diseases such as uroliths, tumors, and urinary tract infections. Abdominal radiographs +/- ultrasound are helpful to rule out the presence of cystoliths. Urethral uroliths are more challenging to see on ultrasound because of the superimposition of the pelvic bone. In some cases, a cystourethrogram is necessary to evaluate the urethra. Because of the severe pollakiuria, it is difficult to collect urine in cats with FIC.

**Urolith composition identification**
The most common uroliths in cats are composed of calcium oxalate or struvite (magnesium ammonium phosphate) accounting for ~90-95% of all feline uroliths around the world. Urate uroliths are the next most common (~5% of feline uroliths). Other types (cystine, xanthine, dried solidified blood) are less common. The radiographic appearance of uroliths is the gold standard for predicting urolith composition. Each stone type has a characteristic shape, opacity, and surface contour on radiographs. However, there are some exceptions to the rules. If the urolith’s nidus is of a different type than the shell, the shape, contour, and opacity can be misleading. Urinalysis (crystal types, and urine pH) is also helpful. The presence of urinary tract infection does not suggest the presence of struvite uroliths in cats as they are usually sterile. An Android and iOS app is available that incorporates breed, age, and stone prevalence to assist urolith prediction (z.umn.edu/mnurolithapp). Definitive determination of composition can be performed by submission to a urolith evaluation laboratory, something that is readily available for feline uroliths.

**References available upon request**
Update on how to diagnose chronic renal disease in cats?

Serum creatinine concentration (sCreat) and urine specific gravity remain the gold standard for detection of chronic renal disease. However, Symmetric methylarginine (SDMA) has been available since 2015 and has some advantages over sCreat.

Based on partial nephrectomy studies in dogs, at least 75% of nephron mass must be lost in dogs before sCreat increases above the upper end of the reference interval (RI). Seventy five percent loss of renal mass corresponded to approximately a 50%-60% or 35%-45% decrease in renal function based on inulin clearance (one month) or 13 months post-nephrectomy, respectively. Creatinine concentration is influenced by non-renal factors, especially muscle mass. Debilitated patients have falsely low sCreat concentration, whereas well-muscled animals, such as Greyhounds, have higher concentration.

Variations in sCreat are minimal within an individual healthy cat over weeks to months to years. For this reason, a small increase in sCreat within the RI can reflect a significant decrease in GFR in an individual patient. The greatest challenge in the early diagnosis of kidney disease is the steep curvilinear relationship between sCreat and GFR (FIGURE 1). Therefore, a significant reduction in GFR from normal is required before a corresponding increase in sCreat occurs. Meanwhile, in advanced kidney disease, a small change in GFR will have a large impact on sCreat, making sCreat a useful marker of the progression of kidney disease. Therefore, evaluating serial sCreat measurements in an individual animal (trending) can improve the sensitivity of sCreat to detect early kidney disease by looking for increases that likely reflect a worsening of kidney function. A recent study also showed that healthy geriatric cats 9 to 12 years of age had higher total lean body mass, sCreat, and GFR as compared to cats over 15 years of age. For this reason, sCreat may underestimate kidney function in older or thinner cats.

Since 2015, symmetric dimethylarginine (SDMA) quantification has been available to practitioners worldwide through IDEXX. Since only one company offers the test, SDMA has potentially reduced analytical variability. Similar to sCreat, SDMA correlates strongly and inversely with GFR measured by exogenous plasma iohexol clearance in dogs and in cats. Since SDMA is less influenced than sCreat by muscle/body mass and age in dogs and cats, there is less interindividual variability of serum SDMA as compared to sCreat. For this reason, SDMA might be more clinically relevant than sCreat in older or thin animals. Studies in cats suggest that SDMA detects a decrease in GFR before sCreat, when based on established reference limits. In a study of 21 geriatric laboratory cats with naturally occurring CKD, SDMA was increased an average of 17 months (range 1.5 - 48 months) before sCreat was increased above the upper RI of 186 µmol/L. In the same study, using a decrease of greater than 30% GFR from the median of healthy controls, SDMA had 100% sensitivity, 91% specificity, 100% negative predictive value, and 86% positive predictive value. Because SDMA demonstrated an exponential relationship with GFR (similar to sCreat), SDMA had limited ability to detect early decline in renal function. However, no studies yet have
evaluated the potential benefit of trending SDMA as has been discussed for sCreat. The upper RI for SDMA has been questioned. At the current upper limit of 14 µg/dL, the sensitivity was found to be 90% and specificity 50% for detecting > 40% decrease in GFR. Using a cut off ≥ 18 µg/dL increased the specificity to 83% for detecting kidney disease without compromising sensitivity. The RI has recently been adjusted for puppies, with the upper limit set at 16 µg/dL. It is not known at this time if the older populations of dogs and cats should have a different RI. Overall, the superiority of SDMA over sCreat has been questioned over time. Although its levels rise earlier than those of sCreat, similar to sCreat, SDMA is a marker of decreased function rather than subclinical kidney injury. Further research is required for the influence of other comorbidities to be established.

Is urinalysis useful for cats with upper urinary tract disease?
Inappropriate USG is the hallmark of renal disease. Normal USG in cats is > 1.035. Although USG < 1.035 is considered abnormal in cats with dehydration or azotemia, concentrating ability is occasionally intact in cats with CKD. Furthermore, inappropriately dilute urine can be due to nonrenal causes, for example, secondary to glucosuria, diabetes insipidus, hypoadrenocorticism, hyperthyroidism, diuretic therapy, liver disease, or electrolyte imbalances. Several parameters can suggest renal disease, including an acidic urine pH, glucosuria, and proteinuria. Although urine pH is usually acidic in patients suffering from acute kidney injury (AKI), it can also be alkaline in the presence of a bacterial urinary tract infection (UTI). Mild to moderate glucosuria may be seen with acute tubular damage. Its presence in the urine combined with normoglycemia is indicative of renal damage and occurs, for example, in Fanconi syndrome, leptospirosis, and primary renal glucosuria. Proteinuria can exacerbate renal injury, thus creating a vicious cycle, and may be pre- or post-glomerular. For this reason, proteinuria needs to be interpreted in light of urine sediment findings. If pyuria, bacteriuria, or hematuria is present, a pre- or post-glomerular origin needs to be ruled out. Proteinuria suggests glomerular damage and can be present in both AKI and CKD. In some cases of renal diseases, proteinuria is secondary to impaired tubular protein reabsorption.

While a small number of epithelial cells in urine is normal, a larger number of epithelial cells typically indicates direct tubular cellular injury, and granular casts occur with ischemic or nephrotoxic tubular insult. The presence of erythrocyte casts should alert the clinician to a likely renal hemorrhage. A large number of calcium oxalate crystals suggests ethylene glycol toxicity.

How to diagnose upper urinary tract obstruction
Upper urinary tract obstruction (UUO) in cats most commonly occurs secondary to obstructive ureterolithiasis (mainly calcium oxalate). Obstructive ureterolithiasis is the cause of 75% of acute kidney injury in cats. Other causes of UUO include ureteral stricture (25%), ureteritis, pyelonephritis/pyonephrosis, ureteral neoplasia, blood clots, dried solidified blood calculi, congenital abnormalities, surgical trauma and retroperitoneal fibrosis. Ultrasonography is frequently used to assess cats with azotemia. Findings such as pelvic dilatation and ureteral dilatation proximal to the location of obstruction are usually seen with UUO and the underlying cause may be identified. However, in some cases the cause of UUO may be difficult to identify on ultrasound. The sensitivity and specific of ultrasound varies depending on the cause of UUO. The sensitivity and specificity of ultrasound to detect ureterolithiasis in cats are 98% and 96% respectively and only 44% and 98% respectively for detection of ureteral stricture.

The degree of pelvic dilatation varies among cats with normal kidney function and those values may overlap with those observed in cats with renal disease (such as CKD), pyelonephritis or UUO, thereby complicating its interpretation. In 2 previous studies, renal pelvic dilations of
>13mm were invariably attributed to UUO, but the majority of obstructed kidneys had pelvisses <13mm and there was overlap with other renal diseases. The presence of ureteral dilation appears to be more reliable in the diagnosis of UUO, but overlapping results still occur in cats with other renal diseases such as pyelonephritis.

A recent study (Lemieux et al) documented that 26% of feline kidneys with UUO had a renal pelvis that measured ≤4mm. Eight percent of kidneys had a pelvis ≤2mm. Median renal pelvis and ureteral diameters were 6.6mm (range 1.1037-37.0) and 3.2 (range 0.0-11.0) respectively. Interestingly, the mean preoperative serum creatinine concentration was significantly higher in cats with a renal pelvis ≤4mm (762umol/l [498-1165]) than in those with a renal pelvis >4mm (409umol/l [333-502]). Renal pelvis size correlated negatively with preoperative serum creatinine concentration (r=-0.52, p=0.0002).

The presence of renal pelvis dilation is an important ultrasonographic sign of UUO, it may fail to occur, or occur later in the process. The degree of pelvic dilation secondary to UUO may be influenced by the degree and the duration of outflow obstruction, urine output and renal tissue compliance against increased pressure within the kidney. For example, a cat with partial UUO, may have a small renal pelvis initially if the condition is acute or if the cat is volume depleted. Renal parenchymal and/or capsular fibrosis associated with CKD may limit renal pelvic dilation and/or renomegaly in obstructed kidney due to decreased compliance of fibrotic tissue despite increased pressure within the renal urine-collection system. For this reason, a UUO cannot be excluded based on the absence of pelvic dilation on ultrasonography. Ureteral dilation may better assess UUO in cats. However, UUO can not be excluded based on the absence of ureteral dilation (possible ureteropelvic junction obstruction). The presence of ureteritis and/or fibrosis may also limit its distension.

In case of equivocal ultrasonographic findings, an antegrade pyelography is considered the most accurate test to diagnose and identify the site of UUO. It consists of injecting a contrast agent directly into the renal pelvis followed by fluoroscopy or radiographs to assess the passage of the contrast through the kidney and ureter.

Percutaneous pyelography consists of injecting contrast agent directly into the renal pelvis. Pyelography allows to somewhat pressurize the urinary system which facilitates identification of ureteral obstructions or tears compared to excretory urography. Also, this technique does not exacerbate AKI, as the contrast is administered post-nephron. While it carries, a minor risk of renal hemorrhage, contrast extravasation, and ureteral rupture, reported, complications are rare.

**Diagnosis of pyelonephritis**

Given the potential severity, accurate and prompt diagnosis is required to institute effective treatment as soon as possible. However, pyelonephritis can be a diagnostic challenge. In particular, differentiating pyelonephritis from progression of chronic kidney disease with concurrent subclinical bacteriuria is difficult. This is particularly true in older cats, where decreasing renal function from non-infectious causes is common, and where subclinical bacteriuria is frequent in that population of older cats with CKD. Positive urine culture alone is not necessarily indicative of pyelonephritis in a cat with AKI or deteriorating CKD. It warrants consideration because pyelonephritis is potentially treatable; however, pyelonephritis is likely markedly overdiagnosed in that population.

The clinical presentation of pyelonephritis is variable but affected cats should have signs that are beyond what can be attributed to AKI or CKD, such as fever, lethargy, abdominal pain, vomiting and diarrhea. A CBC should be performed to look for evidence of an inflammatory response. Culture and susceptibility testing should always be performed. As with cystitis, cystocentesis samples should be used for culture whenever possible. Parallel blood culture may also be useful. Pyelocentesis is ideal but uncommonly performed. When possible, pyelocentesis should be done to collect samples for cytology and culture. Interpretation of susceptibility data
should be based on antimicrobial breakpoints for serum rather than urine concentrations, since renal tissue levels are the key, not urine levels. Imaging is particularly important to determine whether pyelonephritis may be present; however, ultrasound findings can be subtle or non-specific. Potential changes in pyelonephritis include hydronephrosis and hydroureter, but ultrasonographic changes do not need to be present. Pyelectasia can occur from other causes, including IV fluid therapy, so care should be taken not to overinterpret that finding. Ultimately, a combination of clinical signs, physical examination, urinalysis, urine culture and imaging must be considered, and at times, it is hard to determine whether pyelonephritis might be present. In such cases, a course of antimicrobials is reasonable so as not to miss a treatable disease, but also to assess response to treatment as part of the diagnostic process. If appropriate antimicrobials are started and there is no clinical and hematological improvement within 2-3 days, then pyelonephritis is unlikely and other differential diagnoses should be explored.

References


TREATMENT OF INFECTIOUS URINARY TRACT DISEASE
J Scott Weese DVM DVSc DACVIM FCAHS, Alice Defarges, DVM, MSc, DACVIM,

Sporadic bacteriuria cystitis
Optimal empirical choices vary based on the pathogen and resistance patterns in the region. However, amoxicillin is a reasonable first choice in most areas. If amoxicillin without clavulanic acid is not readily available, use of amoxicillin/clavulanic acid is reasonable. Evidence of a need for clavulanic acid is lacking and it may not be necessary, even in infections with beta-lactamase producing bacteria, because of the high amoxicillin concentrations that are achieved in urine. For most cats, especially those without a history of recurrent disease or frequent antimicrobial use, the likelihood of resistance is low. Trimethoprim-sulfonamide is another first tier option but may be associated with greater adverse event concerns and is difficult to recommend over amoxicillin or amoxicillin/clavulanic acid. Fluoroquinolones are excellent drugs for E. coli and achieve high drug levels in urine. However, they are not typically required. As higher tier, broad spectrum drugs, they are best reserved for situations where first line options cannot be used. They may be useful in situations where patient behaviour, owner lifestyle or owner compliance necessitate once daily treatment. Similarly, cefovecin is an effective drug for cystitis but it is a higher tier drug (3rd generation cephalosporin) and provides a longer duration than is necessary. It is a good option for situations where oral treatment is not possible but should be reserved for cases where it is truly required.
The recommended duration of therapy is 3-5 days. The short end of that dosing period may be optimal, but veterinary research is currently limited.
There is no indication for measures beyond monitoring of clinical signs. Provided the full course of antimicrobials is administered correctly, there is no evidence that intra- or post-treatment urinalysis or urine culture is indicated in the absence of ongoing clinical signs of cystitis.

Recurrent bacterial cystitis
Previous guidelines supported long durations (4 weeks) of antimicrobials for recurrent cystitis. However, recurrent cystitis encompasses a broad patient range, some that develop repeated and relatively uncomplicated infections that likely respond quickly to antimicrobials and others that have marked bladder pathology that complicates treatment. In human medicine, several studies support short-course therapy for acute and recurrent bacterial cystitis.
If empirical antimicrobials are initially prescribed, drug selection should be approached as for sporadic bacteria cystitis, and re-assessed when culture results are available, considering both in vitro susceptibility and patient response to initial treatment.
Long-term therapy is not automatically warranted for recurrent bacterial cystitis. Short (3-5d) durations should be considered for cases where re-infection seems to be occurring. Longer courses (7-14d) may be reasonable in persistent, and potentially relapsing, infections, if factors that inhibit response to antimicrobials, such as bladder wall invasion, are suspected to be present. There may also be value in using drugs that are more active in tissue (e.g. fluoroquinolones, TMS, cephalosporins, versus penicillins or nitrofurantoin) if bladder wall involvement is thought to be a reason for poor response to treatment.

Subclinical Bacteriuria
The goal of management is to treat disease. This is not necessarily synonymous with the presence of bacteria in urine. Subclinical bacteriuria is defined as the presence of bacteria in urine as determined by positive bacterial culture from a properly collected (i.e. cystocentesis) urine sample, in the absence of clinical evidence of signs of lower urinary tract disease. Subclinical bacteriuria is not uncommon, even in individuals with no known predisposing factors. Rates of 3-4.6% in healthy cats, 6.1% of healthy cats > 6 years of age, 10-13% of cats > 7 years of age, 18% in cats with CKD, 58% in cats with neurogenic bladder, 25-35% in cats with...
subcutaneous ureteral bypass, 4.3-5% in hyperthyroid cats, 13% of male cats after catheterization because of urethral obstruction and 77-100% of cats with perineal urethrostomy or prepubic urethrostomy. Importantly, there was no association between subclinical bacteriuria and survival or renal disease progression in a study of cats with CKD or in a study of older cats. In humans, there is abundant support for not treating asymptomatic bacteriuria (the human analogue of subclinical bacteriuria), even in most compromised patients. While bacteriuria rates are high in various populations (e.g. diabetics, the elderly, patients with paralysis), treatment guidelines such as Infectious Diseases Society of America Guidelines for the Diagnosis and Treatment of Asymptomatic Bacteriuria in Adults do not recommend treating asymptomatic bacteriuria in almost all patient groups. Exceptions are patients undergoing transurethral resection of the prostate and patients that will be undergoing urologic procedures that result in mucosal bleeding. Screening and treatment of pregnant women is recommended; however, this has recently been questioned because while an association between untreated bacteriuria and pyelonephritis was identified, the low burden of pyelonephritis and potential adverse effects of antimicrobials may not justify universal treatment. Treatment is specifically not recommended for pre-menopausal, non-pregnant women, those with diabetes, older individuals in the community, elderly institutionalized individuals or individuals with spinal cord injuries. Thus, even in what would be considered high-risk populations, treatment of asymptomatic bacteriuria is discouraged and intensive measures are used to reduce the treatment of asymptomatic bacteriuria. These efforts are typically focused on antimicrobial stewardship from an antimicrobial resistance standpoint, but reduction in unnecessary treatment is also desirable because of cost, adverse effects of antimicrobials, and lack of evidence that treatment improves outcome in almost all patient groups. While treatment might eliminate the current bacteriuria event, recolonization often follows. A systematic review in humans concluded that while bacteriuria may be eliminated in the short-term, the effect is not sustained and re-colonization is common, leading to no impact on overall morbidity or mortality. Further, two studies have reported significantly higher bacteriuria recurrence rates in women treated for asymptomatic bacteriuria compared to untreated controls. Treated women also had higher rates of antimicrobial resistance.

There are few indications for culture of urine from animals without evidence of lower urinary tract disease. Culture of urine from animals with no evidence of urinary tract disease should not be performed when there would be no indication to treat based on a positive culture result. This includes patients with comorbidities, particularly those with disease such as hyperadrenocorticism and diabetes mellitus, where subclinical bacteriuria is not uncommon. Bacterial concentration cannot differentiate subclinical bacteriuria from bacterial cystitis. Subclinical bacteriuria is differentiated from bacterial cystitis by the absence of clinical signs and not by the bacterial load. Heavy growth on quantitative culture data (e.g. >100 000 CFU/ml) can be present in animals with subclinical bacteriuria and there is no evidence that high CFU counts indicate a greater risk of disease development. Treatment of subclinical bacteriuria is rarely indicated and is discouraged. In animals where it is unclear whether clinical signs are attributable to cystitis, a short course (e.g. 5 days) of antimicrobials as recommended for sporadic urinary tract infections could be considered. If there is no clinical response, antimicrobials should be discontinued, as an infectious process is unlikely.

Treatment of animals with pyuria or other cytological abnormalities but no clinical signs of lower urinary tract disease is not recommended. Previous guidelines supported treatment of animals with no clinical signs but cytological evidence of inflammation (pyuria). However, treatment of pyuria in humans in the absence of clinical evidence of UTI is not recommended. There is currently no evidence in veterinary medicine that would indicate a different approach to that taken in humans.
Treatment of subclinical bacteriuria caused by plaque-forming (*Corynebacterium urealyticum*) and urease producing (e.g. staphylococci) could be considered because of their associations with encrusting cystitis and struvite urolith formation, respectively.\(^{23-26}\) Because of the potential difficulties in treating these conditions, consideration of a single short course of treatment, as described for sporadic cystitis, could be considered after confirming that bladder wall plaque or uroliths are not present. However, it is unknown whether this is a necessary or effective approach.

**Urolithiasis**
Urolithiasis, including struvite urolithiasis, is not typically associated with infection in cats. However, concurrent urolithiasis and bacterial cystitis can occur. Antimicrobials are indicated when there is concurrent evidence of bacterial cystitis in a cat with urolithiasis, something that can be a challenge to determine with confidence given the overlap between clinical signs of urolithiasis and bacterial cystitis. If an infection is deemed likely based on clinical signs, urinary cytology and ideally urine culture, treatment as per sporadic cystitis is indicated. It might be reasonable to use the longer end of recommended durations (e.g. 5-7 days) given the potential for more bladder wall inflammation and damage compared to routine sporadic cystitis, but clear data are lacking.

**Pyelonephritis**
Given the potential severity, accurate and prompt diagnosis is required to institute effective treatment as soon as possible. Whenever pyelonephritis is suspected, culture and susceptibility testing should always be performed. As with cystitis, cystocentesis samples should be used for culture whenever possible. Parallel blood culture may also be useful. Imaging is particularly important to determine whether pyelonephritis may be present. Interpretation of susceptibility data should be based on antimicrobial breakpoints for serum rather than urine concentrations, since renal tissue levels are the key, not urine levels.

Immediate treatment is indicated, using an antimicrobial with good activity against Gram negative Enterobacteriaceae. If ascending infection is suspected, urine culture results obtained for diagnosis of cystitis might be the basis of initial therapy. If the upper UTI results from hematogenous spread, initial therapy should be based on cultures of blood or the infected site, whenever available. Otherwise, empirical therapy with a drug typically effective against Gram negatives should be chosen.

Fluoroquinolones are the main recommendation based on their spectrum and efficacy for tissue-associated infections. While a higher tier drug, the seriousness of disease and need for confidence in efficacy against *E. coli* in tissue (as opposed to urine, where high levels of many antimicrobials can be achieved) support empirical use of this drug class. If oral treatment can be used, marbofloxacin, orbifloxacin and pradofloxacin are options. Concerns about retinopathy with enrofloxacin preclude its use at doses that would be recommended.

If parenteral treatment is needed, a third-generation cephalosporin (e.g. ceftiofur, cefotaxime) can be used. This can be transitioned to an oral fluoroquinolone or oral third generation cephalosporin (i.e. cefpodoxime) once oral medications are tolerated, or a different drug can be chosen based on subsequent culture and susceptibility results. Parenteral treatment is only needed if the patient cannot tolerate or is unlikely to properly absorb oral medications. There is little evidence to guide duration of treatment. Treatment of 4-6 weeks has been widely used in veterinary medicine, with no data. However, the recommended duration of therapy for acute bacterial pyelonephritis in children is 7-14 days.\(^{27}\) For adult humans, 10-14 days for beta-lactams or trimethoprim-sulfamethoxazole and 7 days for ciprofloxacin are recommended.\(^{28}\) There is no reason to suspect that a longer duration would be necessary for dogs and cats. In the absence of veterinary-specific data, the 10 to 14 days of treatment has now been recommended, with the potential that 7-10 days may be equally effective.
For more details about drug doses and dosing regimens, please reference to OVC’s Firstline guideline app, or web resource: https://app.firstline.org/en-cvma/clients/552-ontario-veterinary-college

References
TREATMENT OF NON-INFECTIOUS URINARY TRACT DISEASE
Alice Defarges, DVM, MSc, DACVIM

When should we intervene on uroliths?
Nephroliths and ureteroliths: recurrent infection, worsening azotemia, or suspected obstruction.
Cystoliths or urethroliths: recurrent infection, clinical signs, or suspected obstruction.

What treatment approach should we use for uroliths?
Surgical intervention to remove upper urinary tract uroliths is associated with high morbidity and mortality rate. Surgical removal of uroliths via cystotomy and urethrostomy has been associated with complications such as urine leakage, wound dehiscence, bleeding, stricture formation, and incomplete urolith removal in 20% of canine patients alone. More than nine percent of recurrent lower uroliths are suture-induced. Traditional cystotomy is associated with a complication rate of 37 to 50%. Therefore, minimally invasive urolith removal should be considered before surgical intervention. Medical dissolution should always be attempted if struvite uroliths are suspected.

Voiding urohydropulsion (VUH) consists of removing small bladder calculi by inducing voiding while the dog or cat is positioned vertically (female dogs or cats) or laterally (male dogs). It can be successfully performed in male or female dogs and female cats. It is not recommended in male cats, because of the risk of urethral obstruction (narrow urethra). Stone size is one of the most important criteria in deciding if VUH can be considered as an option. Female cats may be able to void uroliths <3mm. Before attempting VUH, clinicians should try to catheterize the urethra first to evaluate its size. If a 9-Fr urinary catheter can be passed through the urethra, 3-mm uroliths should be able to be voided. For VUH, the patient is placed under general anesthesia. In the author’s experience, epidural anesthesia facilitates VUH. However, it can delay the patient’s discharge. The urethra is catheterized and the bladder is distended with saline. The urinary catheter is then removed and the patient is positioned vertically. The bladder is gently agitated so that gravity causes the uroliths to fall into the trigone. Bladder palpation increases intravesicular pressure and helps maintain a strong urine stream as small stone fragments are voided. General anesthesia is mandatory for this procedure, and the bladder can rupture if the patient does not have a relaxed, unobstructed urethra. The procedure is repeated until no calculi or stone fragments are passed. Radiography, with or without positive or negative contrast, or cystoscopy can be performed after the procedure to confirm complete urolith removal. VUH can also be used to obtain a few small uroliths for analysis to determine whether medical dissolution is an option. In our experience, using cystoscopy rather than digital urethral catheterization for bladder filling in female dogs and cats can be very helpful. Empiric broad-spectrum antibiotics should be given during the procedure and for 3 to 5 days afterward. To minimize discomfort during urination, nonsteroidal anti-inflammatory drugs or opioids are administered for 1 to 4 days as needed and based on clinical signs.

Intracorporeal lithotripsy consists of fragmenting the uroliths with a Ho:YAG laser fiber with endoscopic guidance. The patient is placed under general anesthesia for routine cystoscopy. Once the uroliths are visualized with the cystoscope, a small-diameter, flexible laser fiber is guided through the working channel of a flexible or rigid cystoscope/ureteroscope. The tip of the fiber is placed in direct contact with the surface of the urolith, and laser energy is transmitted to the urolith via the fiber. The energy is fired straight and absorbed in <0.5mm of fluid, making it safe to fragment uroliths in tight locations (urethra). Uroliths are typically fragmented until the pieces are small enough to be voided using VUH or manually extracted from the bladder using a stone retrieval basket. Most of the published studies evaluated the use of lithotripsy in dogs. Complete urolith removal is typically achieved in 100% of dogs with urethroliths, 83% to 96% of female dogs with cystoliths, and 68 to 81% of male dogs with cystoliths. The median procedure time is shorter for dogs in which intracorporeal lithotripsy is performed only in the urethra compared with dogs in which uroliths were fragmented in the bladder. The procedure time is
longer for males than for females. Complications are typically minor and short-term, including urethral swelling, edema, and mild hematuria, which are medically managed. Bladder perforation is possible if the laser is not manipulated by an experienced operator. Cystoscopy and lithotripsy can be used in female cats to fragment and remove uroliths from the bladder and urethra. However, because of their narrow and tortuous urethra, male cats are not candidate for cystoscopy. Another option for uroliths that are too large for VUH but small enough to fit down the urethra with gentle traction is stone basket retrieval under cystoscopic guidance. Typically, the size of the stone is the limiting factor. Basket retrieval can be considered in female cats with uroliths <3mm. Estimating the urethral diameter based on the weight is really difficult in dogs. Percutaneous cystolithotomy (PCCL) can be an alternative in animals in which transurethral lithotripsy is a challenge (male and small female cats) or if lithotripsy is unavailable. Use of a newer minimally invasive approach, PCCL, has been reported in 27 dogs and cats, with excellent complete stone removal rates (96%) and dramatically shorter procedure times (total procedure time: 40 to 75min) than those seen with transurethral lithotripsy, regardless of patient sex, size, stone number, or species. For this procedure, a single small incision (approximately 1 to 1.5cm) is made in the abdominal cavity just over the apex of the bladder, once the bladder is palpated digitally, a stay suture is used to hold the bladder up to the incision while a laparoscopic screw-tip cannula or port is inserted into the bladder at the apex. A small, rigid, cystoscope is inserted through this port into the bladder, and uroliths are identified and removed with a stone-retrieval basket through the cannula. The best visualization is achieved with a full bladder, therefore, we usually flush fluid into the bladder via a urinary catheter in the urethra. The bladder is typically not exteriorized, if the uroliths are very small, a suction device can be placed into the port and the uroliths can be flushed/suctioned out of the port in a retrograde manner. If the uroliths are larger than the port, they can either be fragmented by lithotripsy or manipulated through the small incision with the stone basket. Concurrent lithotripsy is rarely needed. After calculi are removed from the bladder, the entire urethra is examined with a rigid cystoscope (female dogs) or a flexible ureteroscope (male dogs) to ensure that all uroliths have been removed. A basket retrieval device can be used to remove any remaining urethral calculi through the working channel of the endoscope. In patients with embedded urethral uroliths, laser lithotripsy can be performed. PCCL can also be used to aid in gaining access to the ureters for diagnostic or therapeutic purposes and for resection of bladder polyps or diagnostic evaluation of the bladder and proximal urethra in dogs that are too small for retrograde cystoscopy.

In the past decade, the surgical placement of a subcutaneous ureteral bypass (SUB) has become the standard of care for cats with upper urinary tract obstruction. The SUB system consists of two small tubes implanted in perpetuity in the renal pelvis and bladder, respectively, and connected via a shunting port. Urine then flows through the placed tubes by-passing the ureteral obstruction for direct delivery of urine from the renal pelvis into the bladder.

When and what do we start to decrease proteinuria in cats with chronic kidney failure?
Cats develop proteinuria secondary to their CKD (later stage), due to tubular dysfunction and/or glomerulosclerosis. Proteinuria is associated with a poorer prognosis in cats. Several studies suggested a long-term benefit of ACE inhibitors (benazepril) to decrease proteinuria in cats with CKD. It is generally recommended that cats with renal proteinuria with a urine protein creatinine ratio >0.4 on at least 2 repeated measurement should be treated (0.25-0.5mg/kg benazepril orally q24h). Careful monitoring is required because ACE inhibitors may result in decreased GFR and subsequent worsening of renal function. Because of the negative effect on GFR, ACE inhibitors are not recommended for late stage CKD patients or those in uremic crisis.
Telmisartan is an angiotensin receptor blocker that is licenced in Europe for treatment of proteinuria and in the US for hypertension in cats and has been found to be more effective than
benazepril for treating proteinuria. Telmisartan is an appropriate first line therapy for feline renal proteinuria.

**When and what do we start to decrease hypertension in cats with chronic kidney failure?**
Systemic hypertension is common in cats with CKD (20-65%), but the exact pathophysiologic relationship is unknown. Systemic hypertension can have other deleterious effects such as retinal hemorrhage and detachment, neurologic and cardiac impairment. Elderly cats, particularly those with renal impairment should be routinely screened. If their blood pressure is higher than 200mmHg and or evidence of target organ damage is seen, such as retinal hemorrhage, blood pressure should be rechecked to rule out white coat hypertension. Azotemic cats with blood pressure persistently higher than 160mmHg are candidates for treatment. Amlodipine is documented to be an effective treatment for hypertension in the cat and is currently the most common medication prescribed at 0.625mg/cat q24h. Telmisartan is also an effective anti-hypertensive medication in cats and is approved for this indication in cats (United States: 1.5mg/kg q 12h for 14 days, then 2mg/kg q24h for hypertension) as well as an anti-proteinuric (UK and Europe 1mg/kg daily). For all anti-hypertensive therapy, blood pressure should be rechecked within 7-10 days after initiating treatment. The use of telmisartan may change the long-term monitoring strategy (in comparison to amlodipine) as if used at the labeled dose, dose reduction may be likely. When assessing blood pressure in CKD patients, it is important to note that Gabapentin, which is 100% renally limited and merits dose reduction in CKD patients, may have a significant effect on blood pressure measurement.

**When should we treat hyperphosphatemia?**
The kidneys are the main route of phosphorus excretion and as kidney function declines, hyperphosphatemia develops. Hyperphosphatemia contributes to renal secondary hyperparathyroidism, tissue mineralization, and progression of CKD. Hyperphosphatemia has been identified as a predictor of progression of CKD in cats. Therefore, controlling phosphorus intake through diet and phosphate binders is important in CKD management. The 2022 International Renal Interest Society (IRIS) treatment guidelines suggest maintaining plasma (or serum) phosphorus concentrations in cats with CKD between 0.9-1.5 mmol/l. Although a target of 1.6mmol/l for patients with stage 3 CKD, and <1.9mmol/l for patients with stage 4 CKD was deemed more realistic. If a renal diet is initiated and phosphorus is still elevated after 4-6 weeks, then a phosphate binder is recommended (goal: keep the phosphorus in the low normal range). Because these medications bind the phosphorus in the food, it is critical they are given with each meal. Several phosphate binders are available: aluminium hydroxide (30-100mg/kg/day), calcium carbonate (90-150mg/kg/day) and lanthanum carbonate (30mg/kg/day). Therapy is titrated to effect. Palatability limits the amount that can be administered. Serum calcium should be monitored when a calcium-containing phosphate binder is used. If hypercalcemia develops, an alternative binder (not containing calcium) should be used.

**When and what do we feed cats with chronic kidney failure?**
There is strong evidence that feeding a veterinary therapeutic renal diet can prolong survival and reduce risks of uremic crises in cats with CKD. It is controversial if and/or protein should be restricted in cats with CKD as it may contribute to protein-energy wasting. Several renal diets provide lower phosphorus concentrations and higher protein concentrations. This may be beneficial in some cases to maintain lean body mass. If a cat with CKD develops proteinuria, it is recommended to reduce total dietary protein by 25-50%, depending on the severity of proteinuria, azotemia, and clinical signs. Dietary restriction typically results in a decline in proteinuria within 1 month. If the cat’s diet is already low-to-moderate in protein concentration (near AAFCO minimum recommendation for adult maintenance), it may be preferable to allow medical management time to decrease proteinuria before further restricting dietary protein.
Twenty to 30% of cats with CKD develop hypokaliemia. It can result in muscle weakness, polyuria, polydipsia, and constipation. There is a wide range of potassium currently available in veterinary diets. If hypokaliemia is present, it may be helpful to offer a higher potassium-containing diet or oral potassium supplementation with either potassium gluconate or potassium citrate.

**How can we stimulate the appetite of cats with CKD?**

Mirtazapine has become commonly used as an appetite stimulant in cats. In one placebo-controlled, double-blinded cross-over clinical trial, Mirtazapine was an effective appetite stimulant and resulted in significantly increased appetite and weight. It also appeared to decrease vomiting in cats with CKD. Transdermal administration is also available and has been demonstrated to achieve both appropriate serum levels, appetite stimulation and weight gain in cats. Ghrelin agonist capromorelin (Elura, 2mg/kg orally daily) is not available yet in Canada. It provides additional opportunities to address appetite in cats with CKD by targeting the pathophysiology of appetite regulation. Administration of capromorelin resulted in increased food intake and weight in laboratory cats.

**Management of feline idiopathic cystitis (FIC)**

Cats with idiopathic cystitis have moderate to severe signs without a readily identifiable cause. Various medications have been used but none of them shown a better success than placebo. The goal is to decrease pain and to decrease potential irritation or spasms in the urethra. Gabapentin and Buprenorphine make cats more comfortable and may prevent inappropriate urination. Steroids were used in the past but did not improve the patients. Non-steroidal anti-inflammatory drugs can be beneficial anecdotally but should be used only in cats with normal kidney function and no imminent risk of obstruction. Urethral relaxant (such as Prazocin or phenoxybenzamine) can be used to decrease urethral spasms, though its efficacy has recently been debated. Because poly-unsaturated fatty acids, glycosaminoglycans, and pentoas polysulfonate can reduce bladder inflammation and restore the normal mucous lining to the bladder, they could be beneficial for FIC. However, there is no strong evidence that those drugs are beneficial for cats with FIC.

The corner stone of the FIC therapy is reducing stress in the cat's environment. Cats' private dishes and toys should be readily available, clean and placed in quiet areas. A variety of private locations for the cat should be provided (different locations, heights, materials, styles). Several litter pans and water bowls should be available. Changing regularly toys and distractions is important to stimulate the cat. Finally, synthetic cat pheromone (Feliway®) are available on the market at veterinarians and pet stores. Dietary modification can help some cats with FIC. There are many different options of veterinary prescription diets formulated with stress-reducing agents. In severe cases, anxiolytics may be beneficial (amitriptyline (Elavil®), or fluoxetine (Prozac®)). However, there is no strong evidence that these drugs improve the outcome of cats with FIC.

**References**


FELINE UVEITIS – IT’S JUST INTRAOCULAR LYMPHADENOPATHY
David J. Maggs BVSc (Hons), Diplomate ACVO

Summary
The uvea contains familiar tissues and cell types (lymphocytes, smooth muscle, and blood vessels, for example), is inflamed by familiar antigens (infectious agents, neoplasia, auto-antigens) and reacts with the 5 cardinal signs of inflammation seen elsewhere (heat, pain, swelling, etc.). And yet it can be a very confusing disease. This lecture aims to provide aids to diagnosis and therapy of uveitis by likening it to lymphadenopathy (because it is more similar than it is different) while highlighting differences (because these are helpful). For further information, please refer to a review article from the Journal of Feline Medicine and Surgery upon which these notes are based (2009 Mar;11(3):167-82).

Clinical Signs
Active (acute) uveitis
Uveitis has few pathognomonic signs and these are notably subtler in cats than they are in dogs. Therefore, uveitis in cats often goes undetected by owners and untreated by veterinarians until potentially blinding sequelae such as glaucoma, cataracts, and retinal detachment or degeneration occur. For these reasons, clinicians must maintain a high index of suspicion regarding uveitis in all cats with ocular disease and even those with nonspecific signs such as lethargy, “hiding”, anorexia, or fever.

Uveitis, like inflammation elsewhere, is evident as one or a combination of the 5 cardinal signs of inflammation: heat, pain, swelling, redness, and loss of function:
- **Intraocular pain**: blepharospasm or epiphora; however, cats seem more likely to show subtle and less localizing signs such as lethargy or anorexia
- **Iridal swelling** requires that the eye is examined using a source of magnification (such as the Optivisor®) in association with a bright and focal light source (such as the Finoff transilluminator®) directed very obliquely across the globe. Look for a loss of the normal “texture”.
- **Redness** (scleral injection) can be particularly subtle in many cats.
- **Dysfunction**: Loss of function manifests as breakdown of the blood aqueous barrier (BAB), miosis, corneal edema, and hypotony. Of these, BAB breakdown is pathognomonic. In particular look for **hypopyon** (white blood cells), **hyphema** (red blood cells), and **fibrin, aqueous flare** (albumin and other small proteins) and **keratic precipitates** (white blood cells and inflammatory proteins clumped against the corneal endothelial surface).

Chronic uveitis and its sequelae are evident as **glaucoma** (due to scarring or clogging of the iridocorneal angle), **anterior or posterior synechia** (adhesions between the iris and corneal endothelium or lens, respectively), **phthisis** (globe contracture) or **retinal detachment** (due to contraction of vitreous fibrin). Altered aqueous humor composition and circulation also causes a relative malnutrition of the lens (evident as cataract) and inner cornea (evident as corneal edema, vascularization, fibrosis etc.). **Lens luxation** may occur due to enzymatic lysis or phagocytosis of the lens zonules, secondary to cataract development, or as a sequela to buphthalmos due to secondary glaucoma. Neovascularization of the face of the iris (**rubeosis iridis**) is a pathognomonic sign of subacute or chronic active uveitis. It is one of the signs that is seen more easily in cats than in dogs due to the typically lighter iris color of cats.

Diagnosis
What is a reasonable diagnostic approach to a patient with uveitis?
Having a high degree of clinical suspicion and performing a targeted clinical examination with appropriate ocular diagnostic testing (especially checking for aqueous flare, testing intraocular pressure (IOP), and assessing for miosis) will ensure that uveitis is diagnosed when present. However, detecting uveitis is the beginning of the diagnostic process; not the end. Confirming or eliminating all suspected etiological diagnoses is the essential next step. By conducting an excellent general physical and ophthalmic exam as well as gathering a focused history, the initial goal is to establish whether further diagnostic testing is strongly supported. I do this by categorizing the uveitis as present in a well or systemically ill patient, unilateral or bilateral; exogenous or endogenous; acute or chronic; and as involving the anterior uvea, choroid, or both. An etiologic diagnosis should then be pursued through a diagnostic workup identical to that employed for a cat with lymphadenopathy. Consider CBC, Biochemistry, urinalysis, serology, chest and abdominal imaging, etc. as appropriate for the following agents.

**Etiology**
The known causes of endogenous anterior uveitis in cats are expanding but still too few to explain the majority (~ 70%) of cases.

**Infectious agents as a cause of uveitis**

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<tr>
<th>Viral</th>
<th>Bacterial</th>
<th>Parasitic</th>
<th>Fungal/Algal</th>
<th>Protozoal</th>
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<tr>
<td>FIP</td>
<td>Bartonella spp.</td>
<td>Cuterebra</td>
<td>Cryptococcus neoformans†</td>
<td>Toxoplasma gondii</td>
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<tr>
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<td>Mycobacterium spp.</td>
<td>Larval migrans</td>
<td>Histoplasma capsulatum†</td>
<td>Leishmania spp.</td>
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<td>Ehrlichia spp.¥</td>
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<tr>
<td>CAV</td>
<td>Leptospira</td>
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<td>Aspergillus spp.</td>
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*Via immunosuppression or oncogenesis
† Chorioretinitis predominates
¥ Seroprevalence data only; no clinical evidence

**Treatment**
Treatment of anterior uveitis must be tailored to the individual case based on proven or suspected cause, severity, anatomical location, chronicity, and presence of systemic or other intraocular disease. Regardless, some general therapeutic guidelines are possible. Optimal treatment relies upon identification and removal or reduction of the causative antigen; however, this is rarely possible. Additionally, all patients with uveitis need their intraocular inflammation controlled rapidly and completely, since it is painful and produces vision-threatening sequelae. Thus, immunomodulating drugs form the mainstay of therapy for uveitis. The major decisions are therefore which immunomodulating drugs should be given, via what route and, at what dose.

**Immunomodulatory therapy**
*Corticosteroids* are highly potent, available in topical or systemic forms, relatively inexpensive, generally well tolerated by cats, and can be administered at anti-inflammatory or immunosuppressive dosages. For these reasons, they are commonly used for uveitis. Their systemic use should be reserved until a definitive cause responsive to corticosteroids has been found or, failing this, until causes known to be worsened by glucocorticoids have been
adequately eliminated. In particular, the systemic mycoses must be adequately eliminated as potential causes. Likewise, patients in which lymphoma is possible and which would benefit from a multidrug chemotherapeutic regimen should not be treated with systemic corticosteroids alone. By contrast, topical administration of corticosteroids may be employed safely even when an infectious or neoplastic cause might prevent systemic administration of the same drugs. This is possible because systemic effects are insignificant with short-term topical application. It is possible that topically administered corticosteroids may alter cytologic findings and so, if safe, their use should perhaps be delayed until after ocular centesis is performed. Topical corticosteroids should never be used in the face of corneal ulceration because they can be associated with rapid worsening of the ulcer due to superinfection, collagenolysis, local immunosuppression, and delayed wound healing. Prednisolone acetate (1% or 0.125%) and dexamethasone (0.1%) will penetrate intact corneal epithelium and reach the anterior uveal tract. Hydrocortisone (as found in many combined antibiotic–corticosteroid ophthalmic preparations) does not penetrate intraocularly and should not be used. The frequency of application should be tailored to the severity of the uveitis; starting as frequently as q 2 hours and tapering as a clinical response is noted. When safe, corticosteroids should be administered systemically for posterior uveitis and when more significant immunomodulation is necessary, or when corneal ulceration prohibits their topical use. Typical doses of prednisolone range from 1 mg/kg q 12 hours when notable inflammation is present to 0.5 mg/kg once daily when a more moderate anti-inflammatory effect is desired. As with topical corticosteroids, dose and dose frequency of systemically administered glucocorticoids should be carefully reduced based entirely upon clinical evidence of waning disease. In cats with acute or subacute uveitis, this can be fairly rapid. Animals with chronic idiopathic (immune-mediated) uveitis require slow tapering (perhaps a halving of dose or dose frequency every 2-3 weeks), with the expectation that inflammation may return below a critical dose. In these patients, returning to the previously effective dose will be necessary. Some cats will suffer herpetic recrudescence when receiving corticosteroids, regardless of route.

Compared with corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs) are not immunosuppressive, and may be more expensive, sold in smaller volumes (as topical ophthalmic solutions), and sometimes unavailable in ointment form. These limitations must be borne in mind for cats with uveitis; however, they may be preferred over corticosteroids in patients with diabetes or other endocrinopathies in which corticosteroid use may not be wise. They can also be administered systemically instead of corticosteroids when systemic infectious disease is suspected or proven, or until lymphosarcoma is eliminated as a differential consideration. And they may be given in conjunction with a topical steroid. As such, they may make an excellent choice for initial control of inflammation while likely causes are being ruled in or out. The same general comments regarding dose frequency and route made for corticosteroids apply equally to NSAIDs.

**Iridocycloplegic agents**

Parasympatholytic drugs such as atropine have multiple favorable actions in eyes with uveitis and form a critical component of treatment. These drugs paralyze the parasympathetically innervated iris sphincter and ciliary body muscles causing mydriasis and cycloplegia, respectively. The effects of pupil dilation are numerous and important. Pupil dilation reduces leakage of vascular elements into the aqueous humor by causing radial blood vessels within the iris stroma to “concertina” upon themselves (thus providing a physiological tamponade); decreasing iris surface area (from which inflammatory mediators and vascular components originate); reducing uveal vascular endothelial permeability; and by reducing chances and consequences of posterior synechiation. However, “bunching” of the iris in the periphery does increase the chance of anterior synechia and potentially obstruction of the iridocorneal angle.
Cycloplegia reduces ocular pain but also increases resistance to aqueous outflow. Therefore, pupil dilation and cycloplegia are desirable in all cases of uveitis except those where secondary glaucoma is present or likely. The effect of mydriasis upon IOP can be tested by a single application of the short acting drug tropicamide followed by tonometry when the pupil is fully dilated. If IOP is increased by tropicamide, atropine should not be administered. If atropine is initiated, IOP should be rechecked regularly and application discontinued if IOP increases above normal. In cats, atropine should be applied as an ophthalmic ointment rather than a solution because it is bitter and passage down the nasolacrimal duct can cause violent salivation and frothing that is harmless but disturbing to the cat and its owner. Atropine should be applied to effect. Since cycloplegia cannot be observed, the pupil is used for monitoring dose. Depending on the severity of uveitis, once to twice daily application for the first day or two may be needed to open the pupil. Subsequently, once to twice weekly application will often keep the pupil mydriatic. Posterior synechia will not be resolved with atropine and will prohibit use of pupil size as an indicator of drug efficacy. However, atropine should still be administered to patients with synechia since the analgesia resulting from cycloplegia should not be affected and is still desirable.

**Monitoring and Sequelae**
Prompt specific treatment of uveitis with tapering of therapy based upon reduction of clinical signs may result in some sequelae but these are usually mild and should be static. If they are not, this suggests chronic or recurrent uveitis and further investigations and treatment are necessary. Classic sequelae include corneal fibrosis, cataract, or posterior synechia. None of these changes should result in pain or, unless severe, vision disturbance. By contrast, more severe, unrecognized, persistent, or recurrent uveitis frequently results in a blind and sometimes painful globe. The most common sequelae (and their prevalence in cats) include cataracts in 20-36% of eyes, lens luxation in 11-18%, glaucoma in 16-46% and enucleation in 29%. Many patients experience more than one of these sequelae. For these reasons, frequent and careful monitoring of a patient with uveitis is essential. This should be performed as for patients with immune-mediated disease elsewhere with gradual tapering of medications and re-examination at doubling intervals presuming there is improvement; more often if there is not. Re-examination and tapering of medications should be continued until there is complete resolution of every clinical sign of active uveitis. I believe that tonometry is the most sensitive test with which to monitor uveitis during treatment because subtle hypotony (sometimes only relative to the contralateral eye) can continue long after other more overt signs have normalized. Continued treatment of these patients may prevent or delay development of sight-threatening ocular complications.

*References available from the author on request.*
FELINE CORNEAL DISEASE - ULCERS, EOSINOPHILIC KERATITIS, AND SEQUESTRA
David J. Maggs BVSc (Hons), Diplomate ACVO

Introduction
Probably due to the cat’s susceptibility to feline herpesvirus (FHV-1) and that organism’s predilection for corneal tissue, cats seem to be commonly presented for corneal disease. In addition to acute, chronic, and recurrent ulcers, that require a completely different diagnostic and therapeutic approach than they do in dogs, cats are affected by 2 other rather enigmatic diseases that are not seen in dogs – corneal sequestra and eosinophilic keratitis. These 3 corneal diseases will be the focus of this session.

Corneal ulcers
The feline cornea is relatively infrequently exposed to many of the known causes of ulcers in dogs. Due to their ocular anatomy and physiology, along with their lifestyle, cats seem less predisposed to ectopic cilia, distichia, entropion, trichiasis, foreign bodies, indolent ulcers, KCS etc. In fact, the lack of alternate etiologic diagnoses has led to the truism that “corneal ulcers in cats are due to FHV-1 until proven otherwise”. I certainly use this assumption when examining a cat with corneal ulceration. My goal then is to adequately eliminate other known causes with a thorough examination of eyelid anatomy and function, and of the conjunctival fornices. I begin with assessing palpebral response (and judging the completeness of the blink elicited by stimulation of the medial and lateral canthi. Then using a focal light source and some magnification, carefully assess the eyelids (especially the margin) and the conjunctival fornices. Prior to applying the fluorescein stain, I aim to categorize any corneal changes – in particular, is there a corneal defect (stromal loss), a loose lip of non-adherent corneal epithelium, corneal edema (blue and “fluffy”), malacia (gelatinous or “oozing”), blood vessels (pay particular attention to the limbus) or corneal stromal infiltration with white blood cells (yellowish green). Following application of fluorescein stain, I aim to categorize the staining pattern as:

1. Sharply demarcated region of stain in the floor of the ulcer only (superficial and likely already in the process of healing)
2. Walls and floor of the ulcer are stained (deep stromal ulcer)
3. Walls of the ulcer only stained (Descemetocele)
4. Ulcer floor only stained but with a hazy margin like a halo (likely a non-adherent epithelial lip)

My singular diagnostic and therapeutic goal at the end of this initial examination is to categorize the ulcer as simple or complicated. Simple ulcers are present less than 7 days (based on history and corneal vascularization) and are superficial (based on assessment of staining pattern and stromal loss when viewed by transverse illumination). To be categorized as complicated, ulcers need lack only one of these two features; i.e., they need to be either chronic or have stromal loss. The saying I have the students remember is:

“Simple ulcers are short AND shallow; whereas complicated ulcers are chronic OR craterous”

I treat simple ulcers by:

1. Eliminating the primary cause (perform a thorough lid and conjunctival exam)
2. Preventing bacterial super-infection (Use a broad-spectrum bactericidal antibiotic)
3. Treating pain (your favorite systemic NSAID/analgesic, and a single topical application of atropine in the exam room)

4. Monitoring self trauma. I usually do not recommend an E-collar unless there is marked evidence of self trauma, since I fear that a collar stresses them more than it helps and therefore exacerbates stress-induced herpetic disease. While cats can and do traumatize their own eyelids, I do not think they traumatize their own cornea often.

5. Recheck inside 7 days and reclassify again as simple or complicated.

Here is a critical difference between ulcer therapy in dogs and cats! If we believe that FHV-1 is the most likely cause of the ulcer and if we do not use an antiviral drug, then we must admit that we have not fulfilled the primary therapeutic goal listed above and have left in place the cause of the ulcer. This is perfectly acceptable therapy as animals with viral infections are often treated in a supportive manner while they are expected to clear the viral infection themselves. However, we should not be surprised if at the 7-day recheck we do not see resolution of the ulcer. In fact, this might make us even more certain that FHV-1 is the likely cause. It is these chronic (now, by definition, complicated) ulcers for which we should consider antiviral therapy.

The most common reasons an ulcer becomes complicated in cats are:

1. They are (and probably always were) primarily infected with FHV-1.
2. They have become secondarily infected with bacteria.

Fortunately, each of these ulcers has a distinct appearance permitting us to diagnose cause of complication far more easily than we can the initiating cause of the ulcer. Herpetic ulcers remain epithelial only, since FHV-1 is not cytolytic in the corneal stroma. In addition, a non-adherent corneal epithelial lip may be present. Although these look identical to indolent ulcers of dogs, I do not use this term as they share none of the same factors involved in causation or pathogenesis. Debridement with a cotton-tipped applicator can be considered at this stage, but it is essential that a grid keratotomy (or any other anterior stromal puncture technique) is not used on chronic non-healing ulcers of cats as this has a high risk of initiating corneal sequestrum formation (see below). These cats must have a Schirmer tear test performed as these ulcers may well be associated with dry eye syndrome. Topical hyaluronate tear replacement is very wise. In addition, an antiviral agent should be considered if the primary cause is believed to be FHV-1. Topically applied antiviral agents can be cytotoxic and poorly tolerated; systemically administered famciclovir given at 90 mg/kg PO BID reaches effective concentrations in the tear film and can be used instead. Other therapy appropriate for an ulcer such as topical antibiotics and control of reflex uveitis via topical application of atropine and potentially systemic administration of a NSAID should be continued. Topically applied atropine should be kept to a minimum as I think many of these ulcers are associated with tear deficiency in cats. Systemic or topical NSAIDs may, and corticosteroids will, exacerbate FHV-1 reactivation from the trigeminal ganglia. Therefore, I limit anti-inflammatory therapy to only systemically administered NSAIDs and, even with them, aim to minimize their use. Sometimes I prefer an alternative analgesic such as buprenorphine if the cat seems unduly painful.

By contrast, ulcers complicated by secondary bacterial infection take on some of the classic appearances of infection. They demonstrate a yellowish-green corneal stromal infiltrate (often along with more severe mucopurulent ocular discharge), malacia (corneal “melting”), worsening corneal edema, and often stromal loss. A hunt for the primary cause should still be conducted and an appropriate antibiotic chosen based on culture and sensitivity and cytology. One study suggests that Mycoplasma spp. can cause exactly this sort of severe deep stromal ulcer. Clinical suspicion of mycoplasmal involvement should be highlighted for the microbiology.
laboratory as they may require special sample collection, transport, or culture and sensitivity testing conditions.

**Eosinophilic Keratitis**

Feline eosinophilic keratitis (FEK) appears as a focal, raised, white-to-pink (or sometimes yellow), corneal plaque resembling granulation tissue. Typically, the lateral cornea is involved initially, but in advanced cases the entire cornea may be involved. Areas of corneal ulceration are also possible. Cases may be uni- or bilateral. Eyelid and/or conjunctival involvement is seen relatively commonly along with keratitis and occasionally alone. Diagnosis is suggested by clinical appearance and confirmed using cytology. The cause is undetermined; however, the condition appears to be due to an aberrant immune response. Cytologic evaluation of scrapings from affected tissue (usually cornea or conjunctiva) reveals neutrophils, eosinophils, and mast cells, along with hyperplastic or dysplastic epithelial cells. Histology may reveal lymphocytes and plasma cells. A link between FHV-1 infection and eosinophilic keratitis has been suggested. In one study, PCR testing of corneal scrapings from cats with cytology-confirmed eosinophilic keratitis revealed 76% (45/59) to be FHV-1 positive. However, PCR performed on tears collected onto a Schirmer tear test strip was negative in 10 cats with cytologically proven eosinophilic keratitis. Maybe this disparity reflects the difference in sampling methods between studies. Although the role of the virus in initiation or exacerbation of this disease has not been definitively determined, anecdotally some patients with this syndrome improve with antiviral therapy alone, which suggests a causative role. Regardless of whether the virus detected is causative, its presence produces a dilemma, since use of immunomodulatory drugs, especially topical corticosteroids, for treatment of an eye that is potentially infected with FHV-1 and often ulcerated warrants caution.

My preference is to begin treatment of cats with cytologically confirmed FEK with an antiviral agent. My first choice is famciclovir (90 mg/kg PO BID) since this is the most potent antiviral agent available and because it may protect against viral reactivation from the ganglia, especially during the next step in therapy - administration of a systemic steroidal agent. Typically, I recheck the cat in a week or so. If there is dramatic improvement and the owners are compliant, continuation of this regimen may be all that is necessary. More commonly, addition of some form of immunomodulatory therapy is needed. I do not stop the antiviral agent. Topical administration of dexamethasone, prednisolone or, more recently, 1.5% cyclosporine has been described. Alternatively, subcutaneous injection of triamcinolone (0.1-0.2 mg/kg) may be necessary sometimes repeated about 2 weeks later, and typically followed with long-term, tapered topical use of dexamethasone or prednisolone. Antiviral treatment should be continued for as long as there is evidence of active viral replication and certainly while ulceration is present; it should then be ceased without tapering. Immunomodulatory therapy is tapered judiciously as clinical signs improve. Early diagnosis and treatment of recurrences will limit the need for protracted therapy. Some cats require lifelong therapy.

**Corneal Sequestration**

Corneal sequestration is an entity unique to the cat. It is an area of ulcerated, necrotic cornea characterized clinically by gradual progression of a dark (amber, brown, or black) discoloration usually involving the central cornea. Because cats tend not to develop corneal melanosis, we have a saying that “if it is black, and it’s a cornea, and it’s a cat, then it’s a corneal sequestrum”. Prior, and usually chronic, corneal ulceration is common but not always reported. Blood vessels often extend to the lesion and are deep or superficial, depending on depth of the sequestrum. The necrotic corneal stroma may be surrounded by zones of variably intense corneal stromal edema, inflammatory cell infiltration, or both. Sequestra are usually unilateral but may occur bilaterally. Frequently, the eye appears to be causing the cat pain, but some cats display only
minor signs of discomfort. Histologically, the plaque consists of a sequestered, desiccated region of necrotic corneal stroma surrounded by a variable “foreign body–type” inflammatory cell response with extensive granulation tissue development. The characteristic clinical appearance is considered diagnostic.

Evidence regarding the involvement of FHV-1 varies. Experimentally, FHV-1 inoculation (in cats receiving corticosteroids) did result in corneal sequestration. However, the prevalence of detectable FHV-1 in samples collected from cats with sequestra has varied widely in the clinical setting and the link between FHV-1 and sequestra has not been shown to be causative. It seems likely that sequestration is a non-specific response to stromal exposure or damage and that FHV-1 is just one possible cause of this disease. This is borne out in a study by Nasisse et al who reported identification of FHV-1 DNA in 86 of 156 (55%) of sequestra analyzed (compared with only 6% of clinically normal corneas). A lower prevalence of FHV-1 DNA was found in corneas of Persian and Himalayan cats with sequestration, suggesting that other non-viral causes of sequestration such as lagophthalmos, trichiasis, entropion, poor corneal sensitivity/tear production/blink rate, etc. may be operative in these breeds. Therefore, identification and correction of any underlying causes is important wherever possible. In particular, medial canthoplasty with correction of entropion may help reduce corneal exposure from lagophthalmos, and irritation from trichiasis and desiccation in brachycephalic breeds. For the sequestrum itself, lamellar keratectomy is the treatment of choice, especially if the cat is painful or the lesion deep or chronic. Placement of a sliding corneal or conjunctival graft may limit recurrences, which otherwise occur in about 33% of cases. If the cat appears comfortable, medical management may be attempted as sequestra may spontaneously slough over a period of weeks to months. However, perforation during this natural progression of disease is possible and the degree of discomfort often makes this an untenable option. Medical management includes use of prophylactic topical antibiotics (if there is ulceration), topical or systemic antiviral medications, topical mucinomimetic drugs such as hyaluronate, and treatment of reflex uveitis if present (without use of topical or systemic corticosteroids).

References available from the author on request.
WHAT’S NEW IN OCULAR PHARMACOLOGY
David J. Maggs BVSc (Hons), Diplomate ACVO

One drop or two?
Always administer one drop. The conjunctival fornix of the cat and dog can “hold” about 16 µL and a drop is about 50 µL. Therefore, one drop is already ~3-fold too much. Giving a second drop is a 6-fold excess. But it is not just wasteful; each drop causes reflex tearing, so 2 drops still only delivers 16 µL but that 16 µL is washed out by increased reflex tearing.

Ointments or drops?
This is one of the topics that you will find lots of opinions on and rarely does it make much difference. I am often guided by client preference or apparent patient preference. However, the following guidelines may help:

- Solutions are not practical in large animals unless a subpalpebral lavage system is placed.
- Ointments blur vision more
- Multiple ointments can be applied simultaneously but drops should be separated by > 5 mins.
- Ointments increase contact time (and may permit decreased dose frequency)
- Ointments provide lubrication (and are therefore good for patients with entropion or recent eyelid surgery)
- Ointments protect against desiccation (and are therefore good for patients with KCS)
- Solutions, not ointments should be used when corneal rupture is present or likely
- Solutions, not ointments should be used prior to ocular surgery
- Both ointments and drops retard wound healing and should be used at the minimum effective frequency and stopped ASAP.

When I give a systemic drug, does that get to the eye?
When deciding via what route to medicate the eye, there is a tendency to group all “eye diseases” together. In fact, the eye is composed of a variety of tissues that vary greatly:

- Drugs penetrate through the cornea and sclera very poorly
- Some ocular tissues are vascular and some are avascular
- Some ocular tissues are “behind” the blood-ocular barrier.

Systemically administered drugs typically reach only vascular tissues (eyelids, conjunctiva, parts of the iris, ciliary body, optic nerve, and retina, the choroid, and orbital structures. There are 2 exceptions to this rule: a) inflammation causes a (temporary) breakdown of the blood ocular barriers and b) a few drugs cross the intact blood-ocular barrier and are released into the aqueous or vitreous humors (e.g., chloramphenicol, fluconazole) or reach meaningful concentrations in the tears (e.g., famciclovir, doxycycline).

As a rule, orbital, adnexal, conjunctival, and intraocular disease can be treated via the systemic route but corneal disease cannot.

Which eye drops/ointments penetrate inside the eye?
The cornea is a trilaminar sandwich composed of a hydrophobic epithelium, hydrophilic stroma, and hydrophobic endothelium. Very few topically applied drugs penetrate well through this barrier. Even those that are specially formulated to breach that anatomic barrier become rapidly
diluted by tears (especially in painful eyes with epiphora), absorbed by conjunctival capillaries and taken away from the eye, or are lost down the nasolacrimal duct or onto the face over the eyelid margin. Therefore, even for drugs which do cross the cornea only a small percentage of the topically applied drug enters the eye, and that is rapidly diluted in the aqueous humor before being “washed” out of the eye through the iridocorneal angle. Even those drugs that penetrate very well do not reach clinically meaningful concentrations in tissues behind the lens. Therefore, diseases of the posterior segment (or orbit) should always be treated with systemically administered drugs. Corneal penetration of topically applied drugs is increased in the presence of ulceration.

The following drugs are categorized according to how well they penetrate the eye across an intact cornea following topical application:

<table>
<thead>
<tr>
<th>Class</th>
<th>Penetrates well</th>
<th>Penetrates poorly</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiviral</td>
<td>Trifluridine</td>
<td>All others</td>
</tr>
<tr>
<td>Antibiotic</td>
<td>Fluoroquinolones</td>
<td>All others</td>
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<td></td>
<td>Chloramphenicol</td>
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<tr>
<td>Corticosteroids</td>
<td>Prednisolone</td>
<td>Hydrocortisone</td>
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<td></td>
<td>Dexamethasone</td>
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<tr>
<td>NSAIDs</td>
<td>All</td>
<td>None</td>
</tr>
<tr>
<td>Glaucoma medications</td>
<td>All</td>
<td>None</td>
</tr>
</tbody>
</table>

Some golden rules of ocular pharmacology
These preceding topics lead to some golden rules of ophthalmic pharmacology:
1. Always administer one drop
2. Always leave 5 minutes between drops of a different type
3. Always work “up” in viscosity when applying two or more different drops or ointments to the same eye
4. Drugs required in high concentration in the cornea or conjunctiva usually are best administered by frequent topical application (but no more frequently than 5 minutes)
5. Drops are quickly diluted and eliminated from the eye by tears. This can be overcome by increased frequency of application (but no more frequently than 5 minutes)
6. The cornea is a trilaminar (lipid-water-lipid) sandwich across which many topically applied drugs do not cross
7. Even those topically applied drops that cross the cornea do not penetrate to tissues caudal to the anterior uvea; posterior segment disease must be treated via the systemic route
8. Increased “dose” of topically applied drugs may be achieved by increasing drug concentration in the topical preparation (within physiologically acceptable limits); increasing the frequency of application; slowing absorption (increasing contact time)
9. Systemic absorption of drugs from the conjunctival sac following topical application is rapid and may result in notable blood concentrations.

What ophthalmic drugs should I stock in my pharmacy?
Obviously, no answer to this question is correct for every practice but here are some basics I could not manage without for canine and feline practice. The list is compiled without consideration of availability and price which vary so frequently and widely nowadays.
Antibiotics
Triple antibiotic ointment and solution
Ofloxacin solution
Compounded cefazolin solution (see below)
Doxycycline tablets/oral suspension

Ant-inflammatory/Immunomodulatory agents
Neopolydex ointment and solution
Prednisolone acetate 1% suspension
Diclofenac solution
Cyclosporine ointment and suspension
Tacrolimus suspension
Oral prednisone/prednisolone
Oral NSAIDs

Anti-inflammatories

Antivirals
Idoxuridine solution
Cidofovir (compounded) solution
Famciclovir tablets

Tear substitutes
Hyaluronate solution
Petrolatum ointment

Diagnostics
Fluorescein strips
Proparacaine
Tropicamide

Other
Atropine ointment and solution
Serum

Compounded cefazolin

1. Remove 2 ml from a 15-ml bottle of artificial tear solution and discard.
2. Reconstitute a 500-mg vial of cefazolin with 2 ml of sterile water.
3. Add entire 500 mg of the reconstituted cefazolin (2.4 ml) to the bottle of artificial tear solution.
Final concentration = 33 mg/ml (3.3% solution).
Shelf life: 28 days.
Keep refrigerated.

References available from the author on request.
THE PATIENT’S EYE IS RED – WHAT SHOULD I DO NEXT?
David J. Maggs BVSc (Hons), Diplomate ACVO

Introductory Philosophy
Hyperemia of the surface of the globe or so-called “red-eye” is not only one of the most common reasons for a client presenting their animal for veterinary care, but potentially one of the most critical. As veterinary ophthalmologists, some of the most worrisome cases we see are those where ocular redness was misinterpreted as “just a bout of allergic conjunctivitis” or “he must have got some dust in his eye” and thereby serious and potentially blinding diseases such as ulcerative keratitis, uveitis, or glaucoma were missed. The goal of this lecture is to heighten awareness of the differential diagnoses that may cause ocular hyperemia and to define methods to differentiate these.

Client Education
Clients should be advised to always bring in pets with reddened eyes as a matter of urgency. It is worth explaining to them that your goal is to differentiate conjunctivitis (an annoying disease) from uveitis (a very painful and potentially blinding disease with sometimes even fata causes) and glaucoma (a second blinding and painful eye disease with genetic implications and in which preventative therapy of the contralateral eye is critical.

Ocular Blood Vessels are a Diagnostician’s Best Friend
In many ways, ocular blood vessels are the diagnostician’s best friend since they “always” go to where the problem is. That is, if there is superficial irritation (irritation of the conjunctiva or superficial cornea) then superficial blood vessels will become hyperemic. However, if inflammation involves deeper structures – uveitis, glaucoma, scleritis, or deep (stromal) keratitis - then deeper (episcleral) blood vessels become engorged. Thus, clinically differentiating superficial conjunctival vessels from deep episcleral vessels changes the diagnosis, diagnostic testing necessary, treatment, and prognosis.

Clinically Relevant Anatomy and Physiology
For the purposes of this session we will categorize blood vessels overlying the sclera into 2 distinct and clinically useful classes: deep or episcleral and superficial or conjunctival blood vessels. In the normal animal, the blood vessels in the bulbar conjunctiva are so fine that the conjunctiva appears almost transparent permitting the white sclera to be seen through it. The palpebral conjunctiva is normally a pale pink but can become more obvious with hyperthermia or excitement, and should be approximately the same color as other mucus membranes. Bulbar conjunctival vessels extend right up to the limbus. Episcleral vessels – although larger – are usually not prominent when seen through subconjunctival tissues. The exception is some brachycephalic individuals, particularly dogs, in which one or two obvious episcleral blood vessels are sometimes seen in normal, uninflamed eyes. Episcleral blood vessels supply the intraocular structures via the uveal tract and therefore “dive” through the sclera at the iris root – a millimeter or two behind the limbus. The importance of these anatomical facts will become more obvious when we discuss some of the means to differentiate deep from superficial blood vessels.

Mechanisms of Ocular Hyperemia
In addition to physiologic vasodilation due to hyperthermia, there are (at a mechanistic level) two common ways a blood vessel becomes hyperemic or “injected”.

1. Vasodilatation due to release of inflammatory mediators (i.e., “inflammation”)
2. Hydrostatic engorgement due to decreased venous return.
Inflammation. The release of vasoactive mediators at a specific site acts locally to cause (among other things) dilation of the blood vessels that supply that site. Therefore, this mechanism for hyperemia dictates that a reasonably “focused” vasodilatation should ensue. That is, if there is uveal inflammation, and release of vasoactive factors within the uvea, then the deep or episcleral blood vessels that supply the uveal tract should become injected. By contrast, conjunctival inflammation should incite only conjunctival vessel hyperemia. This strict rule breaks down somewhat with more significant or major inflammation. Insults such as uveitis that are severe enough to induce episcleral congestion will sometimes also produce some “innocent bystander” hyperemia of the overlying and smaller conjunctival blood vessels; however, the inverse is unlikely. Therefore, satisfying yourself that episcleral congestion is not present is the most critical decision whenever “red eye” is a presenting sign.

Hydrostatic congestion. Blood vessels terminating (in the case of arterioles) or originating (in the case of venules) in the conjunctiva and uvea share a common pathway through the orbit to and from the major vessels of the head and neck. Therefore, orbital disease can cause enlargement (“injection”) of deep and/or superficial ocular vessels via hydrostatic pressure (decreased venous return) and via local inflammatory effects on these vessels en route to and from the eye. Therefore, all eyes with hyperemia should be examined for evidence of altered globe position (strabismus, enophthalmos, or exophthalmos) and - so long as there is no risk of globe rupture - should also be retropulsed.

Differentiation of deep episcleral and superficial conjunctival hyperemia
Conjunctival vessels are superficial, small (fine), branch frequently, move easily with gentle pressure from a cotton-tipped applicator or by lateral motion of the upper eyelid, extend to the limbus, and blanch within seconds after application of 1 drop of a topical vasoconstrictor such as dilute (1%) phenylephrine. By contrast, episcleral vessels are larger, branch less, appear to “stop short” of the limbus, and blanch more slowly, if at all, with topical vasoconstrictors.

Potential Clinical Diagnoses in Reddened Eyes Since superficial vessels indicate superficial disease and deep vessels indicate deeper disease, it is possible to compile a list of potential likely causes of red eye in association with deep vascular injection (Orbital disease, deep keratitis, uveitis, glaucoma, or – rarely – scleritis) or superficial vascular injection (blepharitis, conjunctivitis, or superficial keratitis). This list guides diagnostic testing and ensures that painful, vision-threatening or potentially life-threatening diseases are not written off simply as conjunctivitis. The only confusion in this list is brought about by the principle of “innocent bystander” inflammation discussed earlier. Subtle (early) glaucoma, orbital disease, or uveitis can cause only mild conjunctival hyperemia before they progress to a stage where they cause episcleral hyperemia.

Diagnostic Tests for “Every” Red Eye
The following is a brief outline of the diagnostic tests that should be considered for all cases of reddened eye.

Retroillumination is a simple but extremely useful technique for assessment of reddened eyes. A focal light source held close to the examiner’s eye and directed over the patient’s nose from at least arm’s length is used to elicit the fundic reflection. Each eye is illuminated equally and the fundic reflex is used to assess and compare pupil size, shape, and equality. Some general rules help interpret retroillumination findings:

- Conjunctivitis – never associated with anisocoria
- **Uveitis** – often associated with miosis
- **Glaucoma** – often associated with mydriasis

**The Schirmer tear test (STT)** should be performed on all reddened eyes but especially those in which there is mucoid discharge. The only exception is those with an obvious deep ulcer in which this test may be unsafe. Normal STT values for dogs are > 15 mm in 60 seconds. However, STT values in normal cats range widely (3-32 mm; mean = 17 mm in 60 seconds) and are more difficult to interpret than in dogs.

**Tonometry** or measurement of intraocular pressure (IOP) is essential in every reddened eye except those at risk of rupture. Its use will permit differentiation of the two major, vision-threatening conditions in which red-eye is the hallmark feature – uveitis (in which IOP tends to be low) and glaucoma (which is defined by elevated IOP) – from conjunctivitis (in which the IOP will be normal). Across large populations, normal canine and feline IOP is reported as 10-25 mmHg. However, some variation occurs. Comparison of IOP between right and left eyes permits application of a reasonable rule of thumb that IOP should not vary between eyes of the same patient by more than ~20%. Perhaps the most important role for tonometry is the monitoring of progress of these diseases and the titration of medications needed.

**Aqueous flare** occurs as a result of breakdown of the blood-ocular barrier with subsequent leakage of proteins into the anterior chamber. Therefore, is a pathognomonic sign of uveitis and must be performed in every reddened eye. It is best detected using a very focal, intense light source (the small circular aperture on the direct ophthalmoscope works well) in a totally darkened room. The passage taken by the beam of light is viewed from an angle. In the normal eye, a focal reflection is seen where the light strikes the cornea. The beam is then invisible as it traverses the almost protein- and cell-free aqueous humor in the anterior chamber but becomes visible again as a focal reflection on the anterior lens capsule and then as a diffuse beam through the body of the normal lens. If uveitis has allowed leakage of serum proteins into the aqueous humor, then these cause a scattering of the light as it passes through the anterior chamber. Aqueous flare is therefore detected when the beam of light is visible traversing the anterior chamber.

**Application of fluorescein dye** to the cornea should be routinely used in all reddened eyes to diagnose corneal ulcers. It should be performed after all other parts of the exam are completed so as not to alter the STT result or affect visualization of other structures.

**Retropulsion** of the globe is a simple but useful method for investigating orbital disease. This is performed by applying gentle digital pressure to both globes through closed eyelids. The resistance to retropulsion and the resilience with which the globes “spring” back against the retropulsive force are subjectively assessed. Retropulsion of the globe in a variety of directions may further localize orbital masses or outline smaller masses that would be missed by direct caudal retropulsion only. This should not be done in eyes at risk of rupture.

*References available from the author on request.*
OPHTHALMIC EMERGENCIES
David J. Maggs BVSc (Hons), Diplomate ACVO

What Constitutes an Ophthalmic Emergency?
Ophthalmic emergencies are an important part of general practice for a number of reasons: Clients tend to perceive ocular changes as urgent because of the importance humans place on perfect vision; ocular disease can be extremely painful and readily noted by owners; and there is a narrow “window” of opportunity to save sight in some diseases. There are three questions that clients can be asked by telephone to assist in defining true ocular emergencies:

1. **Is the eye painful?** As a rule, painful eye disease should be seen more urgently than non-painful conditions. Blepharospasm and epiphora are common signs of ocular pain.

2. **Are there any obvious colour changes?** Conjunctival and episcleral hyperaemia (“red eye”) and discoloration of the cornea, especially oedema (blue) or cellular infiltration (yellow/green) are often indicators of more serious eye disease.

3. **Is vision affected?** Visual loss tends to be associated with more serious ocular disease in veterinary patients.

Obviously, there is a long list of ocular diseases that fulfil at least one of these requirements; many more than we could go through in one session. Instead, I have chosen a few examples that represent the true diversity of ophthalmic emergencies – some surgical and some medical diseases; diseases that involve the eyelid, orbit, cornea, or intraocular structures, and each with a different set of presenting signs.

**Globe Proptosis**
Globe proptosis usually occurs subsequent to crushing injuries sustained during animal fights or car accidents and is always an emergency. Although prompt replacement of the globe is essential, initial assessment must include particular attention to thoracic and CNS trauma. Poor prognostic indicators for the proptosed eye include proptosis in a cat or non-brachycephalic dog, hyphema, facial fractures, visible optic nerve damage, avulsion of > 3 extraocular muscles, or globe rupture. Although emphasis has been placed on the prognostic importance of pupil size, large variations are seen. The most favourable prognostic indicators are presence of vision, a direct PLR in the proptosed eye, or a consensual PLR from the proptosed to the normal eye. Unless there is obvious ocular rupture or visualization of the optic nerve, the globe is usually replaced in an attempt to salvage at least a cosmetically acceptable and ultimately non-painful eye, even if it is unsighted.

As immediate first aid, the cornea should be protected from desiccation and abrasion by liberal application of an ophthalmic antibiotic ointment without corticosteroids. Provided that there are no contraindications, a systemic dose of corticosteroids may assist in management of shock, and periocular inflammation. After a complete physical assessment, the patient should be placed under brief general anaesthesia and the ocular surface sterilized with 1:50 povidone iodine solution. Clipping of eyelid hair is contraindicated as these end up in the conjunctival fornix once the globe is replaced. The eyelid margins are usually extensively rolled in, and so should be everted until the margin is identified. A muscle or spay hook is useful for this. Gentle outward and rostral tension on the upper and lower eyelid margins, along with retropulsion of the globe will then usually result in reduction of the proptosis. If there is marked resistance to this or if finding lid margins is difficult immediately perform a lateral canthotomy to reduce tension and allow better exposure. The globe should be retained in the orbit and the cornea protected by a temporary tarsorrhaphy. I prefer 4-0 silk with some small soft stents (small IV drip line cut in half is ideal). The tarsorrhaphy should be left open a small amount at the medial canthus to permit...
topical application of medications. The canthotomy should be closed in two layers as for lid lacerations.

Postoperative management includes topical antibiotics (without corticosteroids), topical atropine and systemic anti-inflammatory medications. An E-collar is essential. Systemic antibiotics may be necessary to limit the risk of retrobulbar infection. The tarsorrhaphy sutures should be monitored for “gapping” or irritation of the cornea. If this occurs they should be removed (and sometimes replaced). Permanent removal of the sutures is possible when normal globe position within the orbit returns and adequate blink reflex is regained. Assessment of consensual PLR from the proptosed to the normal eye can be assessed through the medial gap in the tarsorrhaphy and does not require suture removal. Visual loss is usually due to stretch or avulsion of the optic nerve or the globe’s vascular supply. Attention should also be paid to tear production as subsequent keratoconjunctivitis sicca (KCS) is seen frequently. Enucleation is recommended for unresolved pain in a blind eye or phthisis resulting in entropion or exposure keratoconjunctivitis.

**Corneal Ulceration**

A corneal ulcer begins with loss of corneal epithelium. With progression, especially in the presence of collagenase enzymes, corneal stroma becomes involved. Ultimately, corneal perforation and intraocular involvement with visual loss are possible. All ulcers should be initially assessed by asking are they “simple” (superficial) or “complicated” (deep). In both cases, the most important treatment is identification and removal of the inciting cause.

**Treatment of superficial uncomplicated corneal ulcers.** Supportive therapy consisting of broad-spectrum topical antibiotics (usually triple antibiotic), prevention of self-trauma, and sometimes cycloplegia (and pupil dilation) for pain control is usually successful. A recheck examination should always be scheduled inside 7 days. Uncomplicated ulcers should be healed at this recheck, while superficial ulcers that have become complicated must be identified at this recheck. Antiviral agents are indicated for treatment of feline herpesvirus keratitis, which is the most common cause of ulcers in cats. Application of a broad-spectrum topical antibiotic is also necessary in these cases. Topical corticosteroids are totally contraindicated for the treatment of all corneal ulcers.

**Treatment of deep complicated ulcers.** Because corneal thickness is only 0.6-0.8 mm, a good rule of thumb is “if an ulcer seems deep, then it probably is!” Ulcers involving only epithelium will rarely have noticeable “lips” or depth. As soon as stromal involvement is clinically significant, transillumination will identify an obvious depression in the corneal surface. Although all of the rules for treating superficial ulcers still apply, more aggressive diagnostic and therapeutic management is also required. Cytology, culture and sensitivity, alternate antibiotics, topical application of serum, treatment of uveitis, or surgery may all be indicated.

**Serum.** Corneal malacic (“melting”) arises as a result of collagenase production by bacteria, especially gram-negative organisms, and damaged host cells. Anti-collagenase products are used in hope of inhibiting/reducing corneal melting. Serum has been promoted as a preferred product. In addition to its broad-spectrum anti-collagenase properties, benefits are presumed to arise from numerous growth factors contained in serum. A venous blood sample is collected aseptically and allowed to clot in a redtop tube. After centrifugation, serum is separated and stored in a sterile multidose vial or commercially available eye-drop container ([http://www.medi-dose.com/catalog/liquid/steridropper.asp](http://www.medi-dose.com/catalog/liquid/steridropper.asp)) and can be applied to the eye as needed (q 30-60 minutes for a rapidly melting corneal ulcer). Serum should be stored in the refrigerator and
replaced every 7 days. It can be saved frozen for at least 3 months. The serum need not be from the affected individual but should be from a safe done with respect to infectious disease.

**Surgery.** Ulcers that are rapidly progressive, have obvious areas of melting, or are deeper than half corneal thickness, are likely to benefit from conjunctival grafting. Conjunctival grafts provide mechanical support, a continuous blood supply, and an immediate source of fibroblasts for stromal healing. If the owner is unwilling to be referred for conjunctival grafting, a partial temporary tarsorrhaphy may be useful. Although third eyelid flaps do provide a “bandage” which reduces desiccation and frictional irritation of the cornea, they also prohibit application of topical medications and monitoring of progression. By comparison, a temporary lateral tarsorrhaphy is equally easy to perform, provides adequate corneal protection, and allows medication and monitoring of the ulcer.

**Acute Congestive Glaucoma**

Acute congestive glaucoma occurs with elevated intraocular pressure (IOP). Patients may be presented with blepharospasm, epiphora, episcleral injection, mydriasis, corneal oedema and decreased or absent vision. *Inflammatory changes can be surprisingly mild and often overlooked in cats unless IOP is measured.* Acute congestive glaucoma should be treated as an emergency since vision loss appears to be dependent on magnitude and duration of IOP increase. Acute congestive glaucoma should be treated as an emergency since vision loss appears to be dependent on magnitude and duration of IOP increase. Multi-drug therapy is recommended since a synergistic response may be afforded by combining differing drug actions and routes of application. The classic therapy combined 3 drugs, given via three routes, and utilizing three mechanisms:

1. **A hyperosmotic drug** (mannitol 1-2g/kg given as a slow IV bolus over 20-30 minutes), which dehydrates the vitreous body.
2. **A systemic carbonic anhydrase inhibitor,** which reduces aqueous production. Methazolamide (Neptazane®) at 2-10mg/kg PO BID – TID) or dichlorphenamide (Daranide®) at 2-5mg/kg PO BID - TID) appear to be the best-tolerated.
3. **A Topical miotic agent** to promote aqueous outflow. Traditionally, pilocarpine has been applied as regularly as q 30mins to the affected eye until adequate miosis is achieved.

However, latanoprost (Xalatan®) and the other synthetic prostaglandins exert such potent pressure-lowering effects that I now use a drop of one of these agents every 5 minutes for 4 or 5 applications while chatting to the client and before initiating the more complex regimen with catheter placement and mannitol administration.

A single systemic dose of your preferred corticosteroid for “shock” may also be rational since much of the retinal and optic nerve damage during (and after) an acute glaucomatous crisis may involve retinal hypoperfusion, followed by reperfusion injury.

*References available from the author on request.*
Osteoarthritis is the most common joint disease in dogs with approximately 1 in 4 dogs being affected, although it has been suspected that this number may be an underestimation due to this disease being underreported until the later stages. Osteoarthritis is a disease of the entire joint organ with loss and dysfunction of the articular cartilage and is usually highly inflammatory in nature. Resulting changes will progressively impact all structures within the joint, including a thickened joint capsule with inflamed synovium and reduced viscosity of synovial fluid, damage to cartilage and subchondral bones, and development of osteophytes. The etiology of OA is complex with local mechanical as well as systemic and metabolic contributing factors. The chronic and progressive characteristics make it a challenging disease for clinicians to control. When left untreated, OA can progress to a severely debilitating disease with significant functional impairment and pain sensitization. It is these late-stage cases (that had not had appropriate early treatment) that present with a multitude of challenges and are not uncommonly refractory to treatment.

These cases require thorough client education on the current state of OA for owners to understand the severity of the disease, its debilitating effects and its need for appropriate treatment. Treatments are specific for the individual patient and require a multimodal approach and constant fine-tuning and readjusting. The pain experience is unique for every individual, as is their response to treatment(s). Factors including a patient’s personality, receptor genetics, metabolism, and degree of peripheral and central sensitization, which all serve to emphasize the importance of tailoring treatments to an individual patient. NSAIDs remain the cornerstone of OA treatment at all stages of OA and should be used where possible. Additional medications and medicinal supplements are layered in and can include (among others) pregabalin, amantadine, cannabinoids, green lipped mussel and Boswellia based supplements, and most importantly adequate DHA/EPA supplementation. Different modalities like acupuncture, PEMF, photobiomodulation may also help in most cases and specific situations. Joint injections with platelet rich plasma (PRP) or hyaluronic acid (HA)/triamcinolone could be considered at this time if a particular joint is refractory to treatment.

A formal rehabilitation program designed by a rehabilitation practitioner is highly recommended if logistics allow. Rehabilitation ensures appropriate assessment and treatment of pain on a regular basis, aiming to slow down the disease progression with a focus on mobility. A rehabilitation partnership provides support to owners for their dog’s debilitating disease. This support can include QoL assessments and discussions. A rehabilitation team will create an individualized program for the patient that may include targeted therapeutic exercises which focus on core strength and posture, maintaining or gaining range of motion, improving overall physical fitness, and strengthening the musculature that is required to provide stability for arthritic joint.

This emphasizes that dogs with OA still require regular exercise. This is an important aspect of OA management for dogs to help keep the joints mobile, cartilage healthy, and maintain muscle strength to support the joint. Historically “prolonged rest” was prescribed in cases with OA pain. This approach has the disadvantage that when a joint lacks movement, it will stiffen further (fibrosis) and decline cartilage health. A lack of exercise will contribute to muscle atrophy, thereby further reducing joint stability and contributing to pain. The practice of severe activity restriction or rest is generally not recommended, instead regular, low impact exercise is an important part of pain management in OA. Regular physical
activity is crucial to slow down the progression of sarcopenia and maintain physical fitness in dogs with OA, including the geriatric population. To be able to do this required and suggested exercise and rehabilitation program, adequate pain management is a prerequisite.

Exercise will also help maintain and reach an ideal body condition score. Weight management is one of the most important aspects for OA treatment. The influence of adipose tissue attributing to low-grade systemic inflammation has been recognized and a weight loss program has shown both in humans and dogs to have general health benefits and potentially decrease and slow down the progression of OA in humans and dogs and is therefore considered by many an actual treatment option. An increase in body weight has been demonstrated to have negative effects on the osteoarthritic joint load and maintaining optimal body condition should be one of the most important goals for any patient at any OA stage. A specific effort should be made to educate and support owners in a weight reduction plan for their pet. This includes nutrition counselling for the right diet (weight or joint health focused or both), including both caloric and omega 3 fatty acid dose recommendations.

Other aspects of patient care cannot be overlooked, including lifestyle and household modifications for injury prevention and QoL improvement. The idea of simplification of obstacles can be achieved for example through ramps for easier access to stairs/car, baby gates to block off stairs for prevention of falls/injuries, carpet runners or yoga mats over slippery floor to prevent slipping, well-padded dog beds for easier comfort, improved traction with nail covers or grips to prevent slipping and dragging toes, assistive devices such as special harnesses (Help'emUp harness) for improved mobility. Adequate nail trimming is also an underestimated tool to assist with proper biomechanics and appropriate alignment. Improving traction and reducing risk of slipping is further achieved by appropriate trimming of foot fur to allow for pads contacting the floor.

Cases present in this lecture will represent cases that are difficult to treat and require all resources to maintain an adequate quality of life. Discussions include different treatment plans, failure of treatment success, assessment of improvements, and quality of life decision.

The full version of the Canadian Consensus Guidelines on OA Treatment based on COAST Stages 1-4 can be found here:

A LIMPING PUPPY IS (ALMOST) ALWAYS AN EMERGENCY
Lea Mehrkens, DVM, Diplomate ACVS

There are numerous causes of lameness in juvenile dogs. Because puppies have multiple rapid growth phases, the window of time to treat the cause of lameness before lifelong lameness ensues is often narrow. Lameness that does not improve within a few days should be investigated. Lameness should only be attributed to a soft tissue injury, such as a strain or sprain, only after more serious causes of lameness are excluded through thorough orthopedic examination and imaging.

The top differentials for shifting leg lameness in a dog less than 1 year of age are panosteitis and immune-mediated polyarthritis (IMPA). Panosteitis typically affects dogs under one year old, although it has been reported in dogs as old as five years of age. Panosteitis is characterized histologically by empty space within the adipose bone marrow and bone proliferation around the nutrient foramen (Van Sickle 1980). Congestion of the blood supply results in additional new bone formation and local periosteal bone formation. This disease, while painful, is typically self-limiting and is treated symptomatically with non-steroidal anti-inflammatories. When severe, affected dogs may need hospitalization for analgesia and supportive care.

In contrast, IMPA is a progressive and debilitating disease treated with tapering courses of immunosuppressive drugs. Causes include but are not limited to: reactions to vaccines, certain medications, neoplasia, and tick-borne disease. However many cases are found to be idiopathic in origin. If not treated, IMPA can result in irreversible breakdown of the ligamentous structures of the weightbearing joints or cartilage destruction. Dogs with severe breakdown of joints in multiple limbs are unlikely to have a successful outcome, even when multiple surgeries are performed. Distinguishing between these two diseases is critical and can be achieved through survey radiography and arthrocentesis with samples submitted for cytology and bacterial culture.

Another top differential for a skeletally immature dog presenting with lameness, malaise, and pyrexia is hypertrophic osteodystrophy (HOD). No single unifying cause of HOD has been identified, although many have been theorized. The distal radius and ulna are most commonly affected, although the disease has also been described in flat bones such as skull and scapula as well as the vertebrae, ribs, and other long bones. Clinical signs include lameness, and swelling and pain of the affected bones, and systemic signs such as inappetence, lethargy, diarrhea and vomiting (Selman 2022). In severe cases, systemic inflammatory response syndrome has also been documented secondary to profound immune reaction (Safra 2013). The radiographic signs of a double physeal line and periosteal reaction are pathognomonic for this disease. Treatment typically consists of supportive care (IV fluids, feeding tube placement, IV opioid administration) and either anti-inflammatories or corticosteroid treatment. In severe cases, growth plate bridging and synostosis secondary to new bone formation can lead to severe angular limb deformity.

Hip dysplasia is the most common cause of lameness in the pelvic limb in juvenile dogs. Puppies with hip dysplasia often present with histories of being “quiet” or “lazy” puppies and often have a characteristic hip-sway while walking and a bunny-hopping gait while running. While hip dysplasia is technically not an emergency, there is a very early and relatively non-invasive surgery called juvenile pubic symphysiodesis (JPS) that can be performed for puppies from 12-20 weeks of age. Predisposed breeds with palpable hip joint laxity but without severe dysplasia are good candidates for this procedure. JPS consists of cauterizing the ventral
synchondrosis between the two hemipelvices at the pubic symphysis. This arrests the growth of the ventral aspect of the pelvis, allowing the dorsal aspect to continue growing and improving coverage of the femoral head with the acetabulum. The younger the puppy at the time of the procedure, the greater the improvement in femoral head coverage. Past this age, a double or triple pelvic osteotomy may be performed to similarly improve acetabular coverage (Manley 2007). Total hip replacements can be performed as early as 8 -12 months old. Outcomes are improved when the procedure is performed in patients prior to severe muscle atrophy and in cases which are not chronically luxoid. Early referral is thus recommended for severe cases to maximize prognosis.

The two most common causes of forelimb lameness in the dog are elbow dysplasia and osteochondrosis dessicans (OCD). These two diseases are a race against the clock to limit progression and onset of osteoarthritis. Patients with elbow dysplasia and OCD oftentimes present with an alteration in posture characterized by external rotation of the distal limbs. Oftentimes both joints are affected and lameness is less evident. Exercise intolerance in either disease process is typically progressive. Prognosis of OCD of the shoulder is generally excellent with cartilage flap removal +/- osteoallograft transfer. Prognosis for elbow dysplasia after arthroscopy and fragment removal is variable. Some patients with severe disease may require medial compartment resurfacing procedures.

In some lameness cases, the signalment and history of the patient can significantly guide the diagnosis. French bulldogs are amongst the most common breeds to develop a humeral condylar fracture and are 49 times more likely to develop a fracture than other breeds (Schettler 2022). This articular fracture requires immediate surgical repair to minimize long-term arthritis. A delay in surgery of even 3-5 days significantly worsens prognosis as new fibrous tissue begins to form around the fragments and makes a perfect reduction of the articular surface challenging.

Trauma to the physes is a common cause of lameness in juvenile patients. In these cases, puppies are often initially painful for a few days and seem to recover well with analgesic therapy. Months later, however, these puppies present with angular limb deformities or severe arthritis due to joint incongruity. The most commonly affected growth plate is that of the distal ulna, accounting for 63% of all growth plate injuries (Ramadan 1978). This growth plate is conically shaped and provides 100% of longitudinal growth to the ulna. The conical shape predisposes it to injury from vertical and horizontal impact (Fox 1984). These fractures are often difficult to diagnose on radiographs, requiring comparisons with the contralateral limb or radiologist interpretation. Without treatment, the short ulna tethers the growth of the radius, resulting in radial shortening, procurvatum, torsion, and distal valgus. These changes will be less severe if the patient is more skeletally mature at the time of injury and has less potential for skeletal growth. Importantly, these changes also can cause severe humeroulnar incongruity, leading to severe arthritis and potential fracture of the anconeal process. Treatment of choice in growing puppies is dynamic ulnar osteotomy, which frees the proximal ulna to move proximally and maximize congruency of the elbow joint. If done early enough, the release of this “bowstring effect” of an arrested ulna also minimizes the severe torsional and shortening deformities on the radius. When these injuries are not diagnosed until later in development or skeletal maturity, complex angular limb correction surgeries are necessary.

Juvenile patients presenting for lameness warrant a thorough physical and orthopedic examination. In many cases, an early diagnosis and initiation of treatment can prevent subsequent abnormal bone growth, articular cartilage damage, and subsequent arthritis.

References:


Stop Scruffing that Cat! The Move Towards Zero Restraint.
Kelly A. St. Denis, MSc, DVM, DABVP (feline practice)

There is no question that cats generally have a bad reputation in the veterinary clinic. Visits can go sideways quickly, and the effort to get the job done during what are increasingly busy practice hours can take over the need to be patient and understanding with the cat. There is a line in my favourite movie, Moonstruck: ‘It costs money because it saves you money’. Working with cats requires patience, the reward being that things will move along more smoothly and take less time. Cat Friendly ‘costs time because it saves you time’. When we are working with cats, we need to SLOW down, look, listen, and think. We need to start by thinking about what emotions our feline patients are experiencing. Yep, that isn’t a type-o, consideration for a cat’s emotions during a veterinary visit are key to a successful visit. And yes, that takes time.

Cat Emotions
Our feline patients come to us with varied socialization history (ex. rescue, shelter, private home, breeder), various past veterinary experiences, and their experiences on that particular day (ex. dogs in the waiting room). They may be coming for a wellness visit or be unwell. They may be in a state of positive well-being or a poor state of health. All of this taken together impacts the cat’s emotions. The Heath Model of Emotional Health views emotions as being protective (negative) or engaging (positive). It recognizes that all emotions have a purpose in protection and survival and therefore all emotions are good when they benefit the animal.

Protective emotions are sometimes termed ‘negative’ emotions, but this implies that they are not good for the animal. Protective emotions help keep the animal safe, healthy, and alive. Behaviours in response to protective emotions at the clinic include inhibition (‘freeze’), avoidance (‘flee’), and repulsion (‘fight’), with avoidance being the cat’s preferred action.

1. Fear/anxiety: motivates cats to avoid threat and harm
2. Frustration: Induced by an inability to access safety, receiving less than anticipated rewards, inability to control the situation or failure to succeed in purpose of other emotions. Motivates the cat to increase chance of obtaining what it wants. Responses tend to be more rapid and intense.
3. Pain: Motivates cats to avoid tissue damage, protecting painful areas. Pain is included as a component of fear and anxiety because it is both a sensory and emotional response. Fear/anxiety can increase the perception of pain and the presence of pain can increase fear/anxiety.
4. Panic/Grief: Panic ensues when a cat is unable to connect with normal protective responses when fearful. Grief occurs because of loss, including other pets, people or due to major changes in the environment or schedule. Panic or grief motivate the cat to connect with their social attachments that are crucial for survival.

The cat will need to be able to cope with or respond to these emotions to resolve them. For example, during an examination, a cat is likely to experience some degree of fear or anxiety. One successful coping strategy is for the cat to hide (avoidance). By providing soft towels or blankets which can be placed under and over the cat, we can assist the cat in resolving some degree of fear and anxiety. For added comfort, spray the towels with Feliway in advance, and warm the towels with a towel warmer or heated oat bags.
Engaging emotions allow the cat to interact with others, with resources and the environment. Engaging emotions ensure and enhance survival.

1. **Desire-seeking**: motivates cats to find pleasurable experiences and resources required for survival
2. **Social play**: motivates (mostly young) cats to learn and practice social behaviour
3. **Lust**: motivates sexually intact cats to mate
4. **Care**: motivates female cats to care for their young

During the veterinary visit, engaging emotions such as desire-seeking can be utilized to improve the veterinary experience. For example, provision of food rewards such as flavoured tube treats can help to stimulate positive, desire-seeking emotions, thereby reducing fear/anxiety.

**What happens when we restrain?**
Restraint in the veterinary setting can be defined as placing a cat in a physical position against it desire. The cat is often placed into restraint for the team to successfully complete various tasks (ex. vaccination, blood draw etc). There is a distinct loss of control and a lack of consent for the cat. Restraint increases fear/anxiety and frustration. When unable to cope with and resolve these emotions, protective emotions and related behaviours can escalate very quickly.

A group of researchers at the OVC investigated behavioural and physiological responses to different types of handling including passive restraint, clip restraint, scruffing, and full body restraint. Clip restraint resulted in more negative responses than passive restraint and scruff restraint. Full body restraint and clip restraint resulted in the greatest number of negative responses, scruffing resulted in fewer negative responses, and passive restraint showed the least number of negative responses.\(^3\)

Anything that takes away a cat's choice or sense of control will be perceived as a threat, including the above types of restraint, but also including e-collars, masks, thick gloves of any variety, and even so-called kitty burritos or purritos.

**What happens when we take the time to adapt to the cat's emotions?**
More information about feline emotions and how to interpret them can be found in the 2022 AAFP/ISFM Cat Friendly Interactions Guidelines.\(^1\) Our approach to reducing protective emotions and promoting engaging emotions is 3-fold. We need to develop an environment that reduces potential threats that lead to fear and anxiety.\(^2\) We need to be able to interpret feline body language and related emotions, making sure we are ready to provide the cat with what is needed to cope with and resolve these emotions.\(^1\) Finally, in cases where a patient is known to be painful or may be painful, we need to provide analgesics before proceeding with the visit. In situations where we are unable to reduce protective emotions sufficiently to continue with the appointment, we need to be able to recognize and recommend anxiolytics or sedation before protective behaviors escalate. Gabapentin is a useful anxiolytic which can be administered 2 hours prior to departure for the veterinary clinic.\(^4\) A useful chart outlining sedation options for Cat Friendly Interactions can be found in the guidelines.\(^1\)
Points to Consider

- Aggression is not a personality trait. It is a protective behaviour in response to fear, anxiety, pain, and/or frustration, that serves to put distance between the cat and the perceived (or real) threat.
- Words Matter. When practice teams use labels like fractious, feisty, aggressive, jerk, bad kitty, and other negative terms, they bias their own expectations each time the cat visits, and predispose towards an unpleasant visit for all.
- Understand your patient. Knowing your patient’s origin, history, previous experiences, and emotions on the day of presentation will allow the team to adjust to the patient’s needs and result in a more positive visit for all.
- Restraint is a threat to the cat. Restrictive physical interactions with cats increase protective emotions in response to the threat these restrictions present. They will increase, not decrease negative experiences for all. Cats will remember these experiences.
- Freedom to choose reduces protective emotions. Permitting the cat to have choices in the appointment and encouraging engaging emotional responses improve outcomes.


WHY ANTIBIOTICS CAN BE WORSE THAN USELESS FOR DENTAL PATIENTS

Fraser A. Hale, DVM, Dipl AVDC

Introduction:

No one can argue that antibiotics have not been a great boon to modern medicine, treating countless infections and saving countless lives. As it turns out, they have been in use for many centuries with historical accounts of the use of moldy bread, applied topically, going back to ancient Egypt, Nubia, Serbia, China, Greece and Rome (Gould K "Antibiotics: from prehistory to the present day". Journal of Antimicrobial Chemotherapy. 71 (3): 572–575. March 2016). John Parkinson, an English botanist and apothecary (1567-1650) is credited with being the first person to directly document the use of molds to treat infection.

Alexander Fleming is known as the one to discover penicillin in 1928 when he observed an inhibition zone around mold growing on an agar plate upon which he was attempting to grow some pathogenic bacteria. Initially, nothing much came of this observation. Fleming was not a chemist and did not have the resources to explore ways to isolate, purify, stabilize and produce penicillin in a manner that would make it useful as medical treatment.

Then came World War II. In preparation for the assault on Fortress Europe (and the anticipated associated casualties), the Allies commissioned work to find ways to make a stable form of penicillin in quantity. It was only after 1945 that penicillin became available outside of military use. In other words, while most people reading this will have never lived in a world without antibiotics, they have only been around (in a meaningful way) for under 80 years.

In modern-day veterinary general practice, antibiotics, both systemic and topical, are powerful and essential tools in the treatment and management of many bacterial infections. However, there is a large area of veterinary practice in which antibiotics have virtually no place and that is in the management of dental disease.

The conventional wisdom, since the development of “Germ Theory”, has been that gingivitis and periodontal disease are caused by bacteria and there is some truth to this. However, while the oral cavity is certainly home to a great many species of bacteria, they are not alone and the vast majority of them are benign or beneficial.

In his paper “Gene Sequence Analyses of the Healthy Oral Microbiome in Humans and Companion Animals” (J Vet Dent. 2016 Jun;33(2):97-107), Eric Davis points out that the oral microbiomes consist of a staggering array of bacteria, molds, fungi, viruses and protozoan, all living in a complex structure (a biofilm), enjoying symbiotic relationships with each other, sharing nutrients, protective factors, genetic material and more. It has been shown that bacteria living in a biofilm behave very differently than the same species would in a monoculture on an agar plate. Among other things, they are up to 1400 times more resistant to antimicrobial agents. And most of the bacteria resident within the various oral microbiomes will not grow in the artificial world of an agar plate and so have gone undetected until recent years when gene probe sequencing has pulled back the veil on this fascinating world. This is why culture and sensitivity testing of the oral cavity will be of no value. All it will tell you is which, of the hundreds of species of bacteria collected from the test site, will grow happily in the laboratory and, when in that artificial environment, which antibiotics inhibit/kill them. This is of no clinical relevance at all as the bacteria in the mouth do not live in an artificial monoculture and the ones that grow in the lab are usually not the ones causing disease in the mouth.

In health, these organisms also enjoy a symbiotic relationship with their host. In What Am I and Why Do I Do the Things I Do? (Chapter 4: What it means to be human; the tiny picture. Self-Published, Amazon 2019: pp102-139), retired veterinary dentist and deep thinker, Gregg
DuPont points out that the human body is composed of 37 trillion mammalian cells and roughly 100 trillion microbes. We are highly dependent on these life-forms for our own life.

The paper *Oral Microbiome in Dogs and Cats: Dysbiosis and the Utility of Antimicrobial Therapy in the Treatment of Periodontal Disease* by Davis and Weese (Vet Clinics: SAPP. 2022: 52(1): 107-119) makes the point that gingivitis, periodontal disease, contact mucositis and other oral inflammatory disease should not be viewed as simple infection caused by one or a few pathogens but more as a dysbiosis between the host and the residents of the microbiomes that results in upregulation of pro-inflammatory factors leading to tissue destruction.

**Antibiotics not effective for dental patients.**

Antibiotics work well when used to treat an infection caused by the overgrowth of a single pathogen that has demonstrated sensitivity to the antibiotic to be used. However, inflammatory dental disease is caused by a dysbiosis between the host and its highly complex collection of bacteria, molds, fungi, viruses, protozoans, all living within a protective biofilm +/- mineral deposits (calculus). Antibiotics cannot effectively penetrate the biofilm and even if they could, they are only going to have an effect against those few bacteria that are sensitive, while leaving the resistant bugs to proliferate out of balance with the healthy state we are trying to establish.

Even if we view periodontal disease (for instance), as simply being caused by bacterial overgrowth on the tooth surfaces, antibiotics cannot clear the infection as they cannot remove the mineralized deposits or biofilm. Antibiotics may be able to suppress the number of bacteria growing in the adjacent soft tissues, but they cannot remove the source of infection. If you get a splinter in the bottom of your foot and it becomes infected, you do not treat this with antibiotics. You remove the splinter (get rid of the source). If you try to treat dental disease with antibiotics, you might get some temporary symptomatic relief but if you do not surgically/mechanically remove the source of the problem, it will come back, either after you cease treatment or once the pathogens have developed resistance. For fun, watch this 2-minute YouTube video on the evolution of antibiotic resistance - [https://www.youtube.com/watch?v=yybsSqcB7mE](https://www.youtube.com/watch?v=yybsSqcB7mE).

I will acknowledge that there are some cases in which the use of antibiotics, as an adjunctive therapy, may be justified but I state categorically that antibiotics should never be used as a monotherapy for any dental condition and in the vast majority of dental patients, there is no need for them whatever.

What about bacteremia during dental treatment? Is there not a need for antibiotics peri-operatively? Well, actually, no, there is not. Have a look at this brief bulletin: *Do patients with prosthetic joints need antibiotics before invasive dental work?* (Paumier T. Dentaltown Magazine. June 2022). I have also recently reviewed a study submitted for publication that found no justification for the perioperative use of antibiotics in healthy dogs undergoing exodontia. Hopefully this paper will be published in the J Vet Dent sometime this year.

**How antibiotics can actually be worse than useless.**

Remember that we are 37 trillion mammalian cells and 100 trillion microbial cells and we are highly dependent upon healthy, balanced microbiomes occupying the huge number of niches within our bodies (the biome on the tongue is very different from the one on the palate, which is different from the one on the tonsils and so on). If we use systemics antibiotics to try to manage a dysbiosis in one area of the body, we will upset the microbiomes throughout the body, possibly to the great detriment of the patient. Systemic antibiotic can upset the gut flora, reproductive flora, cutaneous flora…There is an ever-growing body of information that points to unhealthy micorbiomes as the root of several ailments. Fecal transplants are now a thing and probiotics have become mainstream. We must accept that antibiotics are not benign and should never be used casually.
As well as harming the patient, antibiotics have the potential to harm your relationship with your clients. I see many histories (on www.VIN.com and in my practice) in which the GP has seen an oral condition, did not really know what it was or did not investigate it but just put the animal on antibiotics. When that inevitably failed, they may have tried another antibiotic or a longer course of the first one and this also failed. The client has now spent considerable time traveling to the clinic and money on these visits and prescriptions and their pet is no better. At some point, the client might start thinking that their vet really does not know what they are doing and may start resenting their wasted investments (of time and money). It is hard to get client compliance with the next set of recommendations when the client has lost confidence.

Among the most troubling misuses of antibiotics I see is when a pet is presented with an oral swelling or mass and the first course of action is antibiotics. I have seen many cases in which diagnosis and treatment of growing cysts, benign growths and oral malignancies was delayed while the patient was treated with antibiotics to see if they would help. Giving a swelling or mass time to get larger, invade deeper or metastasize while you put the animal on an antibiotic speculatively is never going to be a good idea for the patient or their owner.

And of course, there is the mounting concern about antibiotic resistance leading to a real (and necessary) push for far greater stewardship of antibiotics (https://www.aaha.org/globalassets/02-guidelines/2022-antimicrobial/2022-aaaf_aaha-antimicrobial-stewardship-guidelines.pdf). We have only had antibiotics available as a medical treatment for about 78 years. If we do not radically change our ways, I fear that before their centennial, antibiotics will cease to be of any use to us in the management of bacterial infections.

But what about the client who declines proper diagnostics to get to an appropriate treatment plan? What if they back you into a corner where the only option they leave you is to try some antibiotics? Surely doing something is better than doing nothing. Except it is not. Doing nothing is far better than doing the wrong thing (Hale F. Just say no to bad medicine. http://www.toothvet.ca/PDFfiles/Just_Say_No.pdf. 2018).

**Not Never:**

In my referral practice, where is see horrible disease day in and day out, I maybe use antibiotics 5 or 6 times a year, as a single IV dose at anesthetic induction, such as for an ancient patient with sewer-mouth and serious co-morbidities that might constitute immunocompromise.

If an animal presents with an acute facial cellulitis secondary to chronic periodontal or endodontic disease and cannot be scheduled for assessment and definitive treatment that day or the next, providing some temporary symptomatic relief (analgesics +/- antibiotics) until you can get the animal on the table makes sense. However, you MUST ensure that the owners understand that this is just temporary, symptomatic relief and even if the swelling goes down, the pet still needs diagnosis and treatment under general anesthesia ASAP. It will be your task to educate the owners so that they know that the antibiotics absolutely cannot remove the source of the problem before you let them leave the building, drugs in hand.

**Conclusion:**

Remember: “diagnosis first, then treatment”. And that treatment is going to mean physical removal of the source of the problem. Very occasionally, after an appropriate work-up, it may be determined that antibiotics should be part of the overall treatment plan, but they should never be used as a monotherapy and should not be used prior to arriving at a credible diagnosis. In the vast majority of cases, there is no reason to give them at all and many reasons to avoid them completely.
Retropulsion of Urethral Calculi in the Dog
Howard B. Seim III, DVM, Diplomate ACVS
Colorado State University

If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to www.videovet.org or contact videovet@me.com.

INTRODUCTION
Uroliths are defined as calculi lodged in the urethra causing partial or complete obstruction and urethritis. Uroliths may be caused by infection, diet, or they may be metabolic.

Generally, small cystic calculi migrate to the neck of the bladder during micturition and pass into the urethra. In the male, urethral calculi most commonly lodge caudal to the os penis. In the female, calculi may lodge at any location along the length of the urethra. Urethral obstruction is more common in the male than female.

CLINICAL SIGNS
Clinical signs include stranguria, hematuria, pollakiuria, and occasionally blood dripping from the prepuce. Patients with complete urinary obstruction may present with a painful, distended abdomen, anuria, and azotemia. The severity of signs is often dependent upon degree and duration of urethral obstruction.

DIAGNOSIS
Diagnosis is generally based on suspicious clinical signs, inability to pass a catheter into the urinary bladder, and survey radiography or positive contrast retrograde urethro- cystography revealing a urethral obstruction.

DIFFERENTIAL DIAGNOSIS
Diagnostic differentials include neoplasia, urethral stricture, urethritis (e.g., granulomatous), and urethral trauma.

TREATMENT
Immediate care is dependent upon patient presentation and severity and duration of urinary obstruction. In animals with complete obstruction of a duration long enough to cause azotemia, temporary urinary diversion is provided by either passing a urinary catheter alongside the calculus, performing a prepubic cystostomy, or frequent cystocenteses. Treatment of azotemia with crystalloid IV therapy is performed prior to calculus removal.

RETROGRADE HYDROPULSION
See the DVD for a detailed video of this technique.

Technique
This technique should result in a 90-95% success rate of retropulsing urethral calculi into the urinary bladder!
1. Select the largest diameter sterile high density polypropylene or nylon urinary catheter (not a red rubber feeding tube) that will fit past your patient's os penis (generally 6, 8, or 10 French diameter)

2. If the selected catheter turns out to be a 6 French diameter then mix 30cc of Sterile KY Jelly with 70cc of sterile physiologic saline solution.

3. If the selected catheter turns out to be an 8 or 10 French diameter then mix 40cc of Sterile KY Jelly with 60cc of sterile physiologic saline solution.

4. Thoroughly mix the sterile saline and KY Jelly in a 35 or 60 cc syringe and attach the syringe to the urinary catheter.

5. Anesthetize the patient, extrude the penis and pass a generously lubricated nylon or polypropylene urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.

6. Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure. Be certain the catheter tip hits the calculus like a battering ram to help dislodge it and encourage the saline-lubricant mix to surround the calculus and coat the urethral wall. During injection the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

This technique is successful regardless of how many stones are in the urethra and no matter where the calculi are lodged.

If the above technique fails, place a finger in the rectum, palpate the urethra and occlude its lumen (this dilates the urethra); repeat the above maneuvers and when maximum pressure is exerted on the urethra by the saline/lubricant mix (i.e., the urethral is maximally dilated), suddenly release digital urethral occlusion allowing lodged calculi to flush into the urinary bladder.

**Urethrotomy** (an incision over the calculi) may be performed to remove calculi that cannot be retropulsed. It is usually performed in the prescrotal or perineal region.

**Urethrostomy** (a permanent opening to allow calculi to pass) may be indicated in animals that are chronic recurrent calculi formers (e.g., urate calculi in Dalmatians). Scrotal urethrostomy is the technique of choice.

**PATIENT MONITORING**
Patients requiring a cystotomy only can be expected to pass small quantities of blood and blood clots for 2 - 3 days postoperatively. Animals presenting with complete urinary obstruction and postrenal azotemia are continued on crystalloid IV therapy until serum urea nitrogen and creatinine return to normal.
Patients requiring a urethrotomy or urethrostomy may hemorrhage from the urethral stoma and is the most common immediate postsurgical complication. It generally occurs 4 - 5 days postoperatively, but occasionally will last up to 2 weeks. Mild dripping is managed with cage rest and tranquilizers to decrease blood pressure. Moderate hemorrhage is managed by mild pressure with a cold compress placed directly over the urethrostomy site. In some cases it is necessary to apply an Elizabethan collar to prevent self-mutilation.
Reducing the Greenhouse Gas Impact from Anesthetic Delivery

Craig Mosley, DVM, MSc, DACVAA
Staff Anesthesiologist, VCA Canada, 404 Veterinary Emergency and Referral Hospital

Key points
- Anesthetic gases are potent greenhouse gases (GHG)
- Anesthetic gases represent significant scope 1 (direct/internally controlled) emissions
- Small changes in behaviour can make significant impact on GHG emissions associated with anesthetic gases
  - Choosing to use lowest impact anesthetic gases
  - Lower fresh gas flows
  - Reducing anesthetic gas requirements; proper use of premedicants/analgesics, intravenous infusions, locoregional anesthetic techniques
- Future solutions might include; capture & reuse and/or capture & destroy

Background
Chlorofluorocarbons (CFC) and hydrofluorocarbons (HFC) are potent greenhouse gases (GHG), that are often used as industrial refrigerants, propellants and include the anesthetic gases. The international community under various United Nation’s agreements, the 1987 Montreal Protocol and later the 2016 Kigali Amendment, aim to phase out and reduce the use of CFC’s and HFC’s but the anesthetic gases have been excluded and their ability to contribute to GHG emissions is largely unknown to most, even those working with them daily. While the relative contribution of anesthetic gases to overall carbon emissions in veterinary medicine (and human medicine) is relatively low (~3-5%), they do represent a significant proportion of scope 1 or direct emissions (~15-20%); emissions derived directly from “in-house” activities such as vehicle emissions, generators owned and used by a facility and the use of anesthetic gases. In contrast to scope 2 and scope 3 or indirect emissions; such as the emissions coming from the utilities used to power the facility and the goods, supplies and equipment used to run a practice, scope 1 emissions are more easily controlled and influenced directly and hence the individual practitioner or hospital facility has the ability to more easily control or influence these emissions.

The two most commonly used anesthetic gases isoflurane and sevoflurane are considerably more potent than carbon dioxide in their global warming potential. It should also be apparent to anyone involved in the delivery of inhalant anesthetic that in fact very little of a delivered inhalant anesthetic is metabolized or taken up by the body and that almost all of the amount delivered is simply released to the atmosphere. In other words, the entire contents of an inhaled anesthetic bottle is simply released to the environment once it’s done its job keeping our patients sufficiently anesthetized.
<table>
<thead>
<tr>
<th>Agent</th>
<th>GWP&lt;sub&gt;100&lt;/sub&gt;</th>
<th>Atmospheric lifetime (yrs)</th>
<th>MAC in dogs (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoflurane</td>
<td>510</td>
<td>3.2</td>
<td>1.3</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>130</td>
<td>1.1</td>
<td>2.3</td>
</tr>
<tr>
<td>Nitrous oxide</td>
<td>265</td>
<td>110</td>
<td>~235</td>
</tr>
<tr>
<td>Carbon dioxide</td>
<td>1</td>
<td>74</td>
<td>n/a</td>
</tr>
</tbody>
</table>

GWP<sub>100</sub> Global warming potential over 100 years, MAC Minimum alveolar concentration

In looking to reduce the impact of anesthetic gases the obvious solutions are to choose the lowest impact gas (i.e. sevoflurane), reduce the amount administered/required for anesthesia and to collect and recycle or destroy the delivered gas (i.e. the gas that exits the scavenging system).

**Agent selection**

Nitrous oxide use as an anesthetic adjunct in North America is relatively uncommon and complete elimination of its use should probably be encouraged due to its limited positive impact on anesthetic quality and the inherent risk of possibly delivering a hypoxic gas mixture. Sevoflurane has favorable characteristics in terms of its global warming potential and despite requiring twice the amount (MAC is nearly twice that of isoflurane) its use could still reduce the overall impact of anesthetic contribution to GHG emissions by roughly 50%. Though there are cost considerations, the GHG emissions associated with the procurement of new equipment and the potential for a potentially nephrotoxic metabolite (Compound A) to be produced when using very low fresh gas flow rates. However, this later concern may be more theoretical than practical based on available in-vivo clinical studies.

There is also an excellent verified [anesthetic gases calculator](http://example.com) developed by Dr. Tom Pierce that can be used to estimate both the carbon dioxide equivalence and costs associated with various anesthetic gases and flowrates.

**Fresh gas flow rates**

Given that the vast amount of delivered anesthetic is simply released to the atmosphere, very little is metabolized or taken up by the patient, reducing fresh gas flows (FGF) and hence the volume/amount of gas delivered can have considerable impact on GHG emissions. For example, reducing maintenance FGF rates from 2 l/min to 1 l/min or even 0.5 l/min will reduce GHG emissions by roughly 50 and 75% respectively. However, the FGF rates should be considered in the context of the breathing system being used. If a non-rebreathing system is used, flow rates must be sufficiently high (150-250 ml/kg/min) to prevent rebreathing of exhaled gases (i.e. CO<sub>2</sub>) as the proper functioning of these systems are flow dependent. Non-rebreathing systems are best reserved for smaller patients, those under 3-5 kg. Using these systems on patients greater than 5 kg is a missed opportunity to lower GHG emissions. However using rebreathing systems (i.e. circle) on smaller patients does require increased attention to detail in terms of reducing unnecessary dead-space associated with the overall breathing system (i.e. endotracheal tube, airway gas sampling adapter, humidification/filters and breathing circuit). Rebreathing systems are flow independent meaning that they will function properly (i.e. prevent the rebreathing of CO<sub>2</sub>) regardless of flowrate. This of course
assumes the system is functioning properly with sufficient active CO₂ absorbent. Using lower FGF rates with rebreathing systems can reduce the use of oxygen and the amount of inhalant anesthetic being released to the environment. However there are consequences in terms of inhaled anesthetic delivery characteristics and potential risks to patient safety should gas flows fall below the patients metabolic oxygen consumption (3-5 ml/kg/min) and/or if the reservoir/rebreathing bag fails to contain sufficient gas volume for the patient to inspire. Practically it is probably safest to use higher relative fresh gas flows (50-100 ml/kg/min) for the first 15 minutes of anesthesia to ensure sufficient inhalant delivery to the patient (during the rapid uptake phase of anesthesia) followed by a reduction in flow rates (10-20 ml/kg/min) when less rapid inhalant uptake is occurring (i.e. maintenance phase) to minimize waste of both oxygen and inhalant.

Reducing anesthetic gas requirements

There are several complete intravenous anesthetic options (i.e. propofol, alfaxalone, ketamine) that will eliminate the GHG effects of volatile anaesthetic agents. However, this yields an environmental cost that must also be considered, resulting from the manufacture, transport, disposal, and electricity consumption of this method of anesthesia delivery. Not to mention total intravenous anesthesia alone is a unfamiliar technique for many of those delivering and monitoring anesthesia. There are also several intravenous adjuncts (i.e. opioids, lidocaine, ketamine, alpha-2 agonist infusion etc) and locoregional techniques that can help reduce the amount of inhalant required to maintain general anesthesia. The use of continuous intravenous infusions requires a thorough understanding of the pharmacology of the drugs used in this manner and how they may be altered with continuous infusions as well as potential interactions. It is important that appropriate delivery equipment is used to ensure the safe and consistent delivery of these agents.

It should be kept in mind that appropriate patient premedication alone (i.e. opioids, alpha-2 agonists, acepromazine, benzodiazepines) will also significantly impact the inhalant requirements (MAC reduction) for any given procedure. Locoregional techniques are also key to reducing inhalant requirements and should always be considered for use whenever such a technique is possible as they will have one of the most profound effects on reducing overall anesthetic requirements.

Future solutions

There are several companies developing technology to capture (absorb) waste anesthetic gases preventing their release into the atmosphere. These captured anesthetic gases could then ideally be recycled for subsequent reuse. These technologies remain nascent and the GHG emissions associated with their collection, transport and distillation will need to be considered. Alternatively the collected gases could simply be incinerated and while on the surface this suggestion may be unappealing, the technologies for absorption of anesthetic gases and incineration are currently readily available and could reduce GHG emissions from inhalant anesthetics by as much as 90%.

Conclusion
Unfortunately many of the greatest contributions to greenhouse gas emissions in veterinary medicine are beyond the control of the individual practitioner but through concerted efforts by all those involved from; individuals changing behaviors, energy suppliers moving to more renewable and sustainable energy sources and the suppliers of goods and equipment moving to lower carbon production methods it will be possible to reduce the impact of our profession on our planet.
Practical Tips for Greening Your Practice
Matthew Richardson, AB, PhD, DVM
Chair, OVMA Environmental Sustainability Committee
drmatthew@theanimalclinic.ca

From reports of climate change fuelling natural disasters, to countries banning single use plastics, to oil spills in the North Sea, to pandemics, the news is hard to ignore. The climate is definitely changing, and humans are responsible. There are a growing number of veterinarians interested in reducing the carbon footprint of their practice, but it can be daunting to know where to start, or what initiatives can really make a difference.

The OVMA is launching its Green Checklist at the 2023 conference to help veterinary professionals navigate these issues and know where best to focus their resources. This talk will explore 5 changes a clinic can make to significantly reduce their environmental impact.

1) Reduce Your Practice’s Energy Use!
   1) SaveOn Energy (http://saveonenergy.ca) is hands-down the easiest, most economical way to reduce you practice’s energy use. The program provides rebates of up to $2,000 for lighting equipment upgrades and up to $2,500 for other eligible equipment upgrades.
   2) How the program works: To be eligible, you need to have 50 or fewer employees, and you need to get your electricity from Ontario Hydro or their subsidiaries (unfortunately areas like Cornwall, ON, which actually get their electricity from Quebec, are ineligible).
      1) Call (833) 825-7283 or fill out the form at https://saveonenergy.ca/en/Start-your-lighting-project to schedule an onsite assessment.
      2) The assessment takes about 1 hour. The assessor walks through the practice, identifying all the lighting and heating upgrades that they recommend. At the end of the assessment, they present the options for the upgrades. If the work recommended exceeds the value of rebates available, it is at the discretion of the business owner whether to proceed with all the recommended upgrades, or just cap it at the maximum level of the rebates.
      3) The SaveOnEnergy program finds and contacts the contractor. The business owner is contacted to arrange a time the contractor can come in with a team. Typically the upgrades are all completed in 1 day, and the contractor takes away all the waste (old tubes, old ballasts from the lights, wiring, etc.).
   3) For a typical vet clinic, upgrades will include: replacing incandescent bulbs with LEDs, replacing flourescent tubes with LEDs and rewiring the fixtures to remove the ballasts, and installing smart thermostats. Although the program talks about upgraded refrigeration, it does not include consumer-grade fridges and freezers that are typically found in vet clinics.
   4) The estimated energy savings range from 10% to 85% per year, depending on the type of lights being replaced. Clinics can expect the savings on their energy bills to pay off the cost of the work within 2-3 years. The installed LED builds have an expected lifespan of 50,000 hours (11.4 years running 12 hours a day, 365 days a year).

2) Switch to 100% Clean Energy!
   1) Reducing the amount of electricity we use is a great first step, but running a veterinary hospital is still energy-intensive. Fortunately, in Ontario our electricity grid is relatively
clean. However, if you want to go the extra mile, Bullfrog Power (http://www.bullfrogpower.com) is your best choice, short of installing your own microarray of solar panels.

2) How it works:
   1) Bullfrog power is a voluntary extra charge, based on your typical energy bill. For a small monthly fee, Bullfrog power makes sure that the same amount of energy you use is put back into the grid from clean, renewable sources.
   2) Bullfrog power funds sustainable options for Electricity (sourcing the electricity from wind mills and solar farms), Natural Gas (sourcing the natural gas from Organic Wastes), and Fuel (sourcing the fuel from wastes streams like cooking oils).
   3) Bullfrog Power is an OVMA Member Benefit - as an OVMA member, you get 10% off commercial accounts and a $50 discount on residential accounts. For commercial accounts, email joseph.mcneil@bullfrogpower.com; for residential accounts visit the http://www.bullfrogpower.com/ and use the discount code: OVMAgreen

3) Replace Single-Use Items!
   1) Single-use items have become more prevalent, in the name of safety and convenience; however not all things need to be single-use. With a bit of common sense and safety precautions, reusable items can greatly reduce a clinic’s waste stream and embedded carbon.
      1) Light cleaning duties: use reusable rags, not paper towels (for heavily soiled, contagious, or other common-sense reasons, stick with disposable).
      2) Hand washing: offer reusable hand towels. To limit the risk of contamination, they should be single-use with a small laundry basket available (like a fancy restaurant bathroom).
      3) Surgical gowns and drapes: in most instances, reusable, sterilizable cloth drapes and gowns are as good as single-use disposables
      4) Cotton/polyester instrument wraps for surgical packs: can be re-used multiple times.

4) Recycle All The Things!
   1) Municipal waste streams are complex; each municipality has different rules for what can go in each bin, and that includes recycling. Furthermore, we have all seen news reports about “recycling” programs where no recycling actually happens.
   2) Plastic and mixed wastes can be difficult to recycle, and the veterinary industry, by necessity, uses a lot of single-use plastics. TerraCycle (http://www.terracycle.com) promises to “recycle the unrecyclable,” and offer server a wide variety of different recycling solutions.
      1) Free Recycling Programs: https://www.terracycle.com/en-CA/brigades
         1) Royal Canin packaging recycling - available at REN’s Pet Depot and some VCA and other vet clinics across Ontario)
         2) Recycle products like: Nespresso pods, Febreeze air fresheners, Swiffer products, writing instruments.
         3) NB: All the “free” programs are sponsored by the manufacturer, so are limited to that company’s products, and may require taking the items elsewhere.
         1) Paid boxes are more accepting and convenient than the free programs, but at a cost to the consumer
2) TerraCycle offers boxes to recycle everything from PPE to office supplies to coffee products to lunchroom packaging.

3) NB: some of the paid recycling options may be available for less money at staples.ca than direct from TerraCycle.

5) Ditch The Car!
   1) In Canada, 22-27% of all greenhouse gas emissions are due to transportation\(^1\), and roughly 6% of a household’s greenhouse gas emissions are from commuting. 74% of all commuters drive, and only 12% take public transit\(^2\).
   2) Encourage staff to commute in more environmentally-friendly ways: subsidize transit passes, bike share, ebike rentals, and encourage carpooling among staff.
   3) Reduce trips to the clinic by encouraging clients to order food and medications through MyVetStore; or better yet, partner with a local bike courier company to do deliveries of food and medications. In downtown Toronto, consider www.NRBI.com or www.senditcourier.ca.
   4) Reduce the number of VP/CDMV orders to 1/week.
   5) Personal Challenge: replace 1 car trip a week with walking, cycling or public transit.

6) BONUS ACTION:
   1) Consider donating to environmental causes that are important to you.
   2) 1% For The Planet is an organization that certifies companies that give 1% of their sales back to environmental organizations. https://onepercentfortheplanet.org/

Sources:
2) https://legacy.equiterre.org/sites/fichiers/fmm_transportation_recs.pdf
THE SAFE PROVISION OF VETERINARY CARE TO NON-TRADITIONAL ANIMAL ATTRACTIONS IN ONTARIO

Graham Crawshaw, BVetMed, MS, DACZM
Crawshaw Wildlife Consulting, Pickering, ON
Christopher Dutton, BVSc, MSc, DECZM (ZHM), DACZM
Zoo and Wildlife Veterinary Consultant, Markham, ON

There are many variations in the kinds of facilities that hold wild, exotic, or non-traditional animals. Veterinarians in practice are, for the most part, familiar enough with exotic pets such as reptiles or parrots to be able to provide medical care themselves or direct clients to clinics with more willingness or expertise. Most birds and small reptiles can be transported to a clinic.

However, there is also an increasing number of clients who own species more typically seen in zoos that cannot be readily handled and therefore require on-site care. These animals may be found in wildlife rehabilitation centres, farms with large wild or semi-domestic species, and zoos and exotic animal sanctuaries.

What and where are these non-traditional, non-domestic, exotic, or wild animals? The species range from bats to elephants with everything in between and include large rodents, non-human primates, small and large wild cats, and bison, as well as semi-domesticated camelids, emus, and large reptiles. There are also a few commercial deer farms in Ontario.

Zoos in Ontario include large institutions, such as the Toronto Zoo and African Lion Safari, and many so-called roadside zoos. The larger and perhaps more legitimate zoos are members of, and accredited by, CAZA – the Canadian Association of Zoos and Aquariums. To achieve this distinction, they are required to demonstrate a higher standard in animal care and welfare, veterinary supervision, conservation, education, finance, public facilities, and safety and security.

There is no legislated system for licensing exotic animal collections in Ontario, although for native wildlife, the Ministry of Natural Resources (actually MNDMNRF) requires owners to obtain a permit to keep them. Typically, the rarer “Species-at-Risk” would not be eligible.

There are no federal or provincial regulations covering exotic or even potentially dangerous animals if they were bred in this country and not subject to CITES regulations which cover the import/export and trade in endangered and threatened species. However, many municipalities, especially urban ones, have enacted their own by-laws to prohibit certain species, notably dangerous animals, or permit only named species.

So, what laws or agencies are in place for protecting wildlife in Ontario or Canada? At the Federal level, a private member’s bill to amend the Criminal Code, otherwise known as the Jane Goodall Act, has been introduced to the Senate and may or may not come into law in the future. The bill, supported by several special-interest groups, is designed to help address the commercial global wildlife trade and contribute to the phase-out of roadside zoos in Canada by banning new captivity for many wild species, as well as having other aims.

One piece of legislation that is in effect is the Ontario Provincial Animal Welfare Act (PAWS) which regulates the care of animals and stipulates the basic standards of care that apply to all animals. The PAWS Act also includes specific regulations pertaining to wildlife.
For one hundred years from 1919, the OSPCA was responsible for enforcing provincial animal welfare legislation. In 2019, a judge ruled that it is unconstitutional for the province to allow a private charity to have policing powers without government oversight. So OSPCA gave up neglect and cruelty investigations and, in 2020, the Provincial Animal Welfare Services Act (PAWS) replaced the SPCA Act. The Act is enforced by the Ontario Animal Welfare Services inspectorate, a division of the Ministry of the Solicitor General. PAWS inspectors have the authority to enter and inspect any premises undertaking commercial activity involving animals.

PAWS requirements for the basic standards of care for all animals include the need to provide:
- adequate and appropriate food, water, medical attention and care
- ventilation, light and protection from the elements, including harmful temperatures
- sanitary conditions and space to enable natural movement and exercise
- humane euthanasia to minimize pain and distress
- transportation in a manner that ensures an animal’s physical safety and general welfare

The Act excludes accepted practices of agricultural husbandry, hunting and fishing, and veterinarians providing care.

Section 4 of the Regulations covers Standards of Care for Dogs Tethered Outdoors and includes the requirement that all dogs must be off their tether for at least 60 minutes a day, as well as several other requirements.

Beyond the regulations that apply to all animals, there are specific regulations for wildlife and their habitats, and primates in particular:

Section 5: Standards of Care for Captive Wildlife
5. (1) Wildlife kept in captivity must be provided with adequate and appropriate care, facilities and services to ensure their safety and general welfare.
   (2) Wildlife kept in captivity must be provided with a daily routine that facilitates and stimulates natural movement and behaviour.
   (3) Wildlife kept in captivity must be kept in compatible social groups.

Section 6: Standards for Enclosures for Captive Wildlife
6. (1) A pen (or other enclosed structure or area) for wildlife kept in captivity must be of an adequate and appropriate size
   (a) to facilitate and stimulate natural movement and behaviour,
   (b) to enable each animal in the pen to keep an adequate and appropriate distance from the other animals and people so that it is not stressed, and
   (c) to ensure that the natural growth of each animal is not restricted.
   (2) A pen for wildlife kept in captivity must have
   (a) features and furnishings that facilitate and stimulate the natural movement and behaviour of each animal in the pen,
   (b) shelter from the elements that can accommodate all the animals in the pen at the same time,
   (c) surfaces and other materials that accommodate the natural movement and behaviour of each animal in the pen,
   (d) one or more areas that are out of view of spectators, and
(e) one or more sleeping areas that can accommodate all the animals in the pen at the same time and that are accessible to all the animals at all times.

(3) A pen for wildlife kept in captivity must be made of and contain only materials that are
(a) safe and non-toxic for the animals kept in the pen or other enclosed structure or area, and
(b) of a texture and design that will not bruise, cut or otherwise injure the animals.

(4) A pen for wildlife kept in captivity and any gates or other barriers to it, including moats, must be designed, constructed and locked or otherwise secured to prevent
(a) interaction with people that may be unsafe or inappropriate for the wildlife,
(b) animals escaping from the pen by climbing, jumping, digging, burrowing or any other means, and
(c) animals or people, other than people who are required to enter the enclosure as part of their duties, from entering the pen by climbing, jumping, digging, burrowing or any other means.

(5) A pen for wildlife kept in captivity and any gates or other barriers to it, including moats, must be designed, constructed and maintained in a manner that presents no harm to the wildlife.

Section 7: Standards of Care for Captive Primates

7. Every primate kept in captivity must be provided with
(a) daily interaction with a person having custody or care of the primate,
(b) a varied range of daily activities, including foraging or task-oriented feeding methods, and
(c) interactive furnishings, such as perches, swings and mirrors.

There follow large sections covering marine mammals.

Failure to provide any of the above is deemed “DISTRESS.” The Act defines this as the state of being:
(a) in need of proper care, water, food or shelter,
(b) injured, sick, in pain or suffering, or
(c) abused or subject to undue physical or psychological hardship, privation or neglect.

If inspectors have the opinion that there are significant deficiencies, they will issue either a Letter of Non-compliance or, in more acute cases, an Order to Alleviate Distress (Section 30). The owners of the animal(s) are then required to correct the deficiencies in a specified time.

If orders have been issued and ignored, and the animals are still “in Distress”, inspectors can remove the animals from the property. If no orders have been issued previously, a veterinarian must advise the inspector in writing that alleviating the animal’s distress necessitates its removal, so the responsibility then falls upon the veterinarian and, if appealed, this may well end up in an appeals tribunal or court.

In cases of extreme neglect or apparent cruelty, animals may be considered to be in CRITICAL DISTRESS and the animal(s) may be seized and removed by AWS. CRITICAL DISTRESS means distress that requires immediate intervention to prevent serious injury or to preserve life.
The Act also includes the legal obligation for a veterinarian to report abuse or distress of any species:

**Section 14: Obligation to Report**

14. Every veterinarian ... who has reasonable grounds to believe that an animal is being abused, being subject to undue physical or psychological hardship, privation or neglect, including by participating in fights with other animals, or is being trained to fight another animal shall report his or her belief to an animal welfare inspector.

**References**

Ontario Provincial Animal Welfare Services Act [https://ontario.ca/laws/statute/19p13].

**A Veterinarian’s Role**

So, what is a veterinarian’s role with such facilities, and how can care be achieved legally, effectively, and safely?

As mentioned previously, animal species can include a variety of small and large mammals such as primates, carnivores, and ungulates, birds of various sizes, and even reptiles. Care may be achieved on-site or may require transfer to a more permanent veterinary facility if the patient is manageable. When we asked the College of Veterinarians in August 2022 what category of facility accreditation would be required to see this eclectic mix of animals, for example, remote area companion mobile perhaps, they replied:

“Because our current Minimum Standards for Veterinary Facilities in Ontario do not have a specific category for these types of species, it would be best for the veterinarian to contact the College to discuss what type of veterinary services they wish to provide, the kinds of species, and whether they want to accredit a building or have a mobile. We’d then need to discuss this with the Registrar who would then possibly make a referral to the Accreditation Committee in order for the Committee to place terms, limitations, and conditions on the certificate of accreditation.”

A new model of accreditation is currently being piloted that may provide more flexibility.

The decision to provide effective care to such animals isn’t always an easy one. In well-trained or more domesticated individuals, examination, sample collection, and treatment may be non- or minimally invasive and stress-free, allowing an accurate fast diagnosis, effective treatment, and, therefore, a greater success rate. In contrast, for less habituated individuals and dangerous species, or more invasive procedures, physical and/or chemical restraint will be required with its associated stressors and risks. For example, animals removed for examination may never be successfully returned to their family group, and follow-up treatments in some individuals may be impossible. Furthermore, for some species, diagnosis can be very challenging due to a lack of “normal” values, and there may be risks of a particular drug or treatment.

Once handling is considered necessary, several factors determine the type of restraint required. These include the species; the individual’s physiological, emotional, and health states; the enclosure or handling facilities and whether the animal needs to be moved off-site; the objective and length of procedure; the equipment and expertise available; the environmental conditions; and any inherent risks.
The simplest forms of “restraint” in domesticated and well-trained wild species can be psychological through acclimation and operant conditioning. Unfortunately, effective training for medical procedures is extremely labour-intensive and likely rare in these facilities. The smaller animals may be handled with thick gloves, caught in hoop nets, or trapped in squeeze cages for treatment or before sedation, if such equipment is on-hand [www.wcscanadastore.com; www.animal-care.com]. A responsible wild animal facility should have these items available. Once safely confined, the animal may be moved to a secure area or a clinic for the procedure.

For remote chemical immobilization, specialist items of equipment may be bought (some may also be constructed). They include pole syringes, blowpipe pistols, and dart guns. Darts are typically projected by a compressed gas, usually carbon dioxide, or a powder charge. The darts themselves come in two main forms, depending upon the methodology for drug injection into the animal:
1) Charged, slow-release type, utilizing compressed air or butane within the dart [www.daninjectcanada.ca], or
2) Uncharged, rapid-release type, utilizing a charge within the dart [www.pneudart.com].

We recommend two systems:
1) Dan-Inject Pistol Model CO2 PI, with both the 11 mm and 13 mm bore barrels, and a variety of blow darts (1.5 ml, 3 ml, 5 ml, and 10 ml) and needles (1.5 mm x 25 mm, plain) [www.daninjectcanada.ca], for more gentle, close-range shots (up to 5 m), or
2) Pneu-Dart X2 Pistol, with a variety of type P blow darts [www.pneudart.com], for more reliable and distant shots.

Choosing the correct immobilizing drugs for such a variety of species can be difficult and may include drugs or concentrations not commonly found in veterinary practice. The ideal drug combination should be:
1) Suitable for many (all) of these species with multiple routes of administration, although intramuscular is the most common route,
2) Be highly potent and concentrated, so that only a small but measurable volume is required, minimizing the size of injection and blow dart, and
3) Be readily reversible so that the patient is standing and behaving normally as quickly as possible, particularly if it needs to go back into a group.

Unfortunately, many of the standard formulations of veterinary anesthetics are insufficiently concentrated to be used in the darts and remote systems for larger animals. Other drugs, widely used in zoo and wildlife medicine, may not be licensed in Canada. Chiron Compounding Pharmacy [www.chironcompounding.com] in Guelph is a distributor of some wildlife-specific formulations:
- Medetomidine 10 mg/ml and 20 mg/ml
- Ketamine 200 mg/ml
- Xylazine 300 mg/ml
- Butorphanol 50 mg/ml
- BAM (a pre-mixed 11 ml formulation containing 27.3 mg of butorphanol, 9.1 mg of azaperone, and 10.9 mg of medetomidine designed to provide a low-volume, reversible combination for numerous wildlife species).
Telazol™ (a rapid-acting anesthetic combination of tiletamine and zolazepam for use in dogs and cats in the USA) can be obtained through Zoetis Canada [www.zoetis.ca] but an Emergency Drug Release is required from Health Canada.

Common combinations include ketamine-xylazine, ketamine-medetomidine/dexmedetomidine, Telazol, Telazol-xylazine, Telazol-medetomidine/dexmedetomidine, and BAM.

Although I haven’t mentioned the ultra-potent opioids here, since they are difficult to acquire, I believe that the alpha-2 agonists, particularly in concentrated forms, are an underestimated hazard to human life and an SOP dealing with the dangers of human exposure to these potent drugs must be prepared, equipped (with appropriate human reversals), and practiced.

Once the animal is suitably immobilized, then examination, preventative care, diagnoses, and treatments can proceed. Preventative medicine and surgery are extremely important for any non-traditional animal collection although are often disregarded. Veterinary involvement can include recommendations about hygiene and the control of pests and unwanted domestic animals on-site that can spread disease to the more susceptible “exotic” species, for example, toxoplasmosis to macropods and lemurs.

Collection animals should ideally undergo routine examinations, be uniquely identified, receive vaccinations and parasite control, and any animals entering the collection should be quarantined until confirmed free of disease. Nutritional advice is often essential and nutritional diseases, such as metabolic bone disease and obesity are far too common. In private facilities, animals are commonly removed from their parents at a young age for acclimation and hand-rearing, so advice on neonatal care and nutrition may also be necessary. Finally, reproduction and, more likely, sterilization or contraception may be required, particularly with the introduction of the various new laws described previously.

With good medical care, non-traditional animal species can live far longer than their wild counterparts and so geriatric medicine, regular welfare assessments (perhaps commencing when they reach 80% of their expected lifespan), and proactive euthanasia should be considered, although this is often a subject that is not welcomed by their owners.

To conclude, the provision of veterinary care to such facilities can be achieved but does involve considerable time, further education, and the purchase of expensive equipment and drugs that may only be used occasionally. It is difficult to know whether the costs of such veterinary care will be borne by the non-traditional animal attractions, many of whom find it difficult to fund the most basic of enclosures and husbandry of their collection.

Further information and advice can always be sought from zoo and wildlife medical specialists, such as those who are Diplomats in the American College of Zoological Medicine [www.aczm.org]. Having suitable veterinary reference material is essential and the following are some recommended textbooks:

TOP 10 TIPS FOR AN EXCELLENT EYE EXAM
David J. Maggs BVSc (Hons), Diplomate ACVO

Hint # 1: Avoid sedation
Sedation alters or obscures almost every part of the eye exam. It lowers the Schirmer tear test (STT) result and the intraocular pressure (IOP), it renders assessment of all the critical neuro-ophthalmic responses and reflexes almost impossible to interpret (dazzle reflex, pupillary light reflexes (PLRs), menace response, oculocephalic (“doll’s eye”) reflexes, and palpebral reflex), and causes enophthalmos, ptosis and third eyelid protrusion that collectively obscure much of the eye. As a result, sedation should be avoided whenever possible. Even in a practice where eye exams are all we do, we need to sedate a patient only a few times each year. If it is impossible to conduct the exam without sedation then I recommend that you use the lightest dose possible of a mildly anxiolytic drug. Recall that no part of the eye exam is painful; you require only that the patient is reasonably still and compliant.

Hint # 2: Get at eye level with your patient
Recalling that the eye exam requires the examiner to look through a series of 34 approximately concentric apertures which sequentially reduce in diameter (the orbital rim, palpebral fissure, cornea, and pupil), it is essential that the examination angle is well aligned with the axis of these apertures. So much is missed if the patient is on the floor when examined since, as the head is tilted up by the examiner, the eye rolls down. Instead, put the patient on an exam table and sit in front of the patient at eye level (just as the physician ophthalmologist does with you!).

Hint # 3: Dim the ambient light
The vast majority of the eye is transparent – the tear film, cornea, aqueous humor, lens and vitreous. Therefore, we judge the health and pathology of these tissues by assessing their clarity and surface reflections. This requires a focal light source but is greatly enhanced by eliminating stray, scattered, and confusing/disturbing reflections. Therefore, we should conduct all eye exams in a darkened room. It’s amazing what you miss with the lights on or, worse still, outdoors.

Hint # 4: Use a bright light source and magnification
Because the eye exam requires the examiner to look through the aforementioned series of concentric apertures of sequentially reducing diameter, the inside of the eye is difficult to illuminate without a bright and focal light source such as the Finoff® transilluminator. Additionally, because very small abnormalities can be painful and blinding, a complete eye exam cannot be done without a means of magnification (e.g., Optivisor® head loupe or pharmacy “reader” glasses). Think about how small yet painful and potentially blinding an ectopic cilium is.

Hint # 5: Take an orderly approach
I suspect that you conduct your general physical exams in a repeatable order that suits you and that you have developed so as to be complete. Likewise, the ophthalmic exam should always be orderly and complete. My preference is to examine both eyes and the entire head from a distance, assess all relevant cranial nerves (II through VII), and then to examine each eye from peripheral to axial and from superficial to deep. To do this, I make a mental checklist which includes periocular skin, eyelid margins, cilia, palpebral, fornical, and bulbar conjunctiva, third eyelid, sclera, cornea, limbus, anterior chamber, iris, and axial lens. Following dilation, the remainder of the lens, as well as the vitreous and fundus can be examined. I prefer to examine the unaffected eye prior to the affected eye, and to use a prepared exam sheet.
Hint # 6: Tilt the nose down so that the eye rolls up
Not only should the exam always be conducted with the examiner and the patient at eye level
with each other, at points during the exam, the patient’s nose should be tilted down such that the
eye rolls dorsally. This permits examination of the ventral 1/3 of the eye (conjunctiva, sclera,
cornea, iris, and especially the anterior chamber where small amounts of hypopyon, hyphema,
fibrin, or keratic precipitates collect due to gravity).

Hint # 7: “Always”:
  a) Retroilluminate
  Retroillumination is a simple but extremely useful technique for assessment of pupil
symmetry (anisocoria) and all parts of the transparent ocular media (tear film, cornea,
aqueous, lens, and vitreous). A focal light source held close to the examiner’s eye and
directed over the bridge of the patient’s nose from at least arm’s length is used to elicit the
fundic reflection or reflex. This is usually gold or green in tapetal animals or red in atapetal
individuals. Each eye is illuminated equally and the fundic reflex is used to assess and
compare pupil size, shape, and equality. Additionally, opacities in the ocular media will
obstruct the fundic reflection and are noted for more detailed subsequent examination using
transillumination or retroillumination again after pupil dilation. Both of these subsequent
techniques can be augmented by magnification. Retroillumination is particularly useful for
differentiating nuclear sclerosis from cataract.

  b) Check for aqueous flare
  Aqueous flare is a pathognomonic sign of uveitis and is due to breakdown of the blood-
aqueous barrier with subsequent leakage of plasma proteins into the anterior chamber.
Aqueous flare is best detected using a very focal, intense light source in a totally darkened
room. The passage taken by the beam of light is viewed from an angle. In the normal eye, a
focal reflection is seen where the light strikes the cornea. The beam is then invisible as it
traverses the almost protein- and cell-free aqueous humor in the anterior chamber. The light
beam is visible again as a focal reflection on the anterior lens capsule and then as a diffuse
beam through the body of the normal lens due to presence of lens proteins. If uveitis has
allowed leakage of plasma proteins into the anterior chamber then these will cause a
scattering of the light as it passes through the aqueous. Aqueous flare is therefore detected
when a beam of light is seen traversing the anterior chamber and joining the focal
reflections on the corneal surface and the anterior lens capsule. A slit lamp provides ideal
conditions for detecting flare; however, the beam produced by the smallest circular aperture
of the direct ophthalmoscope held as closely as possible to the cornea in a completely
darkened room and viewed transversely will also provide excellent results. The slit beam on
the direct ophthalmoscope is not as intense and does not provide as many “edges” of light
where flare can be appreciated most easily. Assessment of flare may be easier after
complete pupil dilation due to the apparent dark space created by the pupil. Combined
assessment of IOP and aqueous flare should be performed whenever glaucoma or uveitis is
suspected because of the frequency with which these conditions co-exist.

c) Measure intraocular pressure (Tonometry)
  Tonometry is essential for differentiation of the three major conditions in which red-eye is
the hallmark feature – conjunctivitis, uveitis, and glaucoma. The availability of easily used
and reasonably priced tonometers such as the Tonopen® or TonoVet® make measurement
of IOP easy in all species, particularly cats. Across large populations, normal canine and
feline IOP is reported as approximately 10-25 mmHg. However, some variation is noted
between individuals, technique, and time of day. Comparison of IOP between right and left
eyes is therefore critical to interpretation of results. A good rule of thumb is that IOP should not vary between eyes of the same patient by more than 20%.

While the obvious application for tonometry is the diagnosis of glaucoma (where IOP is generally elevated), it should also be used to confirm the diagnoses of uveitis and conjunctivitis. In uveitis, IOP is lowered due to loss of function of the inflamed ciliary body, and in conjunctivitis the IOP must be normal. Subsequently, tonometry is used to monitor progression or resolution of uveitis and glaucoma, and to guide medication adjustments.

d) Apply fluorescein (but do it last)
Application of fluorescein is an essential part of every eye exam but it affects the interpretation of almost all other parts of the eye exam, and so must be done last. In particular, be sure to measure the STT and IOP (using the TonoVet), and to collect surface samples for culture or cytologic examination prior to fluorescein application. Also, carefully examine the entire globe before staining as the fluorescent green dye will mask or alter the appearance of many ocular pathologies.

Hint # 8: “Always” dilate the pupil to examine the lens, vitreous, and fundus
Examination of any tissues behind the iris is markedly limited by pupillary constriction, especially when a bright light source is used. For this reason, pupillary dilation is essential if one is going to record that they conducted a complete eye exam. Note that I am not saying that dilation is essential in every patient with presenting signs of ocular disease; rather, I am suggesting that we should note in the record when only a nondilated exam was performed. However, if a patient is presented for signs suggestive of posterior segment disease (vison disturbance for example), or if the anterior segment exam suggests posterior segment involvement (e.g., altered menace response, PLRs, or dazzle reflex), or the presence of lenticular opacity (nuclear sclerosis or cataract), then a complete assessment of that patient must include pupil dilation. This is easily achieved about 15 mins following application of 1 drop of tropicamide.

Hint # 9: Always make an etiologic diagnosis
At the end of the eye examination the examiner should have determined which ocular tissues are involved, and by what sort of pathologic process they are being affected; specifically, is it an inflammatory “-itis” or a noninflammatory “-opathy”). However, stopping there will not permit targeted therapy. For example, there is no treatment for “conjunctivitis” other than empirical antiinflammatory therapy. By contrast, there are excellent and specific treatments for herpetic conjunctivitis, foreign body conjunctivitis, allergic conjunctivitis, keratoconjunctivitis sicca, etc. (and some of these will be worsened by a topical anti-inflammatory!). Therefore, it is essential that, after completing the exam, the examiner makes a clinical diagnosis, and before selecting a treatment, the clinician puts an adjective in front of the clinical diagnosis – even if that adjective is “idiopathic”. In my opinion, no medical record should contain a diagnosis of “conjunctivitis” (for example) without an added qualifier.

Hint # 10: Look, look, and look again.
Never were the words “More is missed through not looking than not knowing” truer than for the eye exam where, following completion of a thorough exam, the diagnosis is almost always made without any other diagnostic tests.

References available from the author on request.
Is This Normal or Not?
Radiographic Normal Variants to Look Out For
Shawn Mackenzie, DVM, DVSc, DipACVR

What is normal?
• Determining normal from abnormal can be challenging
  • Diverse population
  • Many congenital or developmental changes that are clinically insignificant
  • Superimposition of structures
  • “Create something from nothing”
  • E.g. Pharyngeal region often over-interpreted in animals with upper airway clinical signs

General Approach
• Symmetry
• Compare to contralateral limb
• Is it repeatable
• Oblique radiographs
• References

Physis
• Often mistaken as fractures
• Important to know closure times
  • Most by 12 months old
• Use symmetry or opposite limb


Acetabular Physis
• Appears at 2-3 months of age
• Closes by 5 months

Tibial Tuberosity Physis
• Appears at 7-8 weeks
• Closes by 12 months in dogs and 10 months in cats
Acetabular Fossa

Nutrient Foramen

Sesamoid bones
- Supinator muscle

Sesamoid bones
- Abductor pollicis longus

Sesamoid bones
- Iliopubic Sesamoid bones

Origin of the Long Digital Extensor Tendon
Retained Cartilage in Tibial Tuberosity

Decreased Opacity at Lateral Aspect of Radius

Summation of ear over pharynx

Thyroid and cricoid cartilage mineralization

Thyro- and Cricopharyngeal Muscles

Costochondral Junction Mineralization
Cranial Mediastinal Reflection & Cupula of Left Cranial Lung Lobe

Skin Folds

Pulmonary Osseous Metaplasia (Pulmonary Osteomas)
Visible Dorsal Tracheal Membrane

Superimposition

Aorta in Older Cats

Fluid Filled Pylorus
GI Wall Thickness

Recap

- Differentiating normal variants from abnormal can be challenging
- Use the tools you have
  - Symmetry
  - Other limbs
  - Oblique radiograph
- References

Cecum

Deep Circumflex Iliac Arteries and Veins

References

SURGICAL MANAGEMENT OF GDV
Howard B. Seim III DVM, DACVS
Colorado State University

If you would like a copy of this surgical procedure on DVD go to www.videovet.org.

Key Points
• Survival is generally determined by early and appropriate presurgical management
• Patients referred for surgery should be decompressed prior to referral with continued decompression provided during transport
• Incisional gastropexy results in a fast, easy, permanent adhesion
• Ventricular tachycardia is a common postoperative complication
• Gastric necrosis signals an unfavourable prognosis

Introduction: Patients with GDV are considered critical care cases; every minute of presurgical treatment is vital to a successful outcome. Survival is generally determined by early and appropriate presurgical management and urgent surgery as soon as the patient is stabilized. Efficient presurgical treatment usually involves a minimum of two people. Gastric decompression and shock therapy should be done simultaneously. If this is not possible; decompression should be performed first. It is stated that gastric decompression is the single most important factor in reversing cardiovascular deficits in patients with GDV.

Decompression: Generally, orogastric intubation can successfully be performed in 80 - 90% of GDV patients. If orogastric intubation is unsuccessful decompression via right flank needle puncture is indicated. It is also suggested that right flank needle puncture is recommended as a first attempt at decompression in severely depressed metabolically deranged patients.

Orogastric Intubation Technique: The stomach tube is measured to the last rib and marked with a piece of tape. A stiff GDV, foal or mare stomach tube with a smooth bevelled tip works best (having several diameter and stiffness tubes is ideal). Apply generous lubrication to the tube. Place a functional mouth speculum; generally a roll of 2” tape secured in the mouth with tape encircling the muzzle. As the stomach tube is passed, you will often meet resistance at the lower esophageal sphincter. Pass the tube firmly in a twisting manner to encourage the tube to pass through the lower esophageal sphincter. Pass the tube firmly in a twisting manner to encourage the tube to pass through the lower esophageal sphincter.

If unsuccessful, place the patient in various positions and attempt to pass the tube (i.e., elevate animal at 45 degree angle with rear feet on floor and front feet on the table, right lateral recumbency, and left lateral recumbancy). This movement may encourage the stomach to rotate enough to allow the tube to pass into the stomach. Be careful not to position the patient in dorsal recumbancy as this will increase abdominal visceral pressure on the caudal vena cava and may exacerbate signs of shock.

If still unsuccessful, try different diameter tubes; try a smaller diameter, more flexible tube and proceed as described above.
If still unsuccessful, attempt to remove some of the air in the stomach by placing an 18 gauge needle at the point of distention in the right flank region. Ping the area to make sure the spleen is not under the proposed trocarisation site. After trocar decompression, attempt to pass the stomach tube as described above.

If still unsuccessful, sedate the dog with a narcotic (e.g., Oxymorphone) and try to pass the tube again. Mild sedation is recommended if the patient strongly resists physical restraint.

Success in passing a stomach tube depends on the skill of the operator and available assistants.

If you are successful at passing a stomach tube and plan to refer the patient to a referral surgical center for gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy incision or place a nasogastric tube.

If a stomach tube was successfully passed, stomach contents should be evaluated for color and presence or absence of necrotic looking gastric mucosa. This may give an impression of gastric viability.

**Fluids:** Shock dosage of polyionic isotonic fluid is carefully administered to expand the vascular compartment. Patients are frequently monitored during fluid administration to help determine ultimate fluid rate and amount. One or two indwelling cephalic catheters are generally placed.

**Referral:** If you are successful at passing a stomach tube and plan to refer the patient to a referral surgical center for gastric derotation and gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy as described below.

Pharyngostomy tube placement:

a. Orally palpate the fossa lateral to the hyoid apparatus until a lateral bulge is seen.

Pharyngostomy tube placement:

b. Make a small skin incision over the bulge and press a curved forceps (substitute for finger) through the soft tissues and skin incision.

c. Pull the stomach tube through the incision with curved forceps; then pass the tube over the arytenoid cartilages, down the esophagus, and into the stomach (measure to the 13th rib).

Disadvantages include: heavy sedation or general anesthesia is necessary for placement of the tube.

Rarely a temporary gastrostomy may need to be performed.

The patient is placed in left lateral recumbancy with the right flank area clipped and surgically prepared. Heavy sedation and local infiltration of lidocaine or light general anesthesia is performed. A 4 - 5 cm incision is made in the skin over the point of greatest gastric distention (generally 1 - 2 cm caudal to the 13th rib and 2 - 3 cm distal to the transverse processes of the lumbar vertebrae). A grid technique is used to gain entrance into the peritoneal cavity. Due to severe gastric distention the stomach wall is pressed against the
abdominal wall and thus easily identified through the flank incision. The stomach wall is sutured to the skin using a simple continuous pattern with 3-0 Maxon. This is done prior to incising into the stomach lumen. A #11 BP scalpel blade is used to puncture into the lumen of the stomach. Gas and stomach contents are expelled under pressure so stand back! The gastric mucosa is evaluated for viability. Disadvantages of gastrostomy include: the stomach is sutured in its rotated position and more time is required when definitive surgical treatment is performed due to the necessity of closing the gastrostomy.

**Successful stomach tube placement:** Once the stomach tube has been passed into the stomach or gastrostomy performed, the stomach is lavaged with warm water. If a stomach tube was successfully passed, the stomach contents should be evaluated for color and presence or absence of necrotic gastric mucosa. This may give an impression of gastric viability.

**Surgical Treatment:**
A specific ‘Surgical Plan’ should be in mind before entering the operating room theatre. This will improve the efficiency of surgery and thus decrease overall surgery time. The ‘authors’ surgical plan is as follows:

Stand on the right side of the patient.

Provide generous abdominal exposure via xyphoid to pubis midline laparotomy.

Remove of all of the falciform ligament to the level of the xyphoid.

Place a 10” Balfour self-retaining abdominal retractor (metal frame toward the head) with full retraction.

Confirm that the omentum is draped over the exposed surface of the stomach (pathognomonic for GDV).

Attempt derotation by:
Standing on the patients' right side, first reach your right hand across the abdomen and place it between the left body wall and dilated stomach.
Slide your right hand along the sublumbar body wall and grasp the deep (dorsal) aspect of the stomach at the level of the spine.
Next, place the open palm of your left hand on the exposed surface of the right side of the dilated stomach.
Using both hands simultaneously, pull the deep part of the stomach with your right hand to begin derotation whilst you push the right surface of the stomach down toward the patients sublumbar body wall with your left hand. This maneuver will be successful in the majority of cases.


Once the stomach is derotated, evaluate the stomach for evidence of questionable viability (particularly the greater curvature and fundus) and for evidence of gastric motility.
Next, exteriorize the spleen from the abdominal cavity. Evaluate color, texture, blood flow (splenomegaly is often present and is NOT an indication for splenectomy). Splenectomy is rarely performed but may be necessary if splenic vessels are thrombosed (veins feel like threads or rubber bands).

If the stomach is full of air or fluid it should be emptied prior to attempting derotation.

If the stomach is full of food and several attempts to derotate (see author’s technique above) are unsuccessful, perform a gastrotomy and manually remove the food from the stomach lumen. Suture the gastrotomy and attempt derotation again.

Commence your gastropexy procedure.

**Incisional gastropexy:** This technique is based on a 3-4cm long seromuscular antral incision sutured to a similar length incision in the transversus abdominus muscle. This is the authors’ technique of choice for permanent gastropexy.
With the Balfour retractors still in place visually locate the ideal position for the antral wall incision. It should be located equidistant between the pylorus and gastric incisure and equidistant between the greater and lesser curvature of the stomach. A 4cm longitudinal sero-muscular incision is made in this antral location. An easy way to safely make the sero-muscular incision is to grasp the full thickness antral wall with your thumb and finger at the site of the proposed incision, gently retract the wall of the stomach until you feel the mucosa and submucosa ‘slip’ out of your thumb and finger. The tissue remaining between your thumb and finger is the sero-muscular layer of the antral wall. Using a straight or curved Metzenbaum scissors cut the tissue remaining in your thumb and finger resulting in a perfect depth of the sero-muscular incision. Extend the incision to a 4cm length and gently undermine the edges to allow generous suture bites in the stomach wall during gastropexy.

Once the antral incision is completed remove the Balfour retractors. When selecting the location on the transversus abdominus muscle for the gastropexy, it is important to first visualize the location of diaphragmatic muscle fibers as they radiate into the abdominal cavity and attach near the costal arch. It is important that the gastropexy site be at least 2cm caudal to the diaphragm muscle insertion. After identifying the attachment of the diaphragm, the bleeding surface of the antral incision is brought to the right body wall. With the stomach in a normal position, the bleeding antral surface is touched to the peritoneal wall approximately 3-4 cm deep to the abdominal wall incision and 2cm caudal to the insertion of the diaphragm. A blood mark is created on the peritoneum at this proposed location. This will be the site for the permanent gastropexy. The peritoneum and transverses abdominus muscle are then incised creating a mirror image defect of the antral incision. The incisional defect in the stomach is then sutured to the incisional defect in the abdominal wall. The defects are sutured in two layers using a simple continuous pattern with 2-0 or 3-0 monofilament or multifilament synthetic absorbable suture.

**Belt Loop Gastropexy:** This technique is based on the construction of a sero-muscular antral flap attached around a segment of transversus abdominus muscle. A horseshoe shaped incision is made in the serosal layer of the antral portion of the stomach with its base at the greater curvature. The sero-muscular portion of the stomach is identified by grasping full thickness antral wall between the thumb and index finger and “slipping” the mucosal and submucosal layers away so only the sero-muscular portion of the wall remains between thumb and finger. The sero-muscular layer is incised with scissors and the horseshoe shaped sero-muscular antral flap is dissected and elevated of the submucosal layer. The stomach is replaced in the abdominal cavity in normal position and the sero-muscular flap lined up with the transversus abdominus muscle. Once this optimal location is discovered, two longitudinal incisions (along the fibers of the transversus m.) are made in the transversus abdominus m. The segment of muscle between the incisions is undermined. The sero-muscular flap from the stomach (i.e., belt) is passed through the transversus abdominus m. (i.e., loop) and sutured to itself to complete the “Belt-Loop” gastropexy. 2-0 or 3-0 monofilament absorbable synthetic suture in a simple interrupted or continuous pattern is used to secure the flap in place. Advantages of belt loop gastropexy include: it is relatively easy to perform
alone and in the middle of the night, it can be performed quickly, and it is an effective means of permanent gastropexy.

**Postoperative management**

In most cases 3 to 4 days of intensive monitoring is necessary for the successful management of GDV patients. Postoperative considerations are listed below:

a. Shock is a postoperative possibility and the patient should be monitored and treated accordingly.

b. Patients are generally held off food and water for 24 hours following surgery. During this time maintenance fluids should be supplied using polyionic isotonic crystalloid fluid. Vomiting may occur following surgery; the NPO period should be extended accordingly. Gastritis and gastric motility disorder may be seen in post op GDV patients.

c. After 24 hours of no vomiting, oral alimentation should begin gradually with a sequence of ice cubes, water, and finally canned dog food. This should occur over a 2-3 day period.

d. Antibiotics should be continued for 7 - 10 days.

e. Routine surgical complications such as infection, dehiscence, seroma, etc. should be watched for and treated accordingly.

f. EKG monitoring: the most common severe postoperative complication is cardiac arrhythmia. Approximately 75% of GDV patients will develop arrhythmia’s in the immediate postoperative period. Arrhythmia’s can be present at the initial time of presentation but most often occur within 24 - 72 hours after surgery. Ventricular premature contractions, progressing to ventricular tachycardia is most common. Etiology is unknown but shock, hypoxia, acid base alterations, endotoxins, myocardial depressant factor (MDF), reperfusion injury, release of free radicals, and hypokalemia have been identified. Occurrence of a total body potassium deficit has been proposed. Etiology of the hypokalemia includes anorexia, vomiting, tremendous outpouring of potassium rich fluids into a dilated stomach, and use of potassium poor fluids in treatment of shock. For this reason, adding 20-30 mEq of potassium chloride per liter of maintenance fluids during and after surgery are recommended.

g. Gastric motility: occasionally GDV patients develop postoperatove gastric motility abnormalities. Patients with gastric hypomotility or gastric stasis noted at the time of surgery should be treated with a motility modifier (i.e., metaclopramide, erythromycin, etc).
INTESTINAL ANASTOMOSIS
Howard B. Seim III, DVM, DACVS
Colorado State University

If you would like a copy of this surgical procedure on DVD go to www.videovet.org.

Key Points
• Pay attention to basic surgical principles
• Submucosa is the layer of strength
• Use synthetic absorbable suture materials
• Appositional techniques are best
• Intestinal sutures should engage at least 3 - 4 mm of submucosa
• Intestinal sutures should be no further apart than 2 - 3 mm
• Always handle bowel wall using atraumatic technique
• Examine the integrity of your anastomosis visually
• 50 - 60% of the ‘small intestine’ of dogs and cats can be resected

General principles of small intestinal surgery
1) Incorporation of the collagen laden submucosal layer in the surgical closure.
2) Minimize trauma and contamination.
3) Maintain good blood supply to the surgical site.
4) Avoid tension across the suture line as this may increase the possibility of leak and/or breakdown.
5) Pay attention to your established criteria when suturing intestinal defects.

Operative Considerations
1) Proper “packing off” of the surgical field using moistened laparotomy pads should be performed around the exteriorized bowell to prevent accidental abdominal contamination from intestinal contents.
2) Keep abdominal contents warm and moist throughout surgery with a warm, balanced electrolyte solution.
3) Handling abdominal viscera should be kept to a minimum. Gentle manipulation of intestine with moistened gloves or stay sutures is helpful in preventing unnecessary tissue trauma. DeBakey forceps are the most atraumatic forceps for handling abdominal visceral organs.
4) The collagen laden, tough submucosa is the layer of strength in the small intestine; this layer must be incorporated into any small intestinal closure.
5) It may be difficult to visualize the submucosal layer due to mucosal eversion. Visualization of submucosa may be enhanced if everted mucosa is trimmed away.
6) Intestinal contents should be "milked" away from the anastomosis site. Intestinal clamps (e.g., Doyen intestinal clampS, Alice tissue forceps with a rubber feeding tube interposed, hair clips, or Penrose drains) may be used to prevent intestinal contents from contaminating the surgical site whilst manipulating intestine during anastomosis.
7) The anastomosis should be irrigated prior to its return to the abdominal cavity and instruments and gloves changed prior to abdominal closure.
8) Abdominal lavage with 2-3 liters of body temperature, sterile, physiologic saline solution should be accomplished prior to closure. The objectives of repeated abdominal lavage include dilution of bacteria and endotoxin and mechanical removal of fibrin and necrotic debris. The fluid of choice is body temperature, sterile, physiologic saline solution with no
additives (i.e. betadine solution, chlorhexidine, antibiotics, etc). Lavage solution is poured into the abdominal cavity using a sterile stainless steel bowl, the abdominal viscera gently aggitated, and fluid and debris suctioned out with a suction device and a Poole suction tip. Injecting antimicrobials or other products into the abdominal cavity is not recommended.

**Suture Material**

**Absorbable suture**

Catgut. Catgut is NOT recommended for any visceral organ surgery. Its unpredictable absorption and rapid loss of tensile strength in such situations may result in an unacceptably high number of anastomotic leaks and/or breakdowns. Use of catgut suture in gastrointestinal surgery is not recommended.

Dexon, Polysorb, and Vicryl. Synthetic absorbable braided suture (i.e., polyglactin, polyglycolic acid) have become very popular. The braided nature however does result in increased tissue drag and difficult knotting ability.

Biosyn and Monocryl. These sutures have similar properties to Dexon, Polysorb and Vicryl however they are monofilament. They were developed to overcome the problem of tissue drag and knot slipping found in the braided synthetic absorbables. Their predictable hydrolytic absorption is unaffected by their immediate environment (i.e., infection, contamination, hypoproteinemia). They retain high tensile strength for a long period of time (2-3 weeks) and have very good handling characteristics. These suture materials are ideal for use in gastrointestinal surgery. These sutures are the authors choice for gastrointestinal surgery.

PDS and Maxon. PDS and Maxon, are synthetic absorbable monofilament suture materials with similar properties to that of Dexon and Vicryl. They have been shown to retain approximately 70% of their tensile strength at 3-4 weeks, and are absorbed by hydrolysis (unaffected by infection, contamination, hypoproteinemia). These suture materials are ideal for use in gastrointestinal surgery. Possible disadvantages include stiffness, a tendency to kink and prolonged absorption time.

**Nonabsorbable suture**

Nylon, Polypropylene. Monofilament, nonabsorbables are excellent suture materials for use in contaminated or infected surgical sites. They have a high tensile strength, are relatively inert in tissue, noncapillary, and do not act as a nidus for infection. These materials pass through tissue with essentially no tissue drag and have excellent knot tying security at sizes 3-0 to 5-0.

Silk, Mersilene, Bronamid, Vetafil. Multifilament nonabsorbable sutures should NEVER be used in gastrointestinal surgery. They may harbor infection for years and may result in suture related abdominal abscesses or draining tracts.

**Suture size**

For the majority of small intestinal surgical procedures in dogs, 3-0 or 4-0 size suture material is adequate; in cats, 4-0 is recommended. The tensile strength of this size suture is greater than the tensile strength of the tissues that are being sutured (i.e., intestinal wall). Larger size suture may contribute to anastomotic failure by increased trauma to tissues and its effect on the blood supply of tissue margins.
**Needles**
Swaged-on "atraumatic" reversed cutting, narrow taper point, or fine taper-cut needles can all be used for gastrointestinal surgery. The author prefers a narrow taper point needle. Needle diameter should approach the diameter of the suture.

**Suture Placement**
When suturing intestine, sutures should be placed 3 - 4 mm from the cut edge of the intestinal serosa and no more than 2 - 3 mm apart. It is important to recognize everted mucosa and be sure the 3 - 4 mm bite in the intestinal wall is not just in mucosa but engages all layers of the intestinal wall. Measure your intestinal wall bite from the cut edge of the serosa.

**Suture Patterns**
There is considerable controversy regarding specific suture pattern for use in small intestinal surgery. Everting, inverting, and appositional suture patterns have been used experimentally and clinically for suturing enterotomies and anastomoses. Appositional patterns are recommended as they cause little lumen compromise postoperatively.

**Everting:** Everting patterns (i.e., horizontal mattress) have been shown to encourage adhesions and result in lumen stenosis. This technique is **NOT** recommended. The everting technique is not to be confused with the mild eversion of mucosa that occurs in the appositional techniques described below.

**Inverting:** In small animals adequate lumen diameter is an important consideration with any technique. Inverting patterns result in substantial lumen compromise of the small intestine and are **NOT** recommended in dogs and cats.

**Apposition:** Anatomic apposition of individual layers of the bowel wall (i.e., mucosa, submucosa, muscularis, and serosa) result in primary intestinal healing. This technique is superior to inverting or everting techniques because apposition of intestinal margins eliminates lumen compromise. This is the authors preferred technique for suturing all hollow viscus organs in the abdominal cavity. Suture patterns of choice include:

1) Simple interrupted apposing. This technique involves suturing all layers of the intestinal wall and tying the knots on top of the serosa to approximate cut edges. The sutures should be tied tight enough to effect a watertight seal, yet not so tight as to Blanch the tissue and cause ischemia of intestinal margins. This technique is simple, fast, reliable, and does not result in lumen compromise.

2) Simple continuous apposing. This technique is similar to the simple interrupted appositional technique however, a continuous suture pattern is used rather than an interrupted pattern. Advantages include faster anastomosis, equal suture tension over the entire anastomosis, airtight-watertight seal, and mucosal eversion is minimized. This is the authors preferred suture pattern for suturing all hollow viscus organs in the abdominal cavity.

**INTESTINAL ANASTOMOSIS:** Intestinal anastomosis is indicated for resection of nonreducible intussusception, necrotic bowel wall secondary to complete intestinal obstruction, intestinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma).
After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water resistant surface (e.g., plastic drape, crib towel) to prevent ‘strike’ through contamination.

Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60° angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

**Everted mucosa:** Occasionally when the segment of intestine to be removed is amputated mucosa ‘everts’ from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is ‘highly’ recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 – 4 mm of intestinal wall with each suture to guarantee adequate bites in the collagen laden submucosa.

**Bowel lumen diameters:** In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1) Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5-10 mm depending upon bowel diameter (e.g., dog vs cat).
2) In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be incised longitudinally to enlarge the lumen diameter.
3) An end-to-side anastomosis can be performed by closing the larger diameter stoma of the intestinal resection with a single layer continuous apposing suture pattern then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.
4) The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine (this technique is often used for subtotal colectomy in cats).

**Intestinal Anastomosis Technique:**
See the Practical Techniques on GI Surgery I DVD for a detailed video description of this technique (www.videovet.org).

When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to begin suturing at the mesenteric border as this allows adequate visualization of mesenteric vessels and helps prevent encircling these vessels when placing the first few sutures. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short-term (i.e., leakage, breakdown) and long-term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis (see the anastomosis video clip at www.videovet.org for detailed description of the surgery tips below:}
1) First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end without the needle.
2) Place a second stay suture in the antimesenteric borders of each segment to be sutured to bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.
3) Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition. Make certain the lumen diameters of each bowel segment are identical.
4) Using the needled segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3 - 4 mm bite in the submucosa and placing each suture no more than 2 - 3 mm apart (2 mm apart in cats). When the anastomosis is complete, tie the suture to the mesenteric stay suture.
5) If a simple interrupted apposing suture pattern is used, be careful to get a 3 - 4 mm bite in the submucosa and place each suture no more than 2 - 3 mm apart.
6) Evaluate the integrity of the anastomosis. The author’s preference for evaluating the integrity of the anastomotic closure is to visually examine each suture to be certain that suture placement has met your strict criteria (i.e., sutures are no more than 2 - 3 mm apart and have a 3 - 4 mm bite in the submucosa.

Postoperative care
Intravenous fluids to maintain hydration and ensure renal function are continued postoperatively, until the patient begins to eat and drink. Intravenous fluids should then be tapered over a 24 to 48 hour period.

Feeding: Early return to enteral feeding is best for the overall health of the intestine. Feeding the postoperative gastrointestinal surgical patient is generally based on the following criteria:
a) preoperative condition of the patient
b) the condition of the bowel at the time of surgery
c) surgical procedure performed (i.e., enterotomy, anastomosis, pylorectomy)
d) presence or absence of peritonitis
e) postoperative condition of the patient.
The earlier patients can be returned to oral alimentation the better.

Complications
The most common postoperative complication of small intestinal surgery is leakage; leak is either associated with breakdown of the anastomosis or improper surgical technique (i.e., improper suture placement, inappropriate suture material, knot failure, sutures to far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel). A presumptive diagnosis may be accomplished by the following:
1) Body temperature (may be up if acute or down if moribund).
2) Abdominal palpation: periodic, gentle abdominal palpation for pain (gas or fluid?).
3) General attitude (depression-anorexia).
4) Incision: examination of the patients incision for drainage (look at cytology if drainage is present)
5) CBC: leukocytosis followed by leukopenia (sepsis), or a degenerative left shift may imply breakdown.
6) Glucose: low glucose generally implies sepsis (this occurs early in sepsis and may be used as a screening
7) Abdominal radiographs: generally not helpful, they are difficult to critically assess due to the presence of postoperative air and lavage fluid. It can take 1 - 3 weeks for peritoneal air to diffuse from the abdominal cavity after routine abdominal surgery. Time variation is dependant upon the amount of air remaining in the abdominal cavity postoperatively (i.e., large deep chested animal vs a small obese animal).

8) **Abdominal tap** (paracentesis): a four quadrant abdominal tap is accomplished by aspirating fluid using a 5cc syringe and 20 gauge needle or placing a plastic IV catheter into the peritoneal cavity and allowing fluid to drip onto a slide. This may be the most sensitive diagnostic test for determining the presence or absence of intestinal leak.

9) Peritoneal lavage (if paracentesis is not productive): infuse 10-20cc/kg of sterile physiologic saline solution into the abdominal cavity, then gently palpate the abdomen and repeat the four quadrant paracentesis. This technique increases the sensitivity of paracentesis to 90%.

Once fluid has been obtained, a smear should be stained and evaluated microscopically. Depending upon the cell types seen, a determination of the presence of leakage can be made.

Below are examples of expected cytology in patients with and without leak.

1) Healthy PMNs with few degenerate PMNs and a moderate number of red blood cells: This cytology may be expected in any postoperative abdominal procedure (e.g., OHE, abdominal exploratory, cystotomy). Your index of suspicion for anastomotic breakdown should be low. However, if clinical signs continue to deteriorate, repeat paracentesis (2 - 3 times daily, if necessary) to determine the “trend” of the abdominal fluid cytology is recommended.

2) Healthy polymorphonuclear leukocytes with bacteria located intra or extracellularly, degenerate PMNs with intracellular bacteria, free bacteria, or food particles--imply breakdown. Exploratory laparotomy is indicated.

In a recent morbidity/mortality study of patients undergoing intestinal surgery it was found that animals requiring a second abdominal surgery to treat intestinal disorders were less likely to survive than patients requiring only one laparotomy. Also, the longer it took to determine whether or not intestinal leakage had occurred the less likely the patient would survive reoperation. **The take home message is:** pay attention to detail during the first surgery and if a leak occurs, diagnose it and treat it as soon as possible.

**Prognosis** The overall prognosis for uncomplicated GI surgery is excellent. The surgeon must pay attention to detail when suturing any hollow viscus organ with liquid contents.
DIAPHRAGMATIC HERNIA REPAIR
Howard B. Seim III, DVM, DACVS
Colorado State University

If you would like a copy of the video of this surgical procedure on DVD, go to www.videovetorg.

Key Points
• Not all diaphragmatic hernias are life threatening
• Suture the hernial rent from dorsal to ventral
• Use a one layer simple continuous appositional suture pattern for hernia closure
• Evacuate all thoracic air prior to abdominal closure

General Considerations and Indications

Three classifications of diaphragmatic hernia may be diagnosed: acute traumatic, chronic traumatic and congenital diaphragmatic hernia.

Acute Traumatic
This is the most common type of diaphragmatic hernia in dogs and cats. It is generally caused by vehicular trauma but can be caused by any form of trauma.

Chronic Traumatic
This classification of diaphragmatic hernia is seen when a patient has an acute traumatic hernia that was undiagnosed at the time of occurrence. Later (months to years) the hernia is diagnosed due to sudden or chronic onset of respiratory difficulty.

Congenital
The most common congenital hernia involving the diaphragm is a peritoneal-pericardial diaphragmatic hernia. Whenever this defect is suspected, a thorough examination (i.e., physical, radiographic, cardiovascular) for evidence of further midline congenital defects (i.e., umbilical hernia, atrial and ventricular septal defects, cleft palate) should be performed.

Applied Anatomy

The diaphragm projects into the thoracic cavity like a dome; it attaches to the lumbar vertebrae, costal arch, and sternum. Fibers arise on these skeletal parts and radiate towards the tendinous center. The diaphragm is composed of only one layer of muscle and two layers of tendon and therefore is weaker than the multilayered abdominal wall. The central tendon of the diaphragm of the cat is relatively small. In its tendinous portion, transverse fibers course from one side to the other as a reinforcing apparatus.

The muscular part is divided into the pars lumbalis, a pars costalis on each side, and the pars sternalis, all of which with the exception of the lumbar portion, have a uniform thickness of 2-3 mm in cats. The pars lumbalis of the diaphragmatic musculature is formed by the right and left diaphragmatic crura, the right crus being considerably larger than the left. Seen from the abdominal cavity each crus of the diaphragm is a triangular muscular plate whose borders give rise to the tendinous portions. The pars costalis on each side consists of fibers radiating from the costal
wall to the tendinous center. The pars sternalis is an unpaired medial part
unseparated from the bilateral costal portions.

The diaphragm domes far into the thoracic cavity, and its costal part lies on the
medial surface of the last few ribs and costal arch (when tears occur here, the costal
arch can be used in the repair). The stomach and liver attach by ligaments to the
concave peritoneal surface of the diaphragm.

Diagnosis

Diaphragmatic hernia is generally diagnosed via thoracic and abdominal
radiographs. Classic findings on thoracic radiographs is loss of the diaphragmatic line,
air filled visceral structures in the thoracic cavity, loss of lung fields. Abdominal
radiographs may reveal a lack of abdominal viscera. Classic thoracic radiographs of a
patient with a peritoneo-pericardial diaphragmatic hernia shows a large, round
pericardial sac. Occasionally, air filled viscera can be identified in the pericardial sac.
Patients that present with an acute traumatic diaphragmatic hernia (e.g., hit by a car)
may have a massive hernia with abdominal contents replacing most of the patients
respiratory capacity.

Preoperative Considerations

Immediate surgical intervention for the repair of a diaphragmatic hernia is rarely
indicated. Emergency surgery should not be undertaken unless the surgeon and
anesthesiologist are prepared to handle any complications and are confident they can
maintain the animal's essential requirements while the animal is anesthetized.
However, prompt surgical repair is indicated in acutely injured animals with severe
dyspnea, cyanosis, and respiratory distress who demonstrate massive herniation, and
in patients that present with an air filled stomach in the thoracic cavity (these patients
can develop life threatening dyspnea if enough swallowed air enters the stomach).

The most commonly encountered patient with diaphragmatic hernia will fall
between the two categories mentioned above and should be handled in a systematic
manner that will not further compromise the patients already reduced breathing ability.
Surgery is not considered an emergency in mildly symptomatic or asymptomatic
animals with congenital hernias or chronic traumatic hernias of at least several days'
duration. Remember that any stressed, dyspneic cat should be handled very carefully
as further stress can produce catastrophic results.

Anesthesia

Patient stress must be kept to a minimum during the anesthetic induction
phase as any exertion by the animal can be disastrous.

Surgical Approaches

A midline abdominal celiotomy (xiphoid to pubis) is the easiest and most
versatile approach. Positioning the patient's head toward the top of the table and
tilting the table at a 30° to 40° angle will facilitate gravitation of abdominal viscera out
of the thorax. Rarely is it necessary to extend the incision into the thorax via a median
sternotomy however the animal should be prepared in case this becomes necessary.

Surgical Procedure
See the DVD for a detailed video description of this technique. When an extra pair of hands is unavailable for retraction, a Balfour self retaining abdominal retractor is a helpful piece of equipment; large Gelpi retractors work well as abdominal retractors in cats and small dogs. Using the abdominal approach, an incision is made from xiphoid to pubis. Once the peritoneal cavity is opened, the falciform ligament is removed and the Balfour or Gelpi abdominal retractors placed. The diaphragm can now be visualized and the situation evaluated. Some hernias, especially in the area of the dorsal attachments of the crura and the aortic hiatus are not easily visualized; therefore, this area should be carefully inspected even when another laceration is present. The herniated contents are replaced in their proper position and inspected for damage.

Using large sponges or laparotomy pads moistened with warm saline, the liver and bowel are retracted caudally. Visualization of the cranial quadrant of the abdomen can be facilitated by removing the viscera from the abdominal cavity and placing it on a moistened laparotomy sponge. The diaphragmatic tear is now more easily visualized so that a careful examination of the thorax can be done both visually and manually. All thoracic fluid should be aspirated.

In acute traumatic diaphragmatic hernia, the lungs should be expanded to remove atelectasis and to inspect for pulmonary tears and persistent areas of collapse.

In chronic traumatic hernias care is taken not to inflate the lungs. When lung parenchyma is atelectatic for such a long period of time the alveoli collapse. If they are suddenly expanded with air the tight junctions of the normal alveoli are damaged and the infated alveolus fills with fluid. This is referred to as re-expansion pulmonary edema. This is a life threatening disorder and should be avoided.

It is recommended to suture the hernia from dorsal to ventral thus making it much easier to visualize the dorsal structures (vena cava, aorta, esophagus) when suturing. The hernia is closed with a single layer, simple continuous suture pattern using synthetic absorbable suture material (Dexon, Vicryl, Biosyn PDS, Maxon) or monofilament nonabsorbable suture material (Nylon, Prolene, Novafil). Suture size recommended in cats is 3-0. It might be necessary to preplace the most dorsal sutures for better visualization of the tear during suturing. It is also helpful to reconstruct the tear with several simple interrupted sutures to facilitate visualization of the rent. When tears near the caval hiatus are sutured, care is taken to avoid constriction of the vena cava by placing sutures close to the cava. The same principle applies to the aortic and esophageal hiatus.

Air can be evacuated from the chest using several techniques.

1. Prior to tying the last knot of the hernial closure, a carmalt forceps is placed in the hernial rent between two sutures and gently spread open to allow access to the thoracic cavity. The lungs are inflated so as to fill the thoracic cavity. The carmals are removed and the last suture tied to provide an air tight and water tight seal.

2. After hernial rent closure a needle or plastic intravenous catheter is placed through the diaphragm and into the thoracic cavity. Thoracic cavity air is evacuated using a syringe.
3. Needle thoracentesis is performed after the procedure is complete.

4. A 12 - 14 French feeding tube is brought into the peritoneal cavity through a paramedian stab incision in the cranioventral body wall. The tube is passed through the diaphragmatic rent between to sutures just prior to its final closure. Make certain that all fenestrations in the tube are beyond the diaphragm. The diaphragmatic rent closure is then completed around the tube. With the use of a 3-way stop cock and 60 cc syringe, air is evacuated from the thorax until a gentle negative pressure is obtained. The celiotomy incision is closed in a routine fashion. When the celiotomy closure is complete, the tube is again aspirated. The patient should then be placed through a series of positional changes (ventral recumbency, right lateral recumbency, left lateral recumbency, and dorsal recumbency) while attempting to aspirate air. When negative pressure is obtained in all positions, the tube is gently pulled from the chest and abdominal incision.

5. A 12 -14 French diameter thoracostomy tube can be placed at the level of the 9th or 10th intercostal space, tunneled to the level of the 7th or 8th intercostal space and placed through the intercostal muscle and into the thoracic cavity. The patient is then placed through a series of positional changes (ventral recumbency, right lateral recumbency, left lateral recumbency, and dorsal recumbency) while attempting to aspirate air. The tube is removed when the patient has had a negative pressure for 12 - 24 hours.

   All patients are monitored carefully for the next six to eight hours. If signs of respiratory abnormalities arise (dyspnea, tachypnea, etc), the right and left hemithorax should be tapped with a needle and syringe.

**Postoperative Care**

   Postsurgical care includes systemic antibiotics and careful monitoring of the patient’s breathing, temperature, and color. Cats should be kept on a warming device for at least 24 hours. Analgesics may be used to relieve patient discomfort, however care should be taken to monitor the effects of various analgesic drugs on respiratory effort. Thoracic radiographs may be taken to evaluate the chest drain and pleural space.

**Summary**

   Successful repair of a diaphragmatic hernia depends on careful preoperative and postoperative care of the patient. During the surgical repair, the surgeon must work quickly and effectively to complete the procedure as efficiently as possible.
CHEST DRAIN PLACEMENT AND MANAGEMENT
Howard B. Seim III, DVM, DACVS
Colorado State University

If you would like a copy of a video of this surgical procedure go to www.videovet.org or contact videovet@me.com.

Key Points
• The most common types of intrapleural pathology requiring chest drain placement are air and fluid.
• Careful handling of severely dyspnic patients during diagnostic work-up can be life saving.
• Careful attention to providing a sub q tunnel during chest drain placement prevents exit point leak.
• Chest drain removal is based on quantity and character of fluid.

Definition: Pneumothorax is defined as the abnormal accumulation of air in the interpleural space. The most common cause of pneumothorax is trauma.

Pleural effusion is defined as the abnormal accumulation of fluid (i.e., blood, pus, water, chyle, pseudochyle) in the interpleural space. As the accumulation of fluid increases, the mechanical restriction of the lungs to normal ventilation may result in significant hypoxia. Pleural absorption of certain exudates (e.g., pus) may result in systemic toxic effects. Closed chest drainage alone (i.e., as for accumulations of blood, water, chyle, or pseudochyle) or closed chest drainage and lavage (i.e., pus) may be necessary to return the patient's pulmonary status to normal.

Synonyms: Thoracostomy tube, chest tube

Diagnosis
Clinical presentation:
  Signalment: Patients that present with disorders requiring the use of chest drain management may be of any age, sex, or breed.
  History: Historical findings in patients requiring chest drains are acute dyspnea or chronic dyspnea with an acute exacerbation of severe dyspnea. A history of trauma is common in patients that present with severe respiratory difficulty (i.e., pneumothorax).
  Clinical signs: The most frequently reported clinical signs in patients requiring chest drain placement is dyspnea. Signs may be mild, moderate, or severe depending upon the amount of interpleural pathology. Occasionally patients present with a chronic history of mild dyspnea then suddenly have an acute and severe attack requiring urgent care.

Physical examination: Physical examination should be done with care. Over manipulation of severely dyspnic patients can result in enough stress to cause respiratory decompensation resulting in a catastrophic outcome. Minimal manipulation and pre-oxygenation can go a long way in preventing the above scenario.

Laboratory findings: Results of a complete blood count, serum chemistry profile, and urinalysis are dependent upon the patients underlying disorder.
Radiography: Diagnosis of interpleural disease can be established with thoracic radiographs. It is important to remember that the stress of positioning can be enough to cause significant decompensation in a severely dyspnic patient. Pre-oxygenation and careful handling are important prerequisites to ensure patient safety. Evidence of air or fluid density in the interpleural space is diagnostic for pneumothorax or hydrothorax, respectively.

Differential diagnosis: Any fluid or air accumulation in the interpleural space causing severe dyspnea is a potential candidate for chest drain placement and management.

Medical management:
Pre-examination management: Patients can have a significant amount of pulmonary atelectasis and ventilatory compromise, yet appear relatively normal. However, the least amount of stress (i.e., physical examination, chest radiographs, ultrasound) can rapidly push them into a respiratory decompensation. It is important to handle dyspneic animals with minimal stress. Generally, a needle thoracentesis can be performed to relieve the immediate respiratory distress. See the DVD for a detailed video description of performing a needle thoracentesis. Supplemental oxygen should be provided with a mask during the thoracentesis procedure. Once the majority of fluid has been removed and the patient is relieved of its life-threatening dyspnea further workup can commence and, if indicated, an indwelling chest drain can be inserted.

Surgical treatment: The objective of chest drain placement is to provide an avenue for removal of contents (i.e., air, fluid) that have accumulated in the interpleural space.

Preoperative management: Supplemental oxygen should be provided with a mask, and needle thoracentesis performed to remove as much air or fluid as possible. Once the majority of air or fluid has been removed and the patient is relieved of its life-threatening dyspnea, anesthetic management can be considered.

Anesthesia: Chest drain placement is an easy and quick procedure; local or general anesthesia may be considered. General anesthesia offers a more controlled situation; intubation and gentle positive pressure ventilation are then available to expand constricted or atelectatic lung lobes.

Surgical anatomy: The lung surfaces are covered with visceral pleura and the thoracic walls with parietal pleura. The parietal and visceral surfaces are held in intimate contact by negative pressure coaptation and fluid exchange.

Positioning: Patients are positioned in lateral recumbancy with the most affected hemithorax uppermost.

Surgical technique:
Chest tube placement using a percutaneous chest tube placement device (Mila): The site of chest tube placement is clipped, aseptically prepared and a local anesthetic placed. An over the needle plastic catheter is placed at the 7th or 8th intercostal space. Once the needle and catheter have entered the pleural space the needle is removed thus leaving the plastic catheter in place. A quide wire is then passed through the plastic catheter and into the pleural space. Make certain the wire is passed at least 8-12 inches in the pleural space.
Once the wire has been adequately advanced remove the plastic catheter being careful to maintain placement of the quide wire. Now grasp the exposed end of the quide wire and pass it into the tip of the chest tube. Advance the chest tube along the quide wire until it passes into the pleural space. Continue to advance the chest tube to its marked hub. Now remove the guide wire from the chest tube and attach a syringe to the chest tube to begin evacuating air or fluid. The chest tube is secured to the chest to prevent premature removal.

**Chest tube placement using a feeding tube or trocar thoracostomy tube:**

The surgical site is clipped and aseptically prepared. A stab incision is made over the 9th or 10th intercostal space. A subcutaneous tunnel directed cranioventral is made over two intercostal spaces with the aid of a curved Mosquito hemostat. Once an adequate tunnel has been developed the hemostat is removed and is clamped at the tip of the feeding tube. If a trocar thoracostomy tube is being used, the long metal trocar is placed in the lumen of the drain so the tip just protrudes from the end of the tube. The hemostat (feeding tube) or trocar (trocar thoracostomy tube) is used to place the drain in the tunnel previously dissected. The hemostat or stylet and drain are then carefully punctured through the intercostal muscles of the 7th intercostal space and into the thoracic cavity. The intercostal vessels located on the caudal aspect of the rib are avoided. If a hemostatic forceps is used, once the drain enters the thoracic cavity, the hemostat is opened and the drain advanced into the pleural space before removing the hemostat. The drain is gently advanced in a cranioventral direction until about 4 to 6 inches are within the pleural cavity. The tube will usually make a gentle curve and rest on the ventral thoracic floor. Generally, sterile, disposable rubber feeding tubes of size #14 – 16 French (cats) or 18 – 20 French (dogs) can be used. These tubes have a large enough inside diameter to preclude obstruction with exudate, yet soft enough to be nonirritating to the pleural surfaces. All chest tube fenestrations must be within the chest cavity to prevent iatrogenic pneumothorax or leakage of pleural contents into the subcutaneous space. Several simple interrupted sutures are placed to close the skin snugly around the tube. A Chinese finger-trap friction suture is used to secure the tube to the patient. A deep simple interrupted suture is placed in the skin and around the subcutaneous portion of the chest drain to incorporate lattisamus dorsi muscle, and exited on the opposite side of the tube. This suture helps ensure a air tight and water tight seal around the exit point of the chest drain.

**Suture material/special instruments:** #14 to 20 French silastic or red rubber feeding tubes, pointed stylet to fit in tube lumen

**Postoperative care and assessment:** An antibiotic ointment is applied to the skin incision and a comfortable bandage applied around the chest to secure and cover the tubing. Initially, all fluid or air is withdrawn from the chest. Assisted respiration is then administered to expand the lungs. In cases with constrictive pleuritis, full expansion cannot be achieved initially. After all fluid is aspirated, radiographs are taken to confirm proper drain placement and possibly provide further evidence of etiology (i.e. congestive heart failure, foreign bodies, neoplasia). Kinks in the tubing may be seen on radiographs and can be corrected by gently maneuvering the tube in and out of the chest. When not in use, the tubing should be double-clamped and a sterile 3 cc syringe taped to the end. In addition, all tube connections should be reinforced using orthopedic wire to engage all parts that could potentially be pulled apart by animal or man. Here is an illustration of how the author secures all tube connections. All drain tubing is then covered with the bandage to insure a closed system.
Tube removal: Tube removal is dependent upon the change in volume and character (i.e., cytology) of the fluid over a period of time. Generally, as the fluid volume decreases and the cytology is consistent with the irritation caused by the presence of the drain, tube removal can commence. A gauze sponge with an oil base ointment is placed over the exit wound, and the tube gently pulled. The gauze sponge remains on the wound to help seal against possible leaks. A bandage is placed to hold the gauze sponge in place. The bandage can be removed in 3 to 4 days.

Prognosis: The short term prognosis for patients that require chest drain placement is generally favorable. If the preoperative management is performed as directed above, drain placement should be successful and will aid in determining the ultimate cause of the pleural pathology. The long term prognosis is dependent upon the underlying disorder.
Split-shot Wound Management

Key Points

• Skin has the ability to stretch when placed under mild tension
• Normal wound contraction often stops before wound edges appose.
• Split-shot wound management can be used to encourage skin edges to contract.

Indications: Use of various appliances to create tension on the local skin of non-contracting open wounds is not new. Subcutaneously buried silastic balloons (i.e., skin expanders) injected every 24 hours with varying amounts of saline will stretch local skin and have been used extensively in human plastic and reconstructive surgery. Skin expanders have also been described for use in veterinary patients. Skin expansion may be indicated in wounds that have undergone normal wound contraction without successful wound margin apposition. The most common locations for inappropriate wound contraction in small animals are extremities, head, and tail.

Applied Anatomy: Skin is made up of several layers that collectively form a complex organ system. Skin is not capable of regeneration. One method of getting ‘more’ skin for wound coverage is encouraging local skin to undergo intussusceptive growth. This can be accomplished by applying tension to local skin around the wound. If tension is constant, skin layers will accommodate the increase tension by becoming thinner thus allowing the skin to ‘stretch’.

Anesthesia: Patients undergoing split-shot wound management should be placed under general anesthesia.

Technique: Positioning: Patients are positioned with the wounded area uppermost.

Patient preparation: Wounds identified for split-shot wound management should be treated as an open wound until there is evidence of a healthy granulation tissue bed. Routine aseptic preparation of the local skin is performed.

Special instruments and suture: Metallic split-shot (i.e., other than lead) can be purchased at any local sporting goods or fishing store. Split-shots are placed in a cold sterilization media for an appropriate time period and thoroughly rinsed prior to use. Monofilament non-absorbable suture with a swaged-on taper needle, size 00 to #1 depending upon location and size of wound is recommended. A sterile rubber bumper is fashioned from a feeding tube or catheter.

If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to www.ivseminars.com and click VideoVet or contact videovet@me.com.
**Split-shot technique:** The wound and surrounding skin are prepared for aseptic surgery. Two bumpers are created by cutting one 1/2 inch piece off the flanged end of a 20 French feeding tube or catheter. This segment of tube is then split in two.

An appropriate size monofilament nonabsorbable suture is selected. The skin edges are gently undermined being careful not to trim the wound edge. The swaged-on needle is placed through the rubber bumper and enters the wound at the commissure. The wound edges are then sutured using a simple continuous pattern. Care is taken to engage the needle in the tough collagen laden subcutaneous tissue. Patients with thin subcutaneous tissue (i.e., cats, small dogs, areas of thin skin) may require penetration of skin instead of subcutaneous tissue. Once the entire length of the wound has been sutured, the suture is passed out through the skin of the remaining commissure of the wound. Knots are not tied in either end of the suture.

Gentle traction is placed on the exiting ends of the suture until mild tension is placed on the wound edges and local skin. A split-shot is placed on each end of the exiting suture against the bumper. The split-shot is then gently but firmly clamped against the suture; this maintains tension on the skin edges and local skin. The wound is bandaged, an Elizabethan collar placed, and the patient confined to a cage. Each day the bandage is removed, the ends of the suture gently pulled and a split-shot is placed between the bumper and the original split-shot. Daily tension is performed without the need for general anesthesia or sedation. Skin may be responsive to tension for 7 to 10 days. When the wound is closed to your satisfaction, the suture and bumpers are removed. The remaining wound is bandaged only if it requires further protection.

**Tie-over Bandage Technique**
If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to [www.ivseminars.com](http://www.ivseminars.com) and click VideoVet or contact videovet@me.com.

**Key Points**
- The most important aspects of wound management are debridement, debridement, debridement.
- The solution to pollution is dilution.
- A tie-over bandage can cover the most difficult to bandage wounds.
- A tie-over bandage can help ‘stretch’ local skin.

If you would like an instructive DVD of this topic, go to [www.ivseminars.net](http://www.ivseminars.net) and click on Video Vet.

**WOUND MANAGEMENT:** The area should be clipped and cleaned as soon as possible to provide a clean environment beneath the bandages that will eventually be applied. Sterile, water soluble gel placed on the wound is a convenient means of temporary wound protection. Dried blood and debris should be removed from the surrounding skin with antiseptic soap, using care to avoid contact between the soap and exposed tissues which can result in lipolysis and tissue damage. The primary goal of wound management is to decrease bacterial numbers and debris and enhance the animal's defense mechanisms (i.e., debridement). Gross particulate matter, hair, etc. should be
removed manually from the wound. Lavage is beneficial in further decreasing infection-promoting debris and bacteria. Saline is indisputably the ideal lavage solution, although dilute chlorhexidine (0.05 to 0.005%), or povidone-iodine (0.01%) may be used. The effectiveness of lavage is dependent upon volume and pressure. Studies have shown that high pressure (25-60 psi) is superior to low pressure (0.5-5.0 psi) when wounds are only lavaged one time. Medium pressure, which has also been shown to be beneficial can be generated using an 18 gauge needle and large syringe (35-60 ml). Surgical debridement of necrotic-appearing tissue and embedded foreign material limits nutrients for bacterial growth and enhances the animal's local defense mechanisms.

OPEN WOUND MANAGEMENT: Open wound management allows optimal drainage and daily inspection, debridement and lavage of tissues. Following surgical excision of necrotic tissue, etc., continued mechanical debridement can be performed using an adherent dressing (wet-to-dry, dry-to-dry, or wet-to-wet). Wide-mesh gauze sponges are ideal for adherent bandages. The type of dressing used depends on wound conditions. Wet-to-dry dressings can be used for wounds with necrotic tissue, foreign matter and viscous exudate. The wet dressing dilutes the exudate and allows absorption. As the dressing dries, necrotic tissues adhere to the gauze and are removed with the bandage. Dry-to-dry dressings have similar indications as wet-to-dry except without the presence of viscous exudate. Wet-to-wet dressings are indicated when viscous exudate is present without necrotic tissues. The contact adherent layer should be covered by an absorbent outer layer. Once necrotic tissues have been removed and granulation tissue begins to form, adherent gauze should be replaced with nonadherent pads (telfa).

SECOND INTENTION HEALING: Second intention healing occurs by formation of granulation tissue, wound contraction and epithelialization. The advantages of this process are drainage remains optimal, wound infections are rare and the time and expense of surgery is avoided. However, second intention healing may cause disfigurement or loss of function due to wound contracture, and the epithelium formed may be easily disrupted.

TIE-OVER BANDAGE: Indications: Large surface area wounds (i.e., abdomen, thorax, back, neck) or wounds in 'difficult-to-bandage' areas (i.e., tail, perineum, head, paraprepuclial,proximal extremeties) may not be amenable to routine bandaging techniques. These areas generally lend themselves nicely to placement of a tie-over-bandage.

Technique: The wound bed is prepared as described above. Several # 0 or #1 monofilament non-absorbable suture loops are placed in the skin on the periphery of the wound. Loop sutures are generally placed 360o around the wound and spaced 2 or 3 cm apart. Appropriate wound covering materials are placed in the wound bed (i.e., wet to dry, gauze, telfa, etc) and a sterile laparotomy pad placed on top to provide protection to the wound. Several lengths of 1/4 inch or 1/2 inch umbilical tape are passed through the loops of suture, over the laparotomy pad and through the suture loop on the opposite side of the wound. The umbilical tape passes over the wound multiple times to
hold the laparotomy pad in place (and therefore the wound covering materials). Enough traction is placed on the suture loops to place mild tension on the skin edges of the healing wound. This bandage is easily removed and replaced for ease of bandage change.

Once the granulation bed is healthy and the wound is considered surgically clean it can be closed primarily (i.e., delayed primary closure). If there are small defects at the time of suture removal these can generally heal by second intention.
ANAL SACCULECTOMY; A NOVEL APPROACH  
Howard B. Seim III, DVM, DACVS  
Colorado State University  

Key Points  
• knowledge of anorectal anatomy and neuroanatomy is important to protect vital structures  
• remove all anal sac epithelium during anal sacculectomy  
• use a Mila Anal Sac catheter or 6F Foley 3cc bulb catheter to facilitate anal sacculectomy  

If you would like a video of this surgical procedure on DVD go to www.videovet.org or contact videovet@me.com. You may click on the ‘Seminar Price’ for any DVD you would like to purchase.  

Introduction: Disorders involving the anus and rectum occur frequently in small animal practice. In order to appropriately diagnose and treat these disorders, knowledge of the regional anatomy, physiology, common clinical signs they produce, and proper physical examination techniques are necessary.  

Anatomy: The location and function of the following anatomic structures should be reviewed prior to surgical management of diseases of the anus and rectum: internal and external anal sphincter muscle, anal sac and duct and caudal rectal nerve.  

The Anal Sphincter Muscle: (From the introduction of a report on hemorrhoidectomy written by WC Bornemeier and published in Am J of Proc, Feb, 1960):  

"The prime objective of a hemorrhoidectomy is to remove the offending varicosity with as little damage as possible to the patient. Of all the structures in the area, one stands out as the king. You can damage, deform, ruin, remove, abuse, amputate, maim, or mutilate every structure in and around the anus except one. That structure is the sphincter ani. There is not a muscle or structure in the body that has a more keenly developed sense of alertness and ability to accommodate itself to varying situations. It is like the goalie in hockey...always alert."  

"They say man has succeeded where the animals fail because of the clever use of his hands yet, when compared to the hands, the sphincter ani is far superior. If you place into your cupped hands a mixture of fluid, solid, and gas and then, through an opening at the bottom, try to let only the gas escape, you will fail. Yet the sphincter ani can do it. The sphincter apparently can differentiate between solid, fluid, and gas. It apparently can tell whether its owner is alone or with someone, whether standing up or sitting down, whether its owner has his pants on or off. No other muscle in the body is such a protector of the dignity of man, yet so ready to come to his relief. A muscle like this is worth protecting."  

Clinical Signs: Common clinical signs associated anal sacculitis include: anal licking, matting of anal hair, anal discharge and scooting. Patients that present with any of the above clinical signs should have a thorough physical examination with emphasis on the anorectal region, including a digital rectal examination.
Physical Examination: A complete physical examination should be performed in all patients with clinical signs specific for anorectal disease in order to rule out systemic disorders that manifest themselves with anorectal abnormalities (i.e., pemphigus).

Specific examination of the anorectal region should include close visual examination of the perineum, circumanal area, and base of the tail, as well as careful digital rectal palpation. In many instances this may be all that is necessary to obtain a definitive diagnosis.

Sphincter muscle atonia or areflexia: This form of incontinence occurs when the peripheral nervous supply to the external anal sphincter muscle or the muscle itself has been partially or totally severed. The external anal sphincter muscle is made up of striated muscle fibers and is partially responsible for the voluntary control of defecation.

Isolated injury of the caudal rectal nerve to the external anal sphincter is uncommon but may occur from iatrogenic causes. Injury can occur during overzealous anal sacculectomy. The caudal rectal nerve originates from the pudendal nerve which lies on the internal obturator muscle deep in the pelvic canal. Injury during anal sacculectomy is therefore caused during the final ‘deep’ dissection of the anal sac rather than during the initial dissection. Thus deep dissection is when the surgeon must use exceptional care during dissection of the anal sac.

Anal Sacculitis: Anal sac impaction and abscessation is the most common anorectal disorder diagnosed by the small animal practitioner. Diagnosis is confirmed by clinical signs, visual and digital rectal examination. Relief of impaction by digitally expressing the anal sacs is easily performed during rectal examination. If an anal sac abscess is present, infusion of an antibiotic preparation may be sufficient to eliminate the infection. Systemic antimicrobial treatment may be required in resistant cases. If the anal sac abscess becomes a chronic recurrent problem, surgical excision of both anal sacs is the treatment of choice. Surgery should be delayed however until the immediate infection or abscess has been controlled medically as described above.

Surgical Techniques for Anal Sac Removal: There are a variety of techniques currently used to successfully remove anal sacs. The best approach would be one that allows constant palpation of the extent of the anal sac and also allow retraction of the anal sac during dissection. One such technique is described below:

MILA Anal Sac Catheter Technique (the authors’ preferred technique) or 6 French Foley Catheter with a 3cc bulb Technique:

A novel approach for safely and completely removing anal sacs relies on the use of a 6 French balloon catheter with a 3cc bulb (MILA or Foley). The balloon catheter is placed into the anal sac through the anal sac orifice and its cuff inflated. Once introduced into the sac, the catheter bulb is inflated with 2-3 cc of air or saline. The bulb distends the anal sac making identification and palpation of the gland simple. The protruding catheter allows the surgeon, or the surgeon’s assistant, to place gentle traction on the gland during dissection. A 360-degree skin incision is made around anal sac duct and the protruding catheter. Care is taken to leave at least 2mm of...
skin from the anal sac duct and the incision. Metzenbaum scissors (curved) are then used to dissect to the plane of tissue between the anal sac wall and external anal sphincter. Identification of the anal sac wall is made by identifying its grayish color in comparison to the deep red color of external anal sphincter muscle fibers that will be carefully dissected off of the anal sac wall. As the dissection progresses constant traction is placed on the Foley catheter to accentuate to sac. When performing the deep dissection of the sac wall care is taken to make certain the dissection does not go deep to the sac wall. This is the location of the caudal rectal nerve fibers. Dissection is continued until the sac is completely dissected free and removed from its surrounding tissue.

Closure consists of suturing together any cut fibers of the external anal sphincter muscle with 3-0 Maxon and the skin closed with 4-0 Biosyn using an intradermal technique. This is the authors preferred technique for anal sacculectomy.

This technique is illustrated on the Anal Sacculectomy video located in the GI Surgery I DVD. Check it out at www.videovet.org.

An alternate technique includes using a pair of Metzenbaum scissors to cut into the anal sac through the duct. The sac is opened to expose the glistening greyish colored interior lining. Hemostats are used to grasp the full thickness of the anal sac wall, being careful to avoid the external anal sphincter muscle fibers. A number 15 BP scalpel blade is used to carefully scrape the gland from the underlying external anal sphincter muscle. The external anal sphincter m., subcutaneous tissue and skin are closed with a synthetic absorbable suture material in a simple interrupted pattern.

An alternate method is to incise laterally over the anal sac, dissect through the subcutaneous tissue, locate the sac and excise it toward the duct.

Regardless of the procedure used, if the entire anal sac is removed and the caudal rectal nerve avoided the prognosis is excellent.
Salivary Mucocele
Howard B. Seim III, DVM, DACVS
Colorado State University

If you would like a copy of the video of this surgical procedure on DVD go to www.videovet.org.

Key Points
- There are 8 salivary glands in the dog and cat.
- The sublingual salivary gland is generally responsible for mucocele formation.
- Removal of the sublingual and mandibular salivary glands are necessary because of their close association.
- Accumulated saliva should be drained.

INTRODUCTION
Salivary glands can be affected by inflammation, trauma, calculus formation and neoplasia; resulting in abscessation, rupture of the duct or gland and formation of a salivary mucocele, or obstruction and atrophy. The mode of therapy is generally dictated by the type of lesion present (abscess, mucocele, neoplasia).

There are four paired salivary glands present in the dog and cat: parotid, mandibular, sublingual and zygomatic glands. The glands most commonly injured or involved in pathological processes (calculi, neoplasia, trauma) are the mandibular and sublingual salivary glands. The mandibular salivary gland is a mixed gland (serous and mucous secretion) located in the junction of the maxillary (internal maxillary) vein and lingual facial (external maxillary) vein as they form the jugular vein. It is adherent to the darker, monostomatic portion of the sublingual gland, and shares a common heavy fibrous capsule with that gland.

The mandibular duct leaves the medial portion of the gland near the sublingual gland, and runs craniomedially, medial to the caudal sublingual gland, between the masseter muscle and mandible laterally and the digastricus muscle medially to empty in the sublingual papilla lateral to the cranial frenulum of the tongue.

The sublingual duct originates at the caudal portion of the gland and joins the mandibular duct. The secretion of the separate lobes of the monostomatic portion of the sublingual gland drain through four to six short excretory ducts into the sublingual duct. The polystomatic portion of the sublingual gland lies under the mucosa of the tongue and secretes directly into the oral cavity rather than through the main sublingual duct.

Diseases of the parotid and zygomatic salivary gland occur infrequently in the dog and cat.

PATHOLOGICAL PROCESSES
Inflammation-Infection
Infection can occur in any of the salivary glands, but is a rare occurrence in the dog and cat. Sialoliths are rare in dogs and cats but may occur in patients with salivary mucocele. Included in the differential diagnosis of intermandibulare mass is intermandibular abscess. Unlike salivary mucocele, intermandibular abscess is painful in palpation, causes fever, and aspiration of the mass generally reveals puss.
Sialoadenitis has been reported in the zygomatic salivary gland. Patients present with similar signs as retrobulbar abscess including pain on palpation of the mouth, reluctance to open or close the mouth, periorbital cellulitis without exophthalmos and epiphora, a fistula below the eye and/or a swelling or drainage into the mouth at the opening of the zygomatic duct. Treatment is similar to retrobulbar abscess including oral drainage near the last molar. Generally, a red inflamed area is visible. A mosquito hemostat can be used to provide ventral drainage by inserting it into the inflamed area and opening the abscess or inflamed gland. Systemic antibiotics are given for five to seven days.

SALIVARY MUCOCELE

Salivary mucocele formation is the most common disorder of the salivary gland in the dog and cat. The mucocele is formed from secretion of saliva from a defect in the gland or duct system. The most commonly affected glands are the mandibular and sublingual salivary glands, with the sublingual gland being the most frequent source of saliva. The lining of the mucocele consists of inflammatory tissue surrounded by granulation tissue. There is no evidence of a secretory lining present in the mucocele and therefore cannot be considered a true "cyst".

There are three major classifications of salivary mucoceles depending upon their location: cervical mucocele, ranula and pharyngeal mucocele.

Cervical mucoceles are generally located on the lateral aspect of the head and neck from the level of the mandibular and sublingual salivary glands to the intermandibular space. The majority of patients present with mucoceles in the intermandibular region.

Ranulas are formed from an accumulation of saliva along the base of the tongue.

Pharyngeal mucoceles appear as a fluctuant, smooth, domed-shaped swelling in the lateral pharyngeal wall. These occur less frequently than cervical mucoceles or ranulas.

The etiology of salivary mucoceles (cervical mucocele, pharyngeal mucocele and ranula) is generally unknown, but things such as trauma, inflammation, sialoliths, foreign bodies, and iatrogenic damage during surgery have been implicated. It is generally felt that mucoceles result from damage to the duct or gland tissue with leakage of saliva into the tissues. The monostomatic (cervical mucocele) and polystomatic (pharyngeal mucocele and ranula) portions of the sublingual salivary gland are felt to be the most commonly involved. Poodles and German shepherds are reportedly the most common breeds affected, but many breeds have been reported to have developed salivary mucoceles.

Cervical Mucocele

Diagnosis is based on history, physical examination, palpation and aspiration of a blood tinged saliva. Differential diagnosis includes cervical abscess, neoplasia, enlarged mandibular lymph nodes and draining tract secondary to foreign body migration. Sialography may be attempted, but is time consuming and difficult with questionable results in many cases.
The treatment of choice for cervical mucocele is removal of the mandibular and sublingual salivary glands and ducts on the affected side and ventral drainage of the accumulated saliva. Quite often, the patient with a cervical mucocele will present with a midline, intermandibular cervical mass making lateralization difficult. Determination of the glands involved, right side vs. left side, can be accomplished by the following methods:

1. Careful historical evaluation may reveal the side that was initially involved.
2. Careful oral examination for the presence of a ranula or pharyngeal mucocele.
3. Gently force the saliva into the intermandibular space and see if the saliva tends to move toward the right or left side of the head and neck.
4. After the patient has been anesthetized and clipped for surgery and placed in dorsal recumbency, manipulate the salivary mucocele to determine the affected side. The saliva will tend to accumulate toward the affected glands.
5. With the patient anesthetized and in dorsal recumbency, inject contrast into the mucocele, massage gently and take a ventrodorsal x-ray. The contrast may be seen more toward one side or the other indicating the affected glands.

If the above techniques fail and you still have doubts about lateralization, you can usually operate on the suspected side and if no communication with the mucocele is found, operate the other side. Bilateral mandibular and sublingual salivary gland resection is generally faster than the time necessary to perform sialography and does not put the patient at risk for losing salivary function.

**Surgical technique:**

*See the DVD for a detailed video description of the surgical technique.*

Removal of the mandibular and sublingual salivary glands is performed by first positioning the dog in lateral recumbency with the affected side uppermost. The neck and jaw should be positioned slightly obliquely and towels or sand bags placed under the neck to elevate the surgical site for better visualization of the bifurcation of the jugular vein.

The incision is made from the ramus of the mandible cranially to the bifurcation of the jugular vein caudally; occlusion of the jugular vein prior to incision will facilitate visualization of landmarks. Dissection is carried into the capsule of the mandibular and sublingual salivary glands. An intracapsular dissection commences and the glands are removed from the capsule. The ducts of the mandibular and sublingual salivary glands are followed craniomedially to the mandible. If you are on the correct side, you should encounter saliva from the mucocele oozing into the incision. In some cases a dilation of the duct can be visualized. Aspiration of the dilated duct will produce saliva of similar color and consistency as that aspirated during diagnosis.

The ducts are followed as far cranially as possible and ligated or stripped out to complete the resection. An incision is made at the most dependant point of the cervical mucocele (when the animal is standing!) and a penrose drain is placed to facilitate postoperative drainage of saliva. Platisma muscle, subcutaneous tissues and skin are closed in a routine fashion.

The drain is removed two to three days postoperatively. If the salivary glandular tissue has an unusual appearance at the time of resection, it should be submitted for histopathologic evaluation.
Complications: Complications associated with salivary gland resection are few, but may include:
1) Lymph node removal instead of the salivary gland
2) Operating the wrong side
3) Infection - if a patient has an infected mucocele prior to surgery, do not operate until the infection has resolved.
4) Recurrence - wrong side operated
5) Recurrence - incomplete removal

Re-exploration of the previous surgery should be done to accurately determine the cause and most appropriate therapy.

Ranula
A ranula is a thin walled linear swelling that results from ruptured sublingual or mandibular salivary ducts below the oral mucosa next to the tongue or rupture of the polystomatic portion of the sublingual gland. Ranulas have been reported in cats. Diagnosis is based on history, oral examination, palpation and aspiration of the mass. Blood tinged saliva on aspiration is diagnostic.

Surgical technique:
See the DVD for a detailed video description of the surgical technique.
Marsupialization or excision as for pharyngeal mucocele are the treatments of choice for ranulas. For marsupialization incise the ranula with a scalpel blade. Next, empty the contents of the ranula. Finally, suture the mucous membrane of the ranula to the oral mucosa. Use a multifilament synthetic absorbable suture of 3-0 size. An alternate technique is to incise the ranula as described above but resect it instead of marsupialize it.

In cases of ranula recurrence, or if the ranula is associated with a cervical mucocele, the mandibular and sublingual salivary glands on the affected side should be removed along with excision or marsupialization of the ranula.

Pharyngeal Mucocele
Patients with pharyngeal mucocele may present with signs related to upper airway obstruction, since the swelling eventually becomes large enough to occlude the laryngeal orifice. Affected patients may have a history of noisy respiration progressing to intermittent dyspnea, cyanosis, and syncope in severe cases.

A presumptive diagnosis can be made by careful oral examination. The pharyngeal mucocele appears as a fluctuant, smooth, dome-shaped swelling in the lateral pharyngeal wall. Aspiration of a blood-tinged saliva is diagnostic, and is generally performed when the patient is under anesthesia to avoid unnecessary stress.

Pharyngeal mucoceles can be treated several ways. Initial treatment consists of opening and draining the mucocele. The pharyngeal wall is allowed to heal by second intention. This procedure generally gives rewarding results initially but recurrence is common. In cases of pharyngeal mucocele reoccurrence, ipsilateral mandibular and sublingual salivary gland resection should be performed as described for cervical mucocele.
Prognosis

The prognosis for patients with cervical mucocele, ranula or pharyngeal mucocele is favorable if the surgeon can identify the involved side and successfully remove all mandibular and sublingual salivary tissue.
INTRODUCTION TO SMALL ANIMAL ACUPUNCTURE

Mitsie Vargas DVM, MSTCVM, CTCVMP, CVBMA, CVMMP, CCRV, FAAVA

www.chiu.edu
Outline

• Introduction
  • What is Acupuncture
  • What is TCVM

• Clinical Application
What is Acupuncture?

• Part of Ancient form of Chinese Medicine

• Evolved over the past 3000 years

• Compatible with current concepts of medicine

• Promote health and prevent disease
History of Veterinary Acupuncture

- **Bo Le**
  - Father of veterinary acupuncture
  - Equine expert
  - 659-621 BC

- **Bo Le Zhen Jing**
  - Boles’s Canon of Veterinary Acupuncture
  - One of the first veterinary books
Ancient needles were found from the Neolithic period (8,000 years ago) • These were made from stone.

During the Shang Dynasty (1600-1100 BC) the stone and bamboo needles were replaced with bronze needles.

Acupuncture = Acus means “Needle” and Pungare means “To Pierce”
Otzi the Iceman Mummy

Europe’s most famous mummy was discovered two miles high in the Ötztal Alps bordering Austria and Italy.

Naturally preserved by more than 5,000 years

Ötzi is carefully tended to by researchers at the South Tyrol Museum of Archaeology in Bolzano, Italy.

Has tattoos in distinctive acupuncture meridians and specific points that would have been able to treat his symptoms!
WHAT IS TCVM?

• TCVM = Traditional Chinese Veterinary Medicine
• Has been used to treat animals for thousands of years in China
• Includes 5 Branches
  • Acupuncture
  • Herbal Medicine
  • Food Therapy
  • Tui-Na
• Exercise or life style
  • TCM = Traditional Chinese Medicine (Human Equivalent) – including Qi Gong or Tai Chi
TCVM – The Basics

• **Recognizes disease**
  • or “dis – ease” as an imbalance in the body

• **Views the body as an INTEGRATED and ENERGETIC structure**
  • Thus, disturbance of energy flow creates or results in dis-ease of the WHOLE organism.

• **Yin – Yang theory and the 5 Element Systems**
  • make up the conceptual foundation for TCVM
TCVM – Yin and Yang

• Duality in the universe

• All conceivable concepts and entities
  • composed of 2 opposing yet complimentary aspects called Yin and Yang

• Comparison b/w opposites
  • that creates meaning for each side

• Opposite sides
  • of the same coin
YIN

- All phenomena associated with
  - Cold
  - Darkness
  - Cloudiness
  - Passivity
  - Downward movement
  - Interior
  - Weakness

- Feminine/Nurturing
- Parasympathetic nervous system

YANG

- All phenomena associated with
  - Heat
  - Brightness
  - Sunshine
  - Activity
  - Upward and outward movement
  - Exterior
  - Strength

- Masculine/Action-oriented
- Sympathetic nervous system
TCVM- Yin Yang Theory Summary

- **Everything in the Universe**
  - has 2 opposite aspects- Yin and Yang

- **Any Yin Yang division**
  - can be further divided into Yin Yang aspects

- **Yin and Yang control each other**

- **Yin and Yang mutually create each other**

- **Yin and Yang may transform into each other**
  - in certain circumstances
Balanced vs. Unbalanced State

When Yin and Yang are balanced, the animal is healthy.

When we have more yang than Yin, we could have a fever, or we could be hyperactive.
Yin/Yang Quiz

- Outgoing, high energy, hyperactive animal?
  - Yin or Yang?
Yin/Yang Quiz

- Quiet, reserved, withdrawn animal?
  - Yin or Yang?
How does it work??????

- Stimulates local nerve reflexes
- Affects local structures near acupoint
- Affects internal organs near acupoint
- Stimulates brain
- Creates generalized effects
- Changes hormones and chemical throughout the body
- Acupuncture Releases β-endorphin, reduces muscle spasms, reduces inflammation and promotes tissue healing
- Gate theory of pain
- Opioid system stimulation
- Fascia stimulation
Clinical Indications of Veterinary Acupuncture
Is it Scientific and evidence based?

• Research has increased exponentially Especially in the past 15 years
  25,995 acupuncture references (April, 2017) 406 veterinary references
• Support the effectiveness of acupuncture For many different disorders In many different species
Pain Management and Musculoskeletal Disorders

- Lameness and neck and back pain
  - Arthritis
  - Tendon/ligament disorders
  - Intervertebral disk disease
  - Muscle injury

- Acupuncture effects
  - Releases β-endorphin etc.
  - Reduces muscle spasms
  - Reduces inflammation
  - Promotes tissue healing
Veterinary Conditions Highly Responsive to Acupuncture

- **Pain management and Musculoskeletal Disorders**
  - Arthritis, osteoarthritis
  - Post-operative
  - Soft tissue injury

- **Neurological disorders**
  - IVDD
  - Seizure

- **Promotion of Quality of Life**
  - Geriatric patients
  - Cancer patients

- **Immune-mediated diseases**
  - Atopy
Neurological Disorders

• Common disorders treated
  – Intervertebral disc disease (IVDD)
  – Seizures
  – Paresis/paralysis
  – Facial paralysis
  – Nerve injuries
  – Many others

• Acupuncture effects
  – Controls seizures
  – Reduces inflammation
  – Promotes nerve regeneration
IVDD: Acupuncture vs Western Medicine

- 50 dogs with IVDD randomly divided into 2 groups:
  - Group 2: Medical treatment only

- The time (mean ± SD) to recover ambulation in dogs with grade 3 and 4 dysfunction in was significantly less than in Group 2 (20.83 ± 11.99 days).

- The success rate (ability to walk without assistance) for dogs with grade 3 and 4 dysfunction in Group 1 (10/10 dogs) was significantly higher than that of similarly affected dogs in Group 2 (6/9 dogs).

IVDD: Acupuncture vs Western Medicine

- The overall success rate (all dysfunction grades) for group one was significantly higher (23/26; 88.5%) than for group two (14/24; 58.3%).

- Conclusion
  - Electro-acupuncture + conventional medical treatment resulted in a shorter time to recover ambulation and deep pain perception, than did the use of conventional treatment alone in dogs with thoracolumbar intervertebral disk disease.

IVDD: Acupuncture vs Surgery

40 dogs with severe neurologic signs to thoracolumbar IVDD were divided into 3 treatment groups:

- Decompressive surgery (DSX)
- Electroacupuncture (EAP)
- DSX followed by EAP (DSX + EAP).

<table>
<thead>
<tr>
<th>Group</th>
<th>DSX (n=10)</th>
<th>EAP (n=19)</th>
<th>DSX + EAP (n=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improvement after Treatment</td>
<td>4 (40%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>15 (78.9%)&lt;sup&gt;b,A&lt;/sup&gt;</td>
<td>8 (72.7%)&lt;sup&gt;b,A&lt;/sup&gt;</td>
</tr>
<tr>
<td>Unchanged after Treatment</td>
<td>6 (60%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4 (21.1%)&lt;sup&gt;b,B&lt;/sup&gt;</td>
<td>3 (27.3%)&lt;sup&gt;b,B&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a,b</sup> Within a row differ significantly (P<0.05)

<sup>A, B</sup> Within a column differ significantly (P<0.05)

IVDD: Acupuncture vs Surgery

Conclusion

Electroacupuncture (EAP) was more effective than Decompressive surgery (DSX) for recovery of ambulation and improvement in neurologic deficits in dogs with long-standing severe deficits attributable to thoracolumbar IVDD.

Promotion of Quality of Life
Cancer patients

- 8 yo MN Australian Shepherd
- Presented 8/2015 post-op metastatic anal sac adenocarcinoma
- Acupuncture Q 2-3 weeks, herbal (Stasis Breaker, Wei Qi Booster, Co-Curcumin) and diet therapy
- 19 months remission with excellent quality of life
Promotion of Quality of Life
Geriatric patients

- 13 yo FS Pug, rescued from puppy mill 5 years ago
- Has been treated with acupuncture, chiropractic, laser and herbal therapy for 5 years for severe DJD in most major joints in her body, chronic tracheal cough and recurrent cutaneous MCT.
- Has enjoyed excellent quality of life, MCT in remission for 3 years.
Dermatologic Disorders

- Common disorders treated
  - Allergic dermatitis
  - Otitis
  - Traumatic and pressure induced wounds
  - Perianal Fistulas

- Acupuncture effects
  - Reduces pruritis
  - Reduces inflammation
  - Promotes tissue healing
Other Conditions Acupuncture Can Treat

- Shock
- Behavior issues
  - Aggression, anxiety
- Gastrointestinal disorders
  - IBD, diarrhea, constipation, vomiting
- Respiratory diseases
  - Cough, asthma, dypsnea
- Urinary and renal disorders
  - Urinary incontinence, UTI, crystal/stone, renal failure
Linda and her 2nd chance...
Summary

• Safe and effective
  – For many different diseases
  – In many species
  – Long history
  – No side effect

• Can be integrated with conventional treatments

• An effective treatment
  – Pain management
  – IVDD and seizure
  – Atopy and skin allergy
  – Promote life quality for cancer and geriatric patients
Take home message
Use Acupuncture successfully:

- Geriatrics with minor complaints – quality of life
- Arthritis and Pain Management
  - Muscle soreness, poor performance
  - Neck pain, back pain
  - Arthritis, CCL (post-op and conservative care), hip dysplasia
- Acute gastro-intestinal problems
  - Vomiting/Diarrhea/Constipation
- Neurological Disease
  - IVDD, Facial Paralysis
Questions ??

drmitsie@gmail.com
Follow
The pethealerpodcast
Five Elements and Clinical Application

Mitsie Vargas DVM, MSTCVM, FAAVA, CTCVMP, CVMMP, CCRV, CVBMA

www.chiU.edu
Outline

• Intro to Five Elements
• External Pathologic Factors
• Elemental Constitutions
• Clinical Applications in SA Practice
Wu Xing

• Wu=Five

• Xing=activity/process (Element)

• A comprehensive organization of natural phenomena into 5 groups or patterns.
Five Elements

• A very important Fundamental Basis of TCM & TCVM

• The Theory of the Five Elements began
  • during the Zhou Dynasties (1046 BC- 221 BC)

• Traditional Chinese Practitioners used 5 Elements
  • to define the relationship between the organ systems of the body and nature
  • to understand the relationship among the body’s organ systems
  • to guides diagnosis and treatment
The Basic Elements

Each element has its own set of characteristics & properties which are used to analyze changes of natural phenomena

- Water: moistens downward
- Wood: can be bent and straightened
- Fire: flares upwards
- Earth: permit sowing, growing & reaping
- Metal: can be molded and hardened
Five Elements

- 5 Element theory was developed as a deep understanding of the laws of nature and universal order of all things in the world - It is a multifaceted and dynamic arrangement.

- Includes seasons, directions, color, sounds, growth, emotions, lifestyles and government along with organs
### Five Elements

<table>
<thead>
<tr>
<th></th>
<th>Wood</th>
<th>Fire</th>
<th>Earth</th>
<th>Metal</th>
<th>Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Season</td>
<td>Spring</td>
<td>Summer</td>
<td>Late Summer</td>
<td>Fall</td>
<td>Winter</td>
</tr>
<tr>
<td>Climate</td>
<td>Wind</td>
<td>Heat</td>
<td>Damp</td>
<td>Dryness</td>
<td>Cold</td>
</tr>
<tr>
<td>Direction</td>
<td>East</td>
<td>South</td>
<td>Center</td>
<td>West</td>
<td>North</td>
</tr>
<tr>
<td>Color</td>
<td>Green</td>
<td>Red</td>
<td>Yellow</td>
<td>White</td>
<td>Gray / Black</td>
</tr>
<tr>
<td>Flavor</td>
<td>Sour</td>
<td>Bitter</td>
<td>Sweet</td>
<td>Pungent</td>
<td>Salty</td>
</tr>
<tr>
<td>Sound</td>
<td>Shouting</td>
<td>Laughter</td>
<td>Singing</td>
<td>Weeping</td>
<td>Groaning</td>
</tr>
<tr>
<td>Emotion</td>
<td>Anger</td>
<td>Irritation</td>
<td>Joy</td>
<td>Preoccupation Worry</td>
<td>Grief</td>
</tr>
</tbody>
</table>

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### Five Elements

<table>
<thead>
<tr>
<th>Growth</th>
<th>Germination</th>
<th>Growth</th>
<th>Transformation</th>
<th>Reaping</th>
<th>Storing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zang Organs</td>
<td>Liver</td>
<td>Heart</td>
<td>Spleen</td>
<td>Lung</td>
<td>Kidney</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pericardium</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fu Organs</td>
<td>Gall Bladder</td>
<td>Small</td>
<td>Stomach</td>
<td>Large Intestine</td>
<td>Bladder</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intestine Triple Heater</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Orifice</td>
<td>Eyes</td>
<td>Tongue</td>
<td>Mouth</td>
<td>Nose</td>
<td>Ears</td>
</tr>
<tr>
<td>Sense</td>
<td>Vision</td>
<td>Speech</td>
<td>Taste</td>
<td>Smell</td>
<td>Hearing</td>
</tr>
<tr>
<td>Tissue</td>
<td>Tendons Ligaments</td>
<td>Vascular system</td>
<td>Muscles</td>
<td>Skin Hair Coat</td>
<td>Bones</td>
</tr>
<tr>
<td>Functions</td>
<td>Purification</td>
<td>Circulation</td>
<td>Digestion</td>
<td>Respiration</td>
<td>Elimination</td>
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</tbody>
</table>
## Five Elements

<table>
<thead>
<tr>
<th>Exterior</th>
<th>Nails</th>
<th>Complexion</th>
<th>Lips</th>
<th>Skin Pores</th>
<th>Head Hair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secretion</td>
<td>Tears</td>
<td>Sweat</td>
<td>Saliva</td>
<td>Nasal Fluid</td>
<td>Urine</td>
</tr>
<tr>
<td>Body Action</td>
<td>Spasms</td>
<td>Mania</td>
<td>Spitting</td>
<td>Vomiting</td>
<td>Coughing Wheezing</td>
</tr>
<tr>
<td></td>
<td>Tantrums</td>
<td>Depression</td>
<td></td>
<td></td>
<td>Trembling Shivering</td>
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<tr>
<td>Body Odor</td>
<td>Rancid</td>
<td>Scorched</td>
<td>Fragrant</td>
<td>Rotten</td>
<td>Putrid</td>
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<tr>
<td>Weakness</td>
<td>Looking</td>
<td>Walking</td>
<td>Sitting</td>
<td>Lying</td>
<td>Standing</td>
</tr>
<tr>
<td>Tongue Part</td>
<td>Sides</td>
<td>Tip</td>
<td>Center</td>
<td>Mid-tip</td>
<td>Rear</td>
</tr>
</tbody>
</table>
Cycle representation
Tissue associations
Sensory Organs
DOG CONSTITUTIONS AKA ELEMENTAL PERSONALITIES:

- **Wood** is the leader. Guard dogs embody it: Rottweilers & German Shepherds. Problems with ligaments, Liver, strokes, emotion is anger, don’t like needles.

- **Fire** is the cheerleader. Boxers and Pugs. Problems with Heart, behavioral issues, diarrhea and tumors in GI. Emotion is Joy or Fear. Scream like babies, use baby needles.

- **Earth** is the hippie loving child. Goldens for sure. Issues with Obesity, cancer and GI. Emotion is worry. Love needles, need a lot.

- **Metal** is the obedient and smart performer. Most herding breeds and show dogs like Border Collies. Cats! Problems with allergies, Diabetes, respiratory and skin. Emotion is Grief & Sadness. Ok with needles.

- **Water** is the shy, introvert. Usually a fear biter. Issues with arthritis, infertility and urinary. Live long. Emotion is Fear.
• **History and Complaints**

  • Healthy until started clusters of seizures 4 weeks ago

  • Kbr and phenobarbital stopped clusters
    • But still seizures (lasting 3-5 minutes) every 2-3 days

  • Conjunctivitis

  ![Lotus, Border Collie, s/f, 2 yo](image)

  [Seizures] → [Eye problems]
• **History and Complaints**

  • Congestive heart failure for 3 years
    • on Enalapril and stabilized
  • Recently, separation anxiety
    • Did not respond to medication

Peanut, 13y, c/m, Toy Poodle
The Five Elements

Wood → Fire → Earth → Metal → Water

“Would fire eat metal well?”

“W FEM W”
The Five Element Theory

- Spring: Green, Wood
- Summer: Fire, Late summer
- Fall: Metal
- Winter: Water
- Winter: Metal

Season changes

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The Five Element Theory

Spring

Fall

Summer

Late summer

Winter

Water

Metal

Green Wood

Fire Red

Earth

Go to TABLE OF CONTENTS
The Five Element Theory

- **Spring**
  - Wood
  - Fire
  - Red
  - Late summer

- **Summer**
  - Earth
  - Green
  - Yellow
  - Crop harvest season

- **Fall**
  - Metal

- **Winter**
  - Water
  - Yellow
The Five Element Theory

- Spring
- Summer
- Late summer
- Fall
- Winter
- Fire
- Wood
- Green
- Earth
- Yellow
- Water
- Metal
- White

The season to repair/make weapons/tools
plough rice fields
The Five Element Theory
The Five Element Theory

- Wood
  - Spring
  - Bile
  - Gallbladder
  - Liver

- Fire
  - Summer
  - Blood
  - Blood Vessel
  - Heart
  - Mind

- Earth
  - Later summer
  - Food
  - Nutrition
  - GI
  - Spleen/pancreas

- Metal
  - Fall
  - Defense
  - Surface
  - Skin
  - Lung

- Water
  - Winter
  - Deep
  - Bones/Spines
  - Kidney/Bladder
Help to determine the LOCATION of TCVM diagnosis

**Wood:** Hepatobiliary system  
- Tendons and ligaments  
- Eyes, feet

**Fire:** Cardiovascular  
- Mind, CNS

**Earth:** GI tract  
- Muscle  
- Mouth, gums, lips

**Metal:** Respiratory tract  
- Skin, nose, sinus  
- Large intestine

**Water:** Renal, urinary tract  
- Bone and joints  
- Reproductive tract
Five Elements

- Wood
- Fire
- Earth
- Metal
- Water
Five Elements: Sheng or Creation Cycle

- Wood nourishes Fire
- Fire nourishes Earth
- Earth nourishes Metal
- Metal nourishes Water
- Water nourishes Wood

The “parent” nourishes the “child”.

Used to treat Deficiency
Five Elements:

- **Wood** controls **Earth**
- **Earth** controls **Water**
- **Water** controls **Fire**
- **Fire** controls **Metal**
- **Metal** controls **Wood**

“The grandparent controls the grandchild”

Use to treat Excess
What is “Health”?

• Balance

• When the 5 Elements are in Balance then the Body is Balanced
Maintaining Balance
Pathology

*abnormal relationship among elements*

- Mother (element) insults Child (element)
- Child (element) insults Mother (element)
- *Cheng Cycle*: Overwhelming cycle (over control by one element)
- *Ru Cycle*: Insulting cycle (reverse control sequence)
Sheng Cycle
Physiology

Pathology
Ke Cycle (Physiology) control

Pathology

Cheng Cycle overcontrol

Ru Cycle counter-control
Treatment strategies

• Tonify Mother for deficiency (to nourish Child)

• Clear (reduce or sedate) Child for Excess

• Strengthen Grandchild for prevention
  - Strengthen SP for LIV problem
  - Strengthen KID for SP problem
  - Strengthen LU for HT problem
  - Strengthen HT for KID problem
External Pathologic Factors

- Wind
- Heat
- Summer Heat
- Damp
- Dryness
- Cold
WIND

- Rapid movement
- Intermittent
  - comes and goes quickly
- Not seen, but felt
- Abnormal motions
  - Trembling, shaking, seizures, convulsions, pruritus
- Associated with the Liver
HEAT

- Hot signs
- Fever
- Panting
- Red tongue
- Yellow coating
- Rapid pulse
- Cool-seeking

- Dry signs
  - mouth
  - Dry stool
  - Scant urine
  - Increased thirst

- Related to the Heart
Summer Heat

• Extreme heat
  • High fever
  • Profuse sweating
  • Weakness

• Shortness of breath

• Occurs in Summer
DAMP

- Wet, soggy
- Sinking
- Sticky
- Swelling, edema
- Lethargy
- Related to the Spleen
DRY

• Dry coat

• Dandruff

• Constipation

• Dry cough

• Related to the Lung
COLD

• Cold limbs and trunk
• Slow pulse
• Pale tongue with white coating
• Watery feces with undigested food
• Heat Seeking
• Poor circulation
• Related to the Kidney
Conclusion

• Five Elements is one of the most important fundamental basis of TCVM

• It guides clinicians to make a TCVM diagnosis

• It guides clinicians to select acupoints.
Questions?

drmitsie@gmail.com
Follow me @
ThePetHealerPodcast
Treating Cancer with Traditional Chinese Veterinary Medicine
Mitsie Vargas, DVM, MS, CVA, CTCVMP, FAAVA, CVMMP, CCRV, CVBMA

According to the American Veterinary Medical Association (AVMA) “Approximately 1 in 4 dogs will, at some stage in their life, develop neoplasia. Almost half of dogs over the age of 10 will develop cancer.” Cancer is one of the most common conditions seen in integrative practices and it can be successfully managed using the Traditional Chinese veterinary medicine (TCVM) approach of acupuncture, herbs and food therapy along with some immune boosting supplements.

There are several types of cancer in TCVM; Phlegm, Blood stagnation and Blood stasis (lumps, internal masses, tumors, neoplasia). Phlegm is an internal Yin pathogen and at the same time a condition. The root of Phlegm includes a deficiency of Spleen Qi, or a Kidney, Heart and Lung imbalance. How are the Zang Fu affected by Phlegm?

According to ancient Chinese philosophy the Kidney fails in its Yang function of transforming and excreting fluids which in turn causes the Lung to fail to diffuse or cause Qi to descend. This is the cause of the Spleen failure to fulfill its Yang function of transforming and transporting.

Clinical signs of Phlegm include a swollen tongue, with a sticky and usually white coating. Also a slippery / wiry pulse and a depressed Shen. Clinical signs of Phlegm depend on location. For example if it is in the Lung meridian you expect a cough, if in the Spleen, expect vomiting and/or stomach pain. If the Phlegm is in other tissues it results in masses, tumors, neoplasms, lumps, and lipomas. When Yang is deficient, Yin prevails and this results in accumulation of fluids that chronically causes nodules, lumps under the skin or in organs. Yin will continue to mistreat Yang and the Phlegm chases Qi throughout the body in this order: internal> external> and lastly is deposited in the meridians. It is considered that Phlegm is interconnected with Blood stagnation (vicious cycle). It can also be mixed with other pathogens such as Wind, Cold, Heat and Humidity.

Cancer etiology according to TCVM:
1. Qi Stagnation is caused by pathogens, emotions or stress.
2. Qi deficiency is caused by aging, constitutional weakness or chronic disease.
3. Trauma or heat can cause Blood to be drawn from the vessels and cause internal bleeding and Blood stagnation.

Clinical signs of Blood stasis include; masses, cysts, bruising, sharp and stabbing pain, a purple tongue and a choppy, weak and occasionally "wiry" pulse. External factors that cause Blood stasis include food and environmental toxins. Internal factors that cause Blood stasis include chronic inflammation and emotional stress.

The TCVM approach principles are to:
1. Boost the immune system (Wei Qi)
2. Break the stasis / move Blood
3. Tonify deficiencies (Blood, Qi, Yin and a Combined deficiency).

Acupuncture treatment include points that act as a general Qi Tonic like;
• LI-10, ST-36, CV-17 * (crossing of SP, KID, SI, TH), * LU-7 (confluent with CV), LU-9 (influences the pulse)

Also points that tonify the Qi of the Spleen and aid in Phlegm transformation like;
• BL-20/21, ST-36 / * 40, * SP2 (Mother) 4 (Chong Mai), 6 (junction), 9 (He-sea), 10 (Sea of Blood), HT7, TH4 (mother element)

Include points that improve immune function like;
• GV-14, LI-4 * / 10/11, GB39 *

In addition is important to add points that soothe the Liver’s Qi to relieve pain;
• LIV-3 * /4, GB-34/41

Treatment of the secondary effects of cancer is also accomplished with acupuncture:
• Anorexia use CV-12, Shan-gen
• Vomiting use PC-6, GB-34, CV-12, ST42
• Diarrhea use GV-1, SP-6, ST 36
• Ascites use SP-6, SP-9, BL-22

When using the Chinese Herbal medicine Approach the goal is to tonify Zheng Qi with the following;
• Qi deficiency with Heat --- Wei Qi Booster
• Deficiency of Qi, Blood and Yang: Shi Quan Da Bu

We also need to attack the Cancer mass (Blood stasis / Phlegm) with these;
• Stasis Breaker = Use for firm and large masses
• Max Formula = Use for soft and small masses, nodules

In order to treat additional symptoms you can add an herb to complement the anti-cancer choice using formulas to treat clinical signs;
• Vomiting: Happy Earth (Wei Cang He)
• Anorexia: 8 Gentlemen (Xiang Sha Liu Jun Zi)
• Diarrhea: Shen Ling Bai Zhu
• Bleeding: Yunnan Bai Yao
• Pain (stagnation of Qi-Blood) - Body Sore (Shen Tong Zhu Yu)

The Chinese Herbal approach is essential to:
1. Boosting the immune system with the formula Wei Qi Booster. Its ingredients include:

   Bai Hua She She Cao (Oldenlandia) - Inhibits cell mutation and tumor growth
   Zhi Lian (Scutellaria) - Inhibit cell mutation, inhibit tumor growth
   Chen Pi (Citrus) - Move Qi and transform phlegm
   Dang Gui (Angelica) - Tonifies the Blood
   Dang Shen (Codonopsis): Tonifies Qi and increases Wei Qi

2. Break the stasis and move Blood using the formula Stasis Breaker. Its ingredients include:

   Bai Hua She She Cao (Oldenlandia) = Inhibits cell mutation and tumor growth
   Ban Zhi Lian (Scutellaria) = Eliminates Heat toxin, inhibits cell mutation, inhibits tumor growth
   E Zhu (Zedoaria) = Purges the inside, breaks the Blood stasis and removes the mass
2. Break the stasis and move Blood you have another formula called Max Formula which includes the following ingredients;

- **Bai Zhi** (Angelica): Eliminates the cold wind and relieves pain
- **Da Huang** (Rhubarb): Clears stagnation and heat
- **Jie Geng** (Platycodon): Opens the upper Jiao and transforms the Phlegm
- **Mu Li** (Ostrea): Softens the hardness and clears the dough
- **Tian Hua Fen** (Trichosanthes): Eliminates Heat and promotes body fluids
- **Xia Ku Cao** (Prunella): Eliminates heat from the Liver and resolves the nodules
- **Xuan Shen** (Scrophularia): Removes heat and cools the Blood

Three other important formulas include the Formula Bone Stasis (for Bone Cancer), the Formula Stasis in the Mansion of the Mind (for Brain tumors) and Formula Stasis on the Mansion of the Blood also known as Xue Fu Zhu Yu (It is used to prevent recurrence of mast cell tumors after surgery).

3. Tonify Blood, Qi, Yin and combination deficiencies

**A-Blood Deficiency**
Clinical signs include: Pale and dry tongue, weak and deep pulse, general weakness, thin coat, dry skin with dandruff (flaky), dry pads, cold limbs, dizziness when standing and being prone to chills. Principles of treatment include to tonify and move the Blood.
Use the following Herbal Formulas:
- **Xue Fu Zhu Yu** (Stasis in the Mansion of Blood)
- **Shao Fu Zhu Yu** (Stasis in the Lower Palace)

**B- Qi deficiency**
Clinical signs include: Generalized weakness, fatigue, shortness of breath, poor appetite, weak pulse, pale tongue, weight loss, muscle atrophy and a weak voice.
Principles of treatment focus on tonifying Qi.
First of all, you can strengthen the Spleen with the herbal formula Four Gentlemen. The herbs in this formula include:

- **Bai Zhu** (Atraylodes) - Strengthens the spleen, tonifies Qi
- **Dang Shen** (Codonopsis) - Tonifies Qi
- **Fu Ling** (Poria) - Drains moisture, strengthens the spleen
- **Huang Qi** (astragalus) - Tonifies Qi

**C- Yin Deficiency**
Clinical signs: Dry and flaky skin, panting, thirsty, seeks cool, red and dry tongue, and a dense and fast pulse.
Principles of treatment are to tonify the Yin with the herbal formula Liu Wei Di Huang Wan (Rehmannia 6). It contains these herbs;
Fu Ling (Poria) - Drains moisture, strengthens the spleen
Mu Dan Pi (Moutan) - Cool the Liver
Shan Yao (Dioscorea) - Tonifica Qi, nourishes the Kidney

Jing Shan Zhu Yu (Cornus) - Nourishes the Yin
Shu Di Huang (Rehmannia) - Nourishes the Yin, the Blood and the Jing

D-Deficiency combinations
1- Deficiency of Qi, Blood and Yang
Clinical signs: General weakness, ears, limbs and back cold, fatigue, weight loss. Also a very pale tongue with deep and weak pulses.

2- Qi and Blood deficiency
Clinical signs: General weakness, fatigue, weight loss, pale tongue and deep and weak pulses.

Herbal formulas to treat these combinations:
1- Shi Quan Da Bu Wan
The general tonic formula Shi Quan Da Bu Wan, is also known as "Great all-inclusive toning decoction" and "Ginseng and Tang Kuei Ten Formula". First published at the end of the 11th century E.C.

2-Eight Treasures (Ba Zhen Tang)
It is more important that an animal eats than to get them to take herbs and supplements. This herbal formula promotes appetite.

General advice is to decrease carbohydrates and increase protein and fat akin to a keto diet. These three supplements have been hailed to be essential in the integrative treatment of cancer.

A- Astragalus
- Antiviral, antitumor, tonifies the digestive Qi and relieves fatigue.
- Protects the bone marrow from chemotherapy / radiation.

B- Medicinal mushrooms (Maitake, Reishi, Shitake, Turkey Tail)
- Antitumor, antiviral, anti-inflammatory, immune enhancer.
- Antiproliferative and anticancer.
- In vitro apoptosis of cancer cells.
- Synergistic with conventional cancer drugs.
- Reduces the side effects of conventional cancer drugs.

C- Essential Fatty Acids
- Inhibit tumor growth, metastasis, cachexia
- Blood and Yin Tonic

An appetite is essential for quality of life. A great option is to use Aqua with B12 on Shan Gen (on the dorsal edge of the nose/ skin).

Cases
1- Kiya
DLH cat was diagnosed with extremely large breast carcinomas in both chains 23/12/2010. The discussion about the poor prognosis and the quality of life began. The owner rejected the referral to specialists and opted for palliative care with TCVM. Sent on food therapy recommendations to relieve inflammation and resolve Phlegm.

(Clam water daily because it is a Yin tonic, facilitates the distribution of liquids and, therefore, moves it from the glands, we also changed to a canned shrimp-based diet to increase Qi and for the ability to download the mucus).

TCVM diagnosis: Blood Stasis and Phlegm
TCVM treatment: Stasis Breaker started on 1/29/11 and continued, acupuncture was performed 4 times.
Outcome: She enjoyed excellent quality of life for 30 months. Euthanasia on account of a thrombus in the spine.

Case 2 Wilson
English Setter, was diagnosed with lung cancer on 12/15/2009 through radiographs that were taken due to the appearance of a potbelly. Radiographs revealed an enlarged liver and a suspected lung neoplasm. Discussed quality of life and recommended an integrative conservative approach. The first year after diagnosis, he used multiple nutraceuticals and antioxidant mixtures along with western medications. The owner used food therapy together with fungi (turkey tail) and other homeopathic anticancer preparations after consulting alternative websites.

TCVM diagnosis: Lung Qi deficiency and Phlegm
TCVM treatment: Acupuncture, gold implants and Chinese herbal medicine Wei Qi booster. Outcome: He enjoyed great quality of life for 7 years!

Case 3 Grizzly
A Chocolate Labrador, sees another full-time veterinarian, arrived in 2010 for acupuncture due to anorexia, jaundice and severely elevated liver enzymes. We treated him with TCVM and he fully recovered and the owner was satisfied with the result at that time. He said he stopped coming to us because of the complete recovery of elevated liver enzymes and that we are more than an hour away for her. The owners returned in 2014 because the patient had a relapse of their liver problems and a biopsy was performed that reveals he has hepatocellular carcinoma. The usual veterinarian gave a poor prognosis and recommended euthanasia due to anorexia and weakness along with the diagnosis of cancer.

TCVM diagnosis: Stagnation and Phlegm of the Liver Qi

TCVM treatment: Acupuncture, Liver Happy and Food Therapy
Outcome: He lived his best life coming for monthly for acupuncture maintenance until his death at 11.5 years.

Cancer in horses is not as common as in small animals. The most common types of cancer include: Sarcoids, Squamous Cell Carcinoma (SCC), Melanomas (gray horses), Bone cancer and Sarcoids (Caused by Papillomavirus).

Sarcoids
Proliferation of fibroblasts and thickening of the skin with ulcerations
Usually diagnosed in young horses, ears and head
TCVM pattern is: Heat Toxin, Qi Deficiency and Blood stagnation
Treatment:
Acupuncture in Gv14, Baihui, LIV3, Li11,10 ST36 / 40
SCC
Most common cancer of the eye and the second most frequent type diagnosed (20% of all masses). It usually occurs in horses of 8-15 years. It is almost always solitary and grows slowly but there is metastasis in 20% of cases.
TCVM pattern is: Qi deficiency and Blood stagnation
Treatment:
Acupuncture in Gv14, Baihui, LIV3, Li11,10 ST36 / 40
Formulas: Wei Qi Booster, Wu Wei Xiao Du Yin; Topical application: Golden Yellow or Stasis Breaker

Melanomas (gray horses)
Most common cancer of horses with light fur, 80% will have Melanomas by the age of 15. 66% of gray horse melanomas are benign while dark-haired horses tend to have malignant melanomas.
TCVM pattern is: Qi deficiency, Phlegm and Blood stagnation
Treatment:
Acupuncture in Gv14, BL20 / 21, LIV3, Li11,10 ST36 / 40 SP6 / 9
Formulas: Wei Qi Booster, Max formula and / or Stasis Breaker

Bone cancer
TCVM pattern is: Qi deficiency and blood stasis in the bone
Treatment:
Acupuncture in Gv14, Baihui, LIV3, Li11,10 ST36 / 40 Sp6 / 9
Formulas: Wei Qi Booster or Stasis Breaker

Conclusion
Although Cancer in pets appears to be a rising medical issue in senior pets, it is one of the most treatable conditions. Phlegm has a root in Spleen Qi deficiency therefore, balancing the Earth element is important in the prevention of cancer. TCVM offers multiple modalities to treat Cancer and a multimodal approach is often the most successful. Using the integrative approach and TCVM, we can extend the longevity of cancer stricken pets and most importantly: significantly improve their quality of life!
Management of GI Disorders with TCVM

How do you apply the Traditional Chinese Veterinary Medicine (TCVM) principles to gastrointestinal disorders (GI)? It starts by taking a thorough history. This is very important because it can affect your TCVM pattern.

When asking for medical records from other veterinarians make sure to inquire the following; the kind of medications that were tried and how often were they given. Also, what was the patient’s response? What other diagnostic procedures were done? In addition, ask about changes in the environment, the appearance and frequency of stool, appetite, temperature preference, sleep patterns and most importantly what kind of diet is the patient being fed.

The constitution may be the most important key to your TCVM diagnosis. Earth, Metal, Water, Wood or Fire all have their peculiarities and of course in the case of a disharmony between Wood and Earth they can manifest into many gastrointestinal disorders.

The digestion process as described using TCVM principles includes the flow of energy or Qi moving upwards from the Spleen and downwards from the Stomach and the rest of the GI loops. The Spleen is like a sponge, if it gets full of water it gets heavy and can't move the energy up. If the sponge is over saturated, the water runs out and waters down, in the form of diarrhea. Whenever there are multiple patterns always treat the Spleen first, if the patient does not eat, there is no life. The Spleen is considered the mother of the Zang organs and is involved in the formation of blood, muscle and GuQi.

The Stomach main function is to receive the food and "mature" or decompose it while the Spleen transports and transforms it. In addition it sends the energy to the
Spleen and helps generate Gu Qi and Blood. The Small Intestine receives the energy from the Stomach and passes it to the Large Intestine. It also digests the food and separates the fluids into two parts: Clear & Turbid. The Large Intestine receives energy from the Small Intestine and reabsorbs fluids. The Heat affects Large Intestine and prevents it from absorbing.

The most common signs of a GI dysfunction include the following clinical signs; 1-Vomiting - Disruption of the Stomach by external pathogens that attack the meridian. Wind, Cold and Summer Heat are usually implicated. It can be a seasonal problem or acute problems and the symptoms include: headache, cranial abdomen pain, and a shallow, floating pulse. The following patterns can be identified;

**Excess**

a- Disruption by external pathogens
Includes Viruses, Bacteria, fungi, parasites, Cold, and DampHeat. The treatment strategy includes regulating the flow downwards and eliminating the pathogens. The commonly used acupuncture points include: PC6, ST36, BL20 / 21 and depending on the pathogen you can add GV14, LI4, GB20, and BL10.

b-Food Stasis
It is caused by over-eating or eating hard-to-digest foods. It causes food to become stagnant in the Stomach. It usually happens after a period without eating, when the animal is very hungry. Clinical signs include: distended abdomen, anorexia, vomiting after eating, vomiting that is acidic fluid or undigested food, bad breath, and feeling good after vomiting. TCVM signs include a slippery pulse and a tongue that has a greasy coating. The treatment principles include to remove stagnation and stop vomiting and to promote digestion. The acupuncture points include PC6, ST36/37/39/44/25, BL 20/21, LIV 3. Also using the herbal formula Bao He Wan will help mitigate the clinical signs.

c- Liver Qi Stagnation
It happens when the animal ingests toxins, different food or has a lot of stress. The Liver Qi tends to stagnate and it tends to over control SP / ST (Grandmother-grandson relationship). The Grandmother makes the energy of the ST go rebellious
and move upwards. Clinical signs include small volume vomit, acid reflux, and irritability. The patient shows sensitivity when you palpate LIV 13/14. The pulse is usually wiry and the tongue is purple. The treatment strategy includes; regulating the Liver, resolving stagnation, and stopping the vomiting. It also should focus on nourishing, cooling and calming the Liver. The treatment includes acupuncture at BL 18/19/20/21, GB41, LIV1/3, LI4, PC7 ST36. Clinical signs can also be controlled by adding the herbal formula Xiao Yao San.

Deficiency

a- Spleen/Stomach Qi/Yang Deficiency
The treatment strategy includes clearing the Heat and tonifying the Qi of the middle Jiao while also resolving the Cold. The treatment involves doing acupuncture at Bl 20/21, Sp9, LI10, PC6, ST36, Baihui, Gv 3/4. In addition using the Herbal formulas Eight Gentlemen or Sheng Ling Bai Zhu can make a big difference.

b- Yin Stomach Deficiency
The stress caused by emotional trauma or taking medicine transforms into heat and consumes Yin. Also chronic fevers and inflammation consume body fluids. If the Yin is not enough to balance the Yang. The Qi of the Stomach begins to move upwards. The clinical signs include; anorexia, vomiting, edema, lethargy, looking for cold surroundings, warm abdomen and legs, small and dry stools. The pulse is superficial and the tongue is red, dry, thin and with cracks in SP position. Treatment strategy to nourish and tonify the Stomach Yin and to resolve the vomit. The acupuncture points recommended are: PC6, BL 14/15/17/20/21, SP6 / 9/10, ST36, KID3. The effective herbal formulas are Jade Lady, and Stomach Happy.

2- Diarrhea Patterns can be subdivided in:

Excess

1- Disruption of the Stomach by external pathogens
Pathogens include Cold, Heat, Moisture and also parasites.
Cold passes by the environment (winter) or by eating and drinking cold things. The stools are watery, patients have pain and cramps, noisy borborygmi, the tongue is violet and the pulse is deep and weak.
The treatment includes heating the middle Jiao. Use acupuncture on GV1 / 3/4, ST36, LI10 and the Happy Earth formula.

Damp Heat etiology varies and it includes an organism that invades the GI like Parvovirus, Clostridium, Parasites. Stools are bloody with mucus, and a strong foul odor. Other clinical signs are anorexia, dark urine, red tongue with yellow coating and a pulse that is superficial and fast.
The treatment strategy is to stop the diarrhea, dry the damp and eliminate the Heat. Acupuncture on GV14 / 1, ST44, ST36, SP6, LI4 is usually effective. Hemo acupuncture at Wei Jian and Er Jian will help clear the Heat. The use of Red Back Door Formula can speed the resolution of the Damp.

2- Food Stasis
When they feed the animal too much, it retains food in the intestines and this situation causes pain. The diarrhea is usually watery. The pet exhibits anorexia, bloating, and the tongue is red with a fatty coating. The pulses are deep. Treatment is aimed at promoting digestion, calming the GI and moving stasis. Use acupuncture on GV1, ST25/36/44, LI10, LIV3, and Shan Gen. The Bao He Wan formula can help resolve the stasis.

3-Liver Qi Stagnation
Stress causes Liver Qi to stagnate and over control the Spleen. The animal is irritated, nervous, with abdominal pain, restless. The tongue is purple or red. The diarrhea is very painful and gassy. Treatment has to move the stagnation and strengthen the Spleen. Use acupuncture on GV1, ST36, LiV3, BL18 / 1920/21. Add the Xiao Yao San formula or Liver Happy to calm the Liver.

Deficiency
1-Spleen Qi deficiency
Any chronic condition affects the Spleen and weakens it. Clinical signs include watery diarrhea with undigested food and borborigmi. The animal is usually weak, lethargic and anorexic. Also, other signs include a pale tongue and a deep pulse. Treatment has to drain Damp, strengthen Spleen Qi and stop the diarrhea. Use acupuncture on BH, GV1, ST25/36, SP6/9, LI10, CV4, BL20/21 and the Shen Ling Bai Zhu formula.

2-Spleen and Kidney Yang Deficiency
Similar to Spleen Qi deficiency but more chronic and with Cold. The clinical signs include watery diarrhea with undigested food. The episodes occur generally in the morning. The animal is weak, lethargic, and anorexic with extremely cold limbs and trunk. The tongue is pale and the pulse is deep. Treatment has to strengthen the Qi of the Spleen and Kidney and stop the diarrhea. Use acupuncture on Bai Hui, Shen Shu, Shen Peng, Shen Jiao, GV1/4, ST36, Bl20/21, KID3/10, LI10, BL20 / 21. The Four immortals formula is also effective.

3- Anorexia

1-Stomach Disruption due to Cold
Drinking cold water with food is not beneficial. The Cold invades the stomach directly and causes stagnation and pain. It interferes with digestion as Qi does not flow well. Winter can cause cold outside to invade inside. Clinical signs include an animal that is weak, lethargic, and anorexic with a cold nose and ears. The tongue is Pale or purple and the pulse is deep. The treatment has to warm the Qi of the Spleen and Stomach and disperse the Cold. Use a combination of acupuncture on Bai Hui, GV4, ST36, Bl20/21, CV12 and Shan Gen. Adding the Happy Earth or Stomach Happy formulas can help.

2-Disruption of the Stomach by Heat
This is caused by a myriad of factors including a very hot environment, the accumulation of food in the Stomach, and working without drinking a lot of water. The clinical signs include: anorexia, thirst, dry stools, little urine, and bad breath. The tongue is red and the pulse is fast. Treatment must eliminate heat and regulate the Spleen and Stomach. Use acupuncture on Shan Gen, LI4, Er Jian, Wei Jian, GV14, ST44, Bl20 21 and the Jade Lady formula.
3-Food Stasis
It is caused by eating too much after working or exercising, eating too fast without chewing the food well, eating difficult-to-digest foods. Clinical signs include: anorexia, lethargy, bloating, bad breath. The tongue color varies but has a greasy layer and a deep and slippery pulse. Treatment has to eliminate stagnation and promote normal digestion. Use acupuncture on Shan Gen, LI4, Er Jian, Wei Jian, GV14, ST25/36, Bl20/21, CV12. The Bao He Wan formula is recommended.

4-Liver Qi Stagnation
Qi stagnates by: Mental and emotional stress, physical stress, chemical stress, and environmental stress. Clinical signs include: anorexia, lethargy, and pain. The tongue is purple and the pulse is strong, and slippery. Treatment principles include to eliminate stagnation, promote the appetite and calm the Liver. Use acupuncture on Shan Gen, LI4, Wei Jian, GV14, ST36, Bl20 / 21, LIV3. The herbal formulas to try are Xiao Yao San or Liver Happy formula.

Deficiency

1-Spleen Qi deficiency
The main causes are excessive work or exercise, poor diet, overeating. Clinical signs include; anorexia, lethargy, weight loss, difficulty excreting, pale, wet tongue and a weak, deep pulse. Treatment has to tonify the Qi of the Spleen and Stomach and promote appetite. Use acupuncture on Shan Gen, LI4, Wei Jian, GV14, ST25 / 36, Bl20 / 21, SP6. In addition the Eight Gentlemen formula can help boost the appetite.

4- Constipation Patterns
Excess
1-Heat
Heat is a pathogenic factor and it is caused by the environment, stasis of food, stagnation of Blood or Liver Qi.
This old adage: “If the river runs dry the boat does not float” is a great metaphor stating that the enemy of the LI is pathogenic Heat and dryness.
The clinical signs include difficulty excreting, dry and hard stools, abdominal pain, difficulty urinating with concentrated urine. There is also a red tongue with a yellow coat and a deep and fast pulse. The treatment has to remove Heat and resolve the stagnation. Uses acupuncture on ST36/44, BL20/21, LIV3, LI4/10/11, BL20/21, Wei Jian. The recommended Chinese herbal formulas are Xiao Zhang San, and Shao Fu Zhu Yu formulas.

2- Qi Stagnation
Symptoms also include excretion difficulty, dry and hard stools, abdominal pain, difficulty urinating with concentrated urine. The tongue is red with a yellow or purple coat and there is a deep and fast pulse. The treatment has to resolve the stagnation. Use acupuncture on ST3/44, BL20/21, LIV3, LI4/10/11, BL20/21, Wei Jian and GV1. The same formulas described above Xiao Zhang San, Shao Fu Zhu Yu are effective. Electroacupuncture works best and oftentimes results in defecation within hours of the session.

Yin/Blood/Qi Deficiency patterns can cause Constipation. The causes can be varied and include chronic diseases, poor nutrition, weak constitutions, advanced age, and overall weakness after childbirth. There is an interrelation of the Qi, Blood deficiency with the meridian of the Large Intestine. If there is Blood or Yin deficiency, the intestines are dried and the boat cannot float if there is no River. Clinical signs for Qi deficiency include excretion difficulties, dry or soft but not moving stools, abdominal pain, and a preference for seeking heat. The tongue is pale and wet, the pulse is deep and weak.

Clinical signs for Blood and Yin deficiency include hard, dry stools, difficulty excreting, The tongue is pale and dry. The pulse is usually deep and weak.

Treatment has to resolve the stagnation by moistening the intestines and strengthening the Qi, Blood and Yin. Use acupuncture on ST36/44, BL20/21, LIV3, Li4/10/11, BL20/21, Wei Jian, GV1 and you can add BL17, SP9/10 for Blood or Yin. Use the formulas Fan Xie Ye for a Qi deficiency and Dan Gui Cong Rong for a Yin or Blood deficiency.
5-Ulcers
It is usually caused by pathogenic Heat or *Yin* deficiency of the Stomach causing Fire in the GI system. Clinical signs include pain, dark blood in the stool, anorexia, vomiting, regurgitation and belching. The treatment includes acupuncture at PC6, ST44/45, SP4/10, LIV3, BL17, and Tiang Ping. In addition, the herbal formulas Stomach Happy and Jade lady can be of help.

There are two common problems in Horses known as Equine Gastric ulcers (EGUS) and Right Dorsal Colitis (RDC). It is verified through multiple studies that the incidence is 91% among Thoroughbreds. A study of 180 horses reveals that 87% are gastric and 63% colonic. They are related to excessive training, medication abuse (NSAIDS), and stress. Clinical signs include; dull hair, change of attitude, picky, weight loss, limp hind legs.

**Clinical Cases**
1-Chevy a 3yr old, M/N Yellow labrador Retriever
Presented as a TCVM consultation for chronic diarrhea and vomiting. He had been intermittently having GI upset issues for 6 months but the last 2 months he had been extremely sick and required visits to the ER, 2 blood transfusions and an exploratory laparatomy to determine the cause but no diagnosis was made. The owner had changed the diet multiple times without improvement. Extensive western diagnostics including a diarrhea panel were negative.
TCVM examination revealed an overweight dog with a deep right pulse, pale and swollen tongue with purple on the SP position. He was an Earth constitution and the owner revealed that he was the runt of the litter and had a severe anemia when he was a puppy. The stools were constant, watery, and yellowish. On the other hand, the vomit was intermittent.
The TCVM diagnosis was a Spleen Qi deficiency.
Balance acupuncture was performed at all the He-Sea points alternating Yin / Yang per leg. Points used were SP9, LIV8, KID10, BL40, GB34, ST36, LI11, TH10, SI8, LU5, HT3, and PC3. The herbal Sheng Ling Bai Zhu was prescribed at 1g bid x 3 months. Food therapy with Qi boosting vegetables and a wholesome home cooked diet was prescribed. Chevy stopped all vomit and diarrhea after just one
session. He received 6 sessions in total and the herbal was discontinued at 3 months and he hasn’t experienced any relapses up to this current time.

2-Sonic 4 years old, M/N Briard
Sonic came for a second opinion due to an intractable intermittent vomiting and diarrhea of 3 year duration. He was the alpha of the house and hated other dogs. He had severe separation anxiety from the owner. He was a Wood constitution with some Water traits (or perhaps poor socialization). He had an extensive GI workup at AVS specialists and the presumptive diagnosis was IBD. The TCVM examination demonstrated a wiry, strong pulse with some signs of heat in the ears. His tongue was purple and wet and his membranes were hyperemic but he was acting very stressed and had to be muzzled. The TCVM diagnosis was Liver Qi stagnation with some Phlegm fire in the Heart. Disharmony between Liver and Spleen was the presumptive cause of the GI upset. Due to the fractious nature acupuncture with vitamin B12 using the Balance Method on Jueyin / Yangming pattern was performed. The points used included; PC8 / 5, LI3 / 11, ST43 / 36, LIV 3 /8. The formulas Xiao Yao San and Happy Earth were prescribed. After just one session the Vomiting and diarrhea resolved completely.

3-Fruit Loops 2 years M/N DSH cat
Fruit Loops came in due to watery, strawberry colored diarrhea that had been ongoing for 8 months and treated with multiple western antibiotics. He had been an 11 pound cat prior to moving, losing a housemate and having tension in the household. The TCVM examination revealed that his personality was Earth, the pulses were deep and weak and his body condition score was a 4/9. His back was wet with watery feces. The TCVM diagnosis was a mixed pattern of liver Qi stagnation and Spleen Qi deficiency. The owner was advised to stop changing the foods and stick to a chicken based dry/canned diet with added fresh shrimp as treats. Balance Method Aquapuncture with B12 was done for a Taiyin / Yangming pattern at the following points; LU9 / 5, LI3 / 11, ST43 / 36, SP3 / 9 The herbs that were prescribed were Sheng Ling Bai Zhu & Liver happy. The cat gained 2.5 lbs in 4 sessions and the watery diarrhea resolved but he still had occasional soft stools. The owner was non compliant with the herbs and still
insisted on changing the foods constantly but the improvement on the body conditioning was dramatic.

4-Angel 15 months old, spayed Pitbull
Most Severe case of Spleen Qi deficiency
She was seen as a second opinion and quality of life assessment due to intractable diarrhea and severe weight loss for 8 months. The diarrhea was watery and smelly, and she had an average of 5 –7 movements a day and was dribbling diarrhea. She had a voracious appetite prior to being started on atopica, azathioprine and vincristine for a presumptive GI lymphoma. A quick scan ultrasound done at our office revealed lemon sized mesenteric lymph nodes and we aspirated and sent for cytology along with blood and fecal samples. She was treated with aquapuncture with B12 on GV1 / 14/20, ST36,37,39, BL20 / 21/23, SP9, LI11, LU7. The results came as inflammatory lymph nodes not lymphoma. The fecal came positive for hookworms. We discontinued the current treatment and started her on Sheng ling Bai Zhu, and a home cooked diet for boosting her Qi. After the first session she started to gain weight and solidify her stools. She received 6 sessions and was deemed totally recovered.

5-Mousse 19 years, neutered DLH
He was presented for anorexia, constipation, and a chronic constriction in the colon. He was on a strictly canned diet and was taking Miralax, cisapride and lactulose daily. He was vocalizing a lot at night and slept during the day. The owner had taken him to internal medicine specialists and endoscopic examination revealed the severe stricture in the descending colon. His body condition score was 4/9. The TCVM examination revealed a dull coat, dry pads, and dandruff. Palpation on Jian Jiao revealed coxofemoral arthritis and decreased ROM with crepitus on both stifles. He suffered insomnia, and had a red tongue with a superficial pulse that was weaker on the left. A TCVM diagnosis of Kidney and Spleen Yin deficiency was made. The treatment dry needle at Shan Gen, Anshen, Er Jan, Bl11,23,24,25 BH, SS, GV20,14,1. The food was switched to fish based and clam water was added daily. The owner reported that after his first treatment he voided that day and had continued to void daily. His appetite returned and his energy was much higher. The stricture in the colon resolved. The owner was extremely grateful because prior to her first visit she had been considering euthanasia and now knew
she had done the right thing. He was euthanized at 20 years old due to a mass in his abdomen.

6-Daniel 7 years old, Neutered DMH cat
Daniel had been suffering with constant diarrhea and chronic vomiting for 18 months. There were no changes in the food or household according to the owner. He was diagnosed with IBD but did not respond to prednisone therapy, in fact, the symptoms worsened. The previous veterinarian had changed the diet and Daniel refused to eat at all and almost died of hepatic failure with a fatty liver. The TCVM exam revealed a Wood personality, a purple and red tongue and a poor body condition 4/9. The TCVM diagnosis was Liver Qi stagnation and Stomach Yin deficiency. He was treated with acupuncture with B12 at Taiyin / Yangming + GV1 every 3 weeks. The owner was ecstatic that the vomiting resolved, and the diarrhea improved about 90%. The owner can not give any herbals and refuses to do any dietary changes but is happy to keep bringing Daniel to us for maintenance aquapuncture every 6 weeks.

Conclusions:
Chronic cases of vomiting, diarrhea, and constipation respond well to Traditional Chinese Veterinary Medicine. Determining the constitution of the patient is crucial to understanding the root of the TCVM pattern and the treatment principles. The Liver Spleen disharmony is one of the most common patterns encountered in the author’s clinical practice and understanding the relationship between the elements can help to find the solution to the problem. When the Liver is in excess it over controls the Earth causing GI upset. Most of the patients included in this paper were Wood or Earth constitution thus their signs of disease manifested in the GI system.
Food therapy recommendations should be directed at energetically correcting the patterns and providing micronutrients lost to commercial dog food processing. Food therapy should be integrated with the appropriate acupuncture points and in cases where there is a mixed pattern acupuncture can be used to correct the excess and food therapy can work on correcting the deficiencies. The use of Shan Gen is indicated in any case involving anorexia.
Using Aquapuncture with Vitamin B12 is especially a good technique for gastrointestinal cases because Cobalamin helps maintain a healthy gut biome. The
cobalamin sources are mostly gut bacteria but it has also been reported to be present at lower levels in other non-animal foods including other edible algae, some mushrooms, and fermented foods.¹

Oftentimes herbal medicine can help resolve chronic cases but identifying the TCVM pattern is extremely important to select the right ones needed to treat gastrointestinal problems with long term success. In some of the cases included in this presentation the herbal formula was given until the pattern changed then was either substituted or discontinued.

Using Shu and He level command points from the Taiyin / Yangming combination is a Balance method that uses ancient theory based on the I Ching Book of changes. The needling is done in Yin and Yang alternating fashion. For example if you needle the front left leg using the Taiying points LU5 & 9 then on the Right front the Yang Ming points LI3 & 11 should follow. Then the right hind leg needles are placed at the Taiyin points SP 3 & 9 and ending with the left hind leg Yang Ming points at ST 36 & 43. This specific needling infrastructure of Tai Yin/Yang Ming combination creates a very stable truss of energy that is effective for any problems in the frontal aspect of the animals which include gastrointestinal, cardiac, respiratory and skin. All the Balance Method cases discussed above resolved with very few sessions and resulted in dramatic turnarounds for those patients.

If you can’t figure out the underlying TCVM pattern, at least use ST36 and GV1 and you might be surprised how much you can help that patient.

References

Management of Kidney Disease With TCVM

The Kidney meridian has been described as the seat of the Ming men fire. This is where the essence of life or Jing is stored as well. We could compare Jing to telomeres, the genetic potential to replicate and restore diseased cells. The Kidney also controls the pupils, opens in the ears and dominates the musculoskeletal system. In western medicine Kidney disease is limited to just the urinary tract but we must consider the Traditional Chinese Veterinary Medicine (TCVM) perspective when approaching patients with renal impairment. The scope of this paper is mainly on the treatment of the patterns of disease of the TCVM Kidney, including those with western diagnostics that indicate elevation of creatinine (creat) and Blood Urea Nitrogen (BUN) blood levels.

The five main TCVM patterns to discuss are Kidney Qi Deficiency, Kidney Yang Deficiency, Kidney Yin Deficiency, the combination of Kidney Qi/Yin Deficiency and Kidney Jing Deficiency. A quick overview of Lin Syndrome, which includes cystitis and urolithiasis, will be included.

**Kidney Qi Deficiency** is characterized by the following clinical symptoms:
Urinary incontinence or dysuria, stranguria, weak hind legs/ back. The tongue is usually pale, wet and oftentimes is swollen. The pulses are deep and weaker on the right side. Treatment with the following acupuncture points are recommended: BL-23, KID-3, KID-7, KID-10, CV-4, CV-6, BL-22 and BL-39. The herbal Jin Gui Shen Qi is a good choice for this pattern.

**Kidney Yang Deficiency** includes patients with pale complexions, aversion to cold and actively seeking heat, lethargy, poor teeth, deafness, clear urine, urinary incontinence, general weakness, edema and a sore back. The tongue is swollen,
pale, wet and oftentimes lavender in the Kidney position (root). The pulses are deep and weaker on the right side. Treatment should include the following acupuncture points: GV-3, GV-4, Bai-Hui, Shen-Shu, Shen-Peng, Shen-Jiao, BL-26, KID-7, KID-10, CV-4, CV-6, BL-22 and BL-39. The herbal formula You Gui Wan is a good choice to raise the Kidney Yang.

**Kidney Yin Deficiency** has the following clinical signs: Dysuria, stranguria, weak hind limbs and back, dry throat, dry skin, insomnia, erythema, hearing loss, infertility and weight loss. The tongue is red and dry. The pulse is deep, threads and weaker on the left side. Treatment should include the following acupuncture points: KID-3, BL-23, SP-6, KID-7, KID-10, CV-4, CV-6, BL-22 and BL-39. The herbal formula Rehmannia 6, also known as Liu Wei Di Huang, has been used to increase the Kidney Yin.

**Kidney Qi/Yin Deficiency** is a fairly common combination problem seen mostly in senior pets. The symptoms are a combination of both Qi and Yin deficiencies and vary with each patient. Treatment varies with the symptoms but the following acupuncture points are recommended: BL-23, KID-3, KID-7, KID-10, CV-4, CV-6, BL-24, Shen Shu, and BL-39. The herbal choices depend on the predominant complaints and symptoms for this pattern.

**Kidney Jing Deficiency** is characterized by premature aging, congenital disorders, infertility, failure to thrive, and early development of either Kidney Yin or Yang deficiencies. The tongue varies and could either be pale or red. The pulse is weak and oftentimes deep. Treatment can include acupuncture points like KID-3, BL-23, BL-26, SP-3, ST-36, BL-21, BL-20, CV-4, CV-6, BL-22 and BL-39. The formula Epimedium Powder (Sheng Jing San) can be used for the first year of life if the diagnosis is done as a puppy/kitten.

What else can we do to help our patients with renal disease? Remember to feed the patient and feed those kidneys. Consider custom homemade diets for individuals, using the energetics according to the pattern. In order to assure that the pet is not
malnourished, consult a veterinary nutritionist or resources like www.balanceit.com. Supplement with omega fatty acids, krill oil, cod liver oil, and nutraceuticals like Amminovast. Use the classical acupuncture point Shan Gen to stimulate appetite.

If there is concurrent Spleen Qi deficiency you will see the following symptoms: nausea, vomiting, lack of appetite, and loss of body weight. The formula Eight gentlemen (Xiang Sha Liu Jun Zi) can restore the Spleen Qi. Acupuncture points to tonify the Spleen Qi include BL-20/21, PC-6, ST-36 and Sp9.

Lower urinary tract infections are the result of Damp Heat accumulation from eating oily, spicy foods, heat pathogens, Spleen qi deficiency, overuse of antibiotics and stress. Clinical symptoms include: Hematuria, stranguria, pain, and spasms. These infections are usually intermittent. Antibiotics damage Wei Qi, because they affect SP and LU. Steroids cause damage to the Yin. The presence of uroliths indicate Damp-Heat and underlying Kidney Qi Deficiency. Treatment should include these Acupuncture points to clear the Heat and Damp; Heat: Wei-jian, Er-jian, BL-66, GV-14, LI-4/11 Damp: SP-6/9 Bladder: CV-3, BL-28, LIV-3, BL-39, BL-22

The Chinese herbal formulas can be used to target the worst symptoms as well. For example;
UTI: Ba Zheng San (eight righteous) Crystal / stones: Pai Shi Wan (Crystal stone formula) Hematuria: Xiao Ji Yin Zi (Red front door)
Some special acupuncture points that are very useful in treating lower urinary tract infections include points in the Triple heater (TH) meridian because it controls the flow of the Qi ascending and descending and also the Qi that enters the organs. Since BL39 is the TH He-sea point it does help any incontinence or dysuria problem. The Ren Mai (CV) channel directs the Lung Qi down and causes the Kidney to receive the Qi. Consider that CV3 is important to open the urethra.

Also, the Lung Qi has to go down and LU7 helps it do that because it's the confluent point with the CV meridian.

How is the prostate considered in TCVM? Although the prostate is part of the reproductive system, it is inter related to the urinary system and belongs to the
Kidney meridian. It stores the prenatal Jing. The excessive sexual activity consumes Jing. Perhaps castrating dogs before a year could have consequences.

Treatment using the following acupuncture points: Sp4, Kid3, Bl66/62/39/28/23, Liv3, Si3, and CV3. The formula Prostate Invigorator along with supplementation with Saw palmetto has been very effective in our practice.

Clinical cases:

1- Shadow was a 16 yrs, M/N Poodle. Constitution was Earth. Had urinary incontinence, was blind (cataract) and deaf. His Creatinine was 3.5 and the Bun 44.
TCVM exam revealed that the tongue was pale and swollen. His pulse was weak, and deep, worse on the right side. He was sent on Hindquarter weakness & Suo Quo Wan and the incontinence was resolved.

2-Biscuit was a 15 yrs F/S Pug. Constitution was Fire. She was presented for quality of life exam because she had urinary incontinence, machine murmur ⅝, cataracts, and was almost deaf.
TCVM exam revealed a pale and swollen tongue. Her pulse was weak and deep, especially on the right side. She was generally weak and presumed to have global Qi deficiency. She was sent on the herbal formulas Jin Gui Shen Qi and Heart Qi tonic. Food therapy was started with sweet potato and pumpkin added to her diet.

3-Phoebe was 13.5 yrs F/S DSH. Constitution was Wood. She was on Stage 5 Kidney Failure with a Creatinine at 5.0 and a Bun at 77. She was vomiting, had steady weight loss, was hiding, and couldn’t jump or climb onto the couch.

TCVM exam revealed a pale tongue, and bilateral deep pulses.

Treatment consisted of acupuncture at Kid3 / 10, HTJJ, Liv3, BL62,60,40,39 GV20,14 Si9 Anshen St36,37,44. She also was prescribed the Chinese herbal formula Hindquarter weakness and showed dramatic improvement and extended longevity. Nutritional therapy for her consisted in shrimp, canned salmon and her fancy feast.

4-Linda was a 4 month old F/S mixed breed. Constitution was Water. She was rescued from the streets, stunted growth, incontinence from baby, exploratory
during OVH was normal. It got worse since the surgery, she didn't respond to Proin. The purpose of their visit was for an euthanasia consult. TCVM exam revealed a weak right pulse yet bilaterally weak on the KID position. Her tongue was pale, her coat was dull, and she was dribbling urine.

She was treated with acupuncture at BL23,39,40,62, KID 3,7,10, SP4, Shen Shu and Bai Hui. She was also sent on the Chinese formulas Suo Quo Wan and Epimedium. Due to her dramatic response to the acupuncture (90% control of incontinence) we placed a gold implant in BL39. That solved the problem 100% and she is still doing great over 4 years later.

5- Katie Sue, 9 months female Shih Tzu. Only survivor of a litter of 6 who died of renal failure before the first year of life due to renal failure (Polycystic Kidney disease). She was fed with Purina NF from 8 weeks and her Creatinine values were 2.6, BUN 33. After her OVH they rose to 4.0 and 44. She was put on Epimedium formula and acupuncture monthly until her death at 11 yrs old. Over her life she had seizures controlled with phenobarbital and the formula Di Tang Tan, also suffered from atopic dermatitis, all related to her Jing deficiency.

Pogo, a male neutered Maltese, was presented at 8 years of age for a second opinion on a chronic cough that had been diagnosed as collapsing trachea. A machine murmur of ⅞ intensity was heard and a radiograph revealed severe cardiomegaly with a globoid heart silhouette, pulmonary hypertension and small bladder uroliths. After weekly acupuncture for 3.5 years due to CHF he presented as an emergency due to urinary obstruction. Radiographs revealed increased uroliths in the bladder, new uroliths in the kidneys and 4 uroliths in the urethra, due to the high anesthetic risk, a urinary catheter was passed and hydropulsion of the urolith into the bladder was done. The owner refused to go to a specialist for surgery and opted TCVM approach. He was prescribed Crystal Stone formula. The same unblocking procedure was performed multiple times during 6 months. However, Crystal Stone formula reduced the stones from 18 to 4 in 7 months.

In conclusion, TCVM is a great tool to treat urinary tract conditions. Usually, treatment also requires allopathic medicine, herbal formulas and diet changes to control symptoms. With acupuncture we can extend the longevity of patients with problems in the Kidney meridian and Zang organ.
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Focus On Infection
IDENTIFYING YOUR CLIENT

There is no such thing as an average backyard chicken flock or client. Some backyard chickens are truly kept in the backyard and are pets, with or without benefitsry. These clients are often willing to spend far more money on treating a sick or injured bird than the cost of replacing the birds or the value of the eggs or meat they produce. These clients range from very knowledgeable to “google smart” and possibly misinformed. This is primarily individual bird medicine, although more than one bird may be affected.

Other clients may keep birds in the backyard but see the birds as primarily as an inexpensive source of eggs. These clients are unlikely to spend much money on treatment, and often will not go beyond a telephone consultation phase (preferably free). These clients are usually not well versed in chicken knowledge and may be apathetic about poultry health or welfare.

The third “type” of backyard chicken keeper is the semi-professional. Supply management rules allow people to keep up to 100 laying hens or 300 broiler (meat) chickens without having quota (essentially a license to produce commercial poultry than needs to be purchased). These small flock owners are producing poultry on a small scale, for profit. Backyard and fancy bird breeders fall into this category as well. Treating these flocks is more of a case of population medicine, with financial constraints. Small flock owners are often very knowledgeable about raising poultry, including on management and nutrition issues, and will invest significant amounts of time, effort and finances, provided it makes economic sense for the flock. Individual bird medicine is not a large component of this type of flock care.

TREAT BACKYARD CHICKENS AS FOOD PRODUCING ANIMALS

There is an aspect of backyard chicken medicine that takes a different mindset from other small animal medicine is that other patients do not produce products directly for food. Because of this, considerations around therapeutic drugs must be taken into consideration. Antibiotic and other treatment have to be evaluated for withdrawal times, and clients need to be informed on the food safety aspects of the patients. There are very few drugs that don’t have a withdrawal time, and during this time, all eggs from treated birds cannot be used for human food. There are also drugs that will render eggs inedible for the remainder of the bird’s life, if used. Examples of medications that result in birds being permanently removed from food production are common, effective drugs like Baytril and Ivermectin. The best way to treat backyard birds safely is to consult with CgFARAD (Canadian Global Food Animal Residue Avoidance Databank cgfarad.usask.ca). gFARAD is a strategic initiative to ensure food safety in all food producing animals. You can send in your treatment regimen (age of bird, drug, dosage, duration of treatment), and they will respond with a withdrawal time for the eggs or meat involved. It is advisable to check with them before treatment so that long term or permanent withdrawals can be avoided. It is also advisable to perform a “dry run” on the site to ensure you know the required information when making a request, so there are no delays when trying to get information on a time-sensitive case.

This human safety concern is the reason that the CFO requires vets who wish to treat backyard poultry to be accredited as a mobile poultry practice. This ensures that vets are conversant with the food safety aspects of treating and managing backyard poultry. The requirements to
acquire this accreditation are designed not to be onerous, and CVO is interested in working with vets to increase the number of vets willing and able to treat backyard poultry.

**NUTRITION**

In North America, our culture is such that people are exposed to pet ownership directly and through many media sources. As a result, there is an inherent widespread knowledge of the basic needs of dogs and cats. The same is not true of poultry. Knowledge of basic husbandry of backyard chickens needs to be acquired, and there are many sources of information available with a wide range of accuracy. Commercial breeds of birds (leghorn and sex-link layers and Cornish meat birds) are genetically driven to lay a lot of eggs and grow quickly. If the nutrition is not excellent, these birds will very quickly (potentially in a couple of days) develop metabolic diseases and irreversible pathology.

If treats and “scratch” make up more than 5-10% of the bird’s intake, the makeup of the ration is significantly altered. Remembering that birds will eat between 100 and 130 g of feed per day, it is easy to overfeed treats. The most reliable way to ensure that nutrient requirements are met is to feed a professionally developed layer or meat-bird ration. If a chicken keeper wants to make their own ration, it is crucial to ensure that the mineral content and protein levels are sufficient. Remember that all calcium sources (egg shell, oyster shell and limestone) are calcium carbonate. Egg shell is calcium phosphate, so if the ration doesn't have appropriate amounts of phosphorus, metabolic issues will result. If feeding a “mixed flock”, a grower ration should be provided, with free choice calcium must be freely accessible so that the birds in lay can meet their mineral requirements.

The best source of current nutrition requirements can be accessed through commercial genetic company guides (Hendrix Genetics, Lohmann, Hy-Line Genetics). The requirements are consistently changing because of the increased number of eggs laid, and increased rate of growth of meat chickens. Backyard chickens will not have as intense a nutritional need, because their environment will not be as encouraging to production as commercial birds, but it is still very high.

**HOUSING**

Similar to nutrition, there is not a “cultural” knowledge of the housing needs for chickens. In Ontario, with its wide range of weather conditions and predators, housing that provides good air quality, litter condition, protection and temperature is complicated. A good resource for the minimum requirements for housing minimum standards are the NFACC Codes of Practice. There are different ones for laying hens and meat birds. These documents contain minimum requirements for housing and care of the different classes of poultry. These standards are welfare based and applicable to backyard flocks and are excellent starting points for evaluating the totality of the environmental needs.

Common problems caused by poor housing conditions include frostbite, which is often the result of high humidity and cold temperatures; corneal burns from high ammonia caused by poor airflow; bumblefoot from moist litter or poor perches; injuries from predators gaining access to the flock, and difficulties accessing water in the winter.

Ventilation is the most difficult aspect of backyard housing to get right. There needs to be a balance between maintaining comfortable temperatures while controlling the moisture and humidity to levels that don’t cause discomfort or disease. The most common mistake is to reduce airflow in the coop to aggressively to maintain temperature, which causes increased risk of frostbite, ammonia burns, and bumblefoot. Chickens can handle quite cold temperatures as
long as the humidity is low, and they have the ability to huddle together to conserve heat. Decreasing the air volume of the coop increases the amount of outside air that can pass through the coop while maintaining comfortable temperatures. The type and amount of bedding, as well as hygiene (how often the coop is cleaned, if they use dropping boards, etc) also have a huge impact on air quality. Because of the wide variety of housing schemes for backyard flocks, evaluation of the welfare of the birds needs to be outcome based, with knowledge of how to manipulate the housing parameters to solve issues.

VACCINATION IS POSSIBLE

There are effective vaccines licensed in Canada that control Mareks Disease (MD), Infectious Laryngotracheitis (ILT), Infectious Bronchitis (IB), Newcastle Disease (ND) Fowl Pox, Salmonella, E. coli and Infectious Bursal Disease (IBD). Unfortunately, poultry vaccines are not available in small aliquots. The majority of vaccines are packaged in 1000 to 10000 dose vials, and have a very limited effective life once mixed. Most vaccines are not considered effective after about 2 hours after mixing. Another complication is that MD vaccine (and any combination products based on MD) need to be stored and maintained in liquid nitrogen.

A potential solution revolves around the fact that most of the commercial industry is interested in improving the disease status of backyard flocks. If you can partner with a commercial hatchery or poultry vet, many will be willing to sell small numbers of vials of vaccine. Acquiring and maintaining a liquid nitrogen dewar is neither cost prohibitive or complicated. Also, despite the large amount of wastage when vaccinating small numbers of birds, poultry vaccines are quite inexpensive on a per-bird basis. Commercial poultry receive all their vaccines at a cost of less than $1 per bird. It is possible to vaccinate backyard flocks for less than $15 per bird cost, which is pretty well accepted by backyard owners.

Mareks vaccine needs to be given before the chicks are exposed to field strains, so the earlier, the better. Ideally, Mareks vaccine is given at day of hatch, but there is no downside to vaccinating after field exposure, except for the fact that the vaccine won’t be effective. During our vaccine clinics, we vaccinate any unvaccinated hens with Mareks, regardless of age. The type of vaccines and age of administration will depend on the region you are in, and the disease prevalence. There are several technologically advanced vaccines which use genetic recombinants to protect against more than one disease at once. Most of these vaccines are based on Mareks disease because herpes viruses are effective carriers. Remember that if the bird vaccinated with a recombinant has already been exposed to MD, not only will the Mareks Disease vaccine not be effective, but there will also be no protection for the disease that was attached to it. Vaccinating for Mareks disease is possibly the most pressing need for backyard chicken health, since MD is one of the most prevalent diseases that affect backyard poultry, it is almost impossible to clean from the environment, and is untreatable once established.

MD vaccine must be given via a SQ injection, but the others are given via eye-drop or orally. These vaccines can easily be given by owners, with training. It is possible for booster vaccines to be sent home reconstituted and administered by owners, as long as they are administered within a couple hours and kept cool during transit. Increasing the number of vaccinated birds in the provincial backyard flock will go a long way to decreasing the amount of illness faced by these birds.

COMMON FLOCK DISEASES

Avian Influenza has been dominating the poultry world this year, with over 140 isolations of the disease across Canada this year, including 77 zones that have been cleaned, certified and revoked. Non-commercial flocks account for just over 50% of the isolations. This disease is
widespread in migratory waterfowl, and spreads through even miniscule exposure to these ducks and geese, or other animals that may predate or scavenge on them. Biosecurity, and keeping birds indoors and isolated is the only defense. The disease is usually 100% fatal within a few days, and all birds in the affected flock will be destroyed via CFIA control programs. This is a devastating disease to have and counselling your clients on biosecurity is crucial.

Coccidiosis is the most common infectious disease of all poultry, including backyard birds. The famous clinical sign is bloody droppings, but there are 5 species of Eimeria that infect chickens, and all of them cause disease including general ill thrift. It is not uncommon for birds on medicated feed to get infected by coccidiosis and need treatment. Cocci infections increase the risk of Necrotic Enteritis caused by Clostridium perfringens, which can be a normal part of gut flora. Young birds are more susceptible to cocci, and birds will develop immunity over time. Once the birds are brought to a vet, diagnosis through fecal float, and treatment with Amprol is simple and effective, even for flocks that are on medicated feed.

Intestinal worms are very common in backyard flocks, but with minor clinical signs. The major signs are mild ill-thrift and the rare roundworm that finds its way into an egg. Nothing will instigate a phone call like a worm in a freshly cracked egg. Panacur aquasol (note, this is different from Panacur/Safeguard, which has a 23 day egg withdrawal) is a very effective anthelmintic and has no egg withdrawal, but is sold in large/expensive aliquots. It is easy to divide up the liquid into backyard flock sized amounts, but make sure when you purchase that the expiry date is long enough so you use up the jug before it expires. Other intestinal worms are uncommon, but not rare, with flatworms and tapeworms sometimes found. Many owners think they have gapeworm, but this is exceedingly rare, and is usually confused with other problems that cause open mouth breathing.

External parasites are also extremely common in backyard flocks. Northern Fowl Mites, Red Mites, lice, and occasionally bedbugs are found in Canadian backyard chickens. The most common are Northern Fowl Mites and lice. These can be treated effectively with Elector PSP or Pemethrim with no egg withdrawal. Often mites and lice are in such high numbers that they cause significant blood loss and illness.

Respiratory infections with Mycoplasma, Coryza, Infectious Bronchitis also represent a significant portion of flock level diseases in backyard flocks. Treatment with appropriate antibiotics (while observing withdrawals) is effective in managing the diseases, but they are extremely difficult to eradicate. Any flock that has tested positive for Mycoplasma should be considered positive for life, and a source of infection for any naïve birds they encounter.

**COMMON CONDITIONS**

Individual bird medicine is made up of a large variety of complaints. Many backyard keepers will attempt diagnosis, but mistakes are common. A lot of birds are brought to the clinic with suspicion of being egg bound, which is usually caused by calcium deficiency, but end up actually being birds with ascites caused by internal laying or tumors. Wry Neck, which can be caused by vitamin E and Selenium deficiencies, are common. It is logistically difficult to source and deliver vitamin E and Selenium supplementation, because of the way these products are marketed for commercial level feed supplementation.

Bumblefoot is a localized infection of the foot that causes a great deal of pain and should be surgically debrided and lavaged. Many owners will do this minor surgery themselves without analgesia, but this should be strongly discouraged. Anaesthetic protocols can be found in the...
exotics formulary, but the range of responses of various chickens to the drugs make all surgeries nerve-wracking.

Injuries are very common, either from inter-bird aggression, predator attacks or just misadventure. Treatment of wounds with flamazine or other colloidal silver ointments is rewarding because of the strong ability of chickens to heal wounds. Fractures heal well too, if the bird can receive internal or external fixation and can be isolated from flockmates, so their aggression doesn’t aggravate the injury.

Some owners are uncomfortable with older hens laying eggs regularly. This can be controlled with Deslorean Implants, which can be used via Emergency Drug Release from a company in Australia. The hormone implants also help birds that have suffered from egg yolk peritonitis caused by internal laying. It is a relatively expensive treatment, but is appreciated by owners who are attached to their pets

EUTHANASIA

As mentioned above, chickens of various breeds respond differently to many pharmaceuticals. This is also frustratingly true of euthanasia. Experimentation with many sedative drugs, and volumes of barbituates has still not resulted in a reliable protocol for euthanasia. It is not unheard of to dose a bird with volumes of drugs appropriate for a large dog, and still have it be ineffective. Do not be afraid to continue to experiment to find a protocol that works consistently. Other options include physical methods such as cervical dislocation or decapitation. These methods are effective and humane but may not be acceptable to owners.
WHAT DOES A POSITIVE PCR REALLY MEAN? LESSONS FROM RINGWORM, PARVO, PANLEUKOPENIA AND COVID-19

Linda S. Jacobson BVSc MMedVet(Med) PhD

Clinicians would do well to follow Dr. Scott Weese’s diagnostic advice: “never do a test without a (good) reason” and “never do a test without a plan to use the result”. To this one might add: “never do a test whose results you are unable to interpret”. The exquisite sensitivity of polymerase chain reaction (PCR) and the ubiquity of test panels represent both opportunities and pitfalls in the diagnosis of infectious diseases.

The first problem to overcome is the security and confidence engendered by a binary result, given that PCR results are typically reported as “Positive” or “Negative”. This may discourage further analysis or questioning by triggering multiple cognitive biases, including anchoring, availability, commission, confirmation, diagnosis momentum, and framing effect.\(^1\)

PCR uses DNA polymerase enzymes to amplify pathogen DNA to a detectable level.\(^2\) RNA can also be detected through reverse transcription, which converts RNA to DNA. Specific primers for the target pathogen are introduced into the sample mixture, and attach to the pathogen DNA if it is present. Multiple heating and cooling cycles follow, with the target DNA doubling during each cycle (Figure 1). PCR is therefore an exponential process. The amount of DNA in the final mixture is proportional to the number of pathogens in the initial sample.

The PCR tests we are familiar with are quantitative real-time (qPCR) tests. This groundbreaking development has allowed PCR to be performed rapidly and quantitatively. Quantity of pathogen is measured by binding a fluorescent marker to the amplified DNA. qPCR tests use a defined number of amplification cycles, typically 40. The cycle threshold value (Ct value) is the number of cycles required to be able to detect the fluorescent signal from the amplified DNA (Figure 2). The more pathogen in the initial sample, the fewer cycles required for detection. The Ct value is therefore inversely proportional to the amount of pathogen in the sample and represents an exponential process. Typically, a large amount of pathogen would yield Ct values of about 15-25, while a small amount of pathogen would yield Ct values of 35-39.99.

PCR can detect as few as 5-10 initial copies of a pathogen, in amounts far too low to be infective and often not reflecting disease caused by that organism. Overdiagnosis is a major potential pitfall of PCR. Assuming an otherwise reliable test, negative PCR results from a positive animal will only occur if there was no pathogen in the initial sample, if the amount of pathogen falls below a diagnostic cut-off, or if the sample was too small or incorrectly stored. “False positive” results can occur through laboratory error, sample contamination, or if the pathogen is indeed present, but the disease is not. True false positive results are not possible for the pathogen, because PCR primers are highly specific, but a “false positive” diagnosis of the disease is certainly possible. It is not possible to directly extrapolate infective dose from Ct values, but it can generally be assumed that the higher the Ct value, the less likely an animal is to be contagious. If a result is positive and there is doubt about the diagnosis or whether the animal is likely to be contagious, Ct values should be requested from the laboratory.
Closely related parvoviruses cause canine parvoviral enteritis and feline panleukopenia. Low-level, clinically insignificant, shedding of these viruses is quite frequently detectable by qPCR, particularly after modified live virus vaccination.\(^3,4\) Virus signal can also be detected for weeks post-infection, long after virus isolation (demonstrating viability) is negative.\(^5\) To avoid misdiagnosis and/or prolonged isolation times, so-called technical cut-off Ct values are set, below which results are reported as positive, and above which they are reported as negative, even if small amounts of virus are detected.\(^6\) Determination of precise cut-offs is complex.\(^7\) The cut-off for the IDEXX canine and feline parvo qPCR is Ct ≤ 26 (≈1.6 \times 10^{6} virus particles per gram of feces). Virus levels close to the cut-off may still be clinically significant. For this reason, Ct values should ideally be reported for all results, and should be requested if they are not provided and results are difficult to interpret.

Positive qPCR results may occur for a few days after modified live virus vaccination, and low levels of vaccine virus can persist for longer periods\(^3,4,8\). Vaccine virus shedding peaks at 3-7 days, as for natural infection. This may pose a diagnostic dilemma. A false positive diagnosis of parvo or panleukopenia could lead to unnecessary isolation or even euthanasia. Vaccine positives are much less likely for rapid antigen tests, which are recommended for initial testing. qPCR should be reserved for animals that are strongly suspected to be infected but have a negative rapid test, or for specific surveillance purposes in high-risk groups.

qPCR was highly sensitive for diagnosis of \textit{Microsporum canis} infection in shelter cats, with the enormous advantage of allowing rapid diagnosis, and rapid rule-out for the many suspects that are ultimately negative. Results are available in 2-3 days, compared with 2 weeks for fungal culture.\(^9\) While dermatophytosis is eminently treatable, it carries a resource and cost burden, requires weeks of isolation in shelters or catteries, increases shelter length of stay, affects kitten socialization, and may result in euthanasia in over-burdened facilities. Rapid confirmation or rule-out has led to substantially improved management.

Long-term shedding of dermatophytes is uncommon. Cats with no skin lesions and a positive PCR test are likely to be so-called “fomite carriers”, having coat contamination from an environment containing spores. This is common in settings with low-level endemic infection. Fomite carriers are distinguished from true positives by the lack of skin lesions and rapid conversion to negative culture status. While semi-quantitative culture is used to distinguish fomite carriers from true positives,\(^10\) no reliable qPCR cut-off has been established. However, a high Ct value would be consistent with carrier status.

qPCR can detect RNA or DNA from non-viable pathogens. In early work on SARS-CoV-2, viral RNA was detected on a high proportion of surfaces in the vicinity of COVID-19 patients, but zero samples (of a total of 97) contained infectious titres of virus on tissue culture.\(^11\) In cats being treated for \textit{Microsporum canis} infection, qPCR remained positive long after fungal cultures were negative.\(^12\) This was most likely due to detection of non-viable fungi as well as possible contamination during sample collection. Ct values were generally unhelpful in predicting when cultures would be negative, other than that values ≥ 35.7 were associated with negative cultures. Prolonged positive qPCR results could be interpreted to mean that a cat is still infected and contagious. This could lead to unnecessarily prolonged treatment and isolation, extra clinical interventions and additional outbreak measures. For these reasons, qPCR is not recommended to determine mycological cure.
COVID-19 has substantially improved our understanding of the relationship between rapid antigen tests and qPCR (Figure 3). This relationship is very similar for parvo and panleukopenia. Positive rapid test results are reliable, but negative results may not be. This is because the antigen test requires high viral loads for a positive result. In both dogs and cats, parvoviral counts peak within several days of infection (typically 3-7).\(^5\) Antigen tests are positive in the early, most acute stages of infection, while qPCR typically remains positive for longer. The rapid test represents a “tip of the iceberg” phenomenon, and is positive at the peak of virus load and infectivity. The rapid test is recommended as the primary test for diagnosing parvo and panleukopenia infection, with qPCR only required under specific circumstances.

qPCR tests are increasingly offered as multiplex test panels, which simultaneously test for a number of pathogens.\(^13\) The advantage of this is time, cost, and avoidance of tunnel vision and confirmation bias. However, interpretation of test panels requires an understanding of the likelihood of asymptomatic shedding, the clinical significance of a positive test and the need for intervention. In some contexts, particularly in shelter settings, results may be uninterpretable. For example, feline upper respiratory pathogens were commonly detected in shelter cats without clinical signs of URI,\(^14\) and enteropathogens were frequently identified in feces from shelter dogs and cats both with or without diarrhea.\(^15,16\) Incorrect interpretation can lead to unnecessary interventions, additional testing, and barriers to adoption in a shelter setting. Clinical guidelines, providing clear case definitions and other guidance, would be of substantial benefit. Outcomes-based studies are also needed, as the benefits of assay panels in human medicine are not clearly established.\(^13\)

Not all pathogens cause disease, not all identified pathogens are the cause of disease in a given animal, and not all pathogens need to be treated. It is extremely important to be able to assess which is which, in the context of qPCR. This leads us back to first principles: assess the context, history, clinical presentation and consequences of action (or inaction), as well as the laboratory result. That result, however authoritative it may seem, is only one piece of information among many.

References


**Figure 1. Basics of PCR**

![Basics of PCR diagram](https://commons.wikimedia.org/wiki/File:Ygonaar_23_09_7_March_2006_(UTC).svg)
Figure 2. Cycle threshold values in PCR

https://www.albany.edu/rna/research-rna-institute/pooled-surveillance-testing

Figure 3. Positivity for rapid antigen test compared with PCR test, for SARS-CoV-2

https://www.nature.com/articles/d41586-021-00332-4
Canine heartworm: updates on diagnostics and treatment
Dr. Allison Collier, DVM, DVSc, residency trained in SAIM

There is a need for increasing knowledge on canine heartworm as heartworm cases are on the risk in North America. Heartworm disease can have numerous different consequences, including pulmonary endothelial damage, villous proliferation and attraction of leukocytes and platelets. Even dead and dying heartworms can induce damage, including thrombosis and villous inflammation. These consequences can potentially lead to different physiologic impacts, including the following:

- Reduced cardiac output
- Pulmonary hypertension
- Glomerulonephritis
- Caval syndrome

Given these potentially deleterious consequences, diagnosing canine heartworm is of utmost importance.

Diagnosis:
Different methods are available for diagnosis of canine heartworm disease, including antigen testing and microfilariae testing.

1) Antigen testing
Antigen tests detect a glycoprotein found mainly in the reproductive tract of the female worm. Only mature infections (>6 months) are generally detected. There are several things that won’t be detected with the antigen test, including:

- Low worm burdens (<2 adult females)
- Infections with only male worms
- Immature infections (<6 months of age)
- Antigen-antibody complexes

Additionally, due to the low prevalence of heartworm infection in many areas, all positive antigen tests should be confirmed prior to starting adulticide therapy in case of a false positive result. Confirmatory testing can be accomplished through:

- Identification of circulating microfilariae
- Attaining a positive result with a different type of antigen test
- Visualization of adult heartworms with ultrasound in the heart or pulmonary artery

Heat treatment of samples is sometimes advised prior to antigen testing. This can lead to increased sensitivity of the antigen test due to release of blocked antigen, although is not routinely advised to be performed. It however can be considered when microfilariae are identified or there is a high degree of suspicion of heartworm disease in a patient with a negative antigen test.
2) Microfilariae testing
Microfilariae testing should particularly be considered when there is a high degree of concern or suspicion for heartworm disease, as antigen-antibody complexes can occasionally lead to false negative results. Thereby, microfilariae testing helps confirm results, detect the patient as a reservoir of infection, and/or inform veterinarians of a high microfilariae burden in a patient. However, microfilariae are not present in all heartworm infected dogs.

Additional tests such as thoracic radiographs and echocardiography are sometimes performed to stage the severity of disease.

Treatment
Good candidates for heartworm treatment are those with confirmed infection, and those that have been stabilized prior to treatment. Treatment is advised as per the recommendations by the American Heartworm Society.

Adulticide therapy:
Melarsomine is the only adulticide therapy approved by the FDA. It has activity against immature (4 month old) to mature adult infections. The three dose protocol of melarsomine is recommended for patients with heartworm disease, except for those with caval syndrome.

The three dose protocol has been shown to kill 98% of worms, and has a decreased complication rate and increased safety as compared to other protocols.

Adjunctive treatments including corticosteroids and doxycycline are utilized throughout the protocol as well, as described below.

Doxycycline:
Doxycycline reduces Wolbachia in all stages of heartworm and has been shown to suppress microfilaremia. It is typically dosed at 10mg/kg BID for 4 weeks. There is then a recommended one month wait period after doxycycline treatment prior to treatment with melarsomine.
Steroids:
Steroids are administered as a 1 month tapering protocol following each melarsomine injection. They are thought to aid in controlling clinical signs of pulmonary thromboembolism.

Macro cyclic Lactones:
Macro cyclic lactones are potent activators of inhibitory receptors found in nematodes. The L3 and L4 stages are susceptible. They are administered monthly throughout the heartworm treatment protocol.

There is a period of time during which some stages of heartworm are not susceptible to macrocyclic lactones or melarsomine; this is known as the susceptibility gap. The protocol by the American Heartworm Society, and monthly administration of macrocyclic lactones helps eliminate the susceptibility gap as worms that have infected the dog in the past 2 months are eliminated, and those infected within the past 2-4 months are allowed to mature to a point that they are susceptible to treatment with melarsomine.

Risks
There are several risks present with treatment for heartworm infection. One of the main risks of treatment is pulmonary thromboembolism. Strict exercise restriction is advised to reduce the risk of thromboembolism.

Caval Syndrome:
Caval syndrome occurs when adult heartworms impede closure of the tricuspid valve. This thereby becomes an acutely life threatening condition involving right sided heart failure and hemolysis. Hemoglobinemia and hemoglobinuria can therefore result. Confirmation can occur via echocardiography, demonstrating heartworms in the tricuspid orifice. Treatment of caval syndrome should be prompt, as the condition is often fatal unless there is surgical extraction of the worms.

Post-treatment testing:
The most reliable method to confirm efficacy of adulticide treatment is with antigen testing. An antigen test is advised 9 months after the last melarsomine injection. A patient that is antigen positive prior to 9 months following the last melarsomine injection should be given more time to clear antigen prior to retreatment.

References:


The Gastrointestinal Microbiota, IBD, and Fecal Microbial Transplantation
Allison Collier, DVM, DVSc

Introduction

The gastrointestinal microbiota is defined as the diverse collection of bacteria, archaea, fungi, protozoa and viruses inhabiting the gastrointestinal tract\(^1\). The term microbiome includes the microbiota, as well as their genetic makeup.

The microbiota serves several essential functions in the body. It is believed to be essential to metabolism, gastrointestinal epithelial vitality, immunologic activity, defense against invading pathogens, nutrition, and other essential functions\(^1\)–\(^3\). As a result, the importance of the microbiota cannot be overstated.

Metabolites produced by the microbiota

Short chain fatty acids (SCFA):
Short chain fatty acids are end products of bacterial carbohydrate fermentation and are produced by certain bacteria in the gastrointestinal tract. Fecal SCFA have been shown to be lower in dogs with chronic enteropathies\(^4\). Short chain fatty acids themselves have several essential functions, including:

- Creating an acidic environment to prevent overgrowth of pathogenic bacteria
- Suppression macrophages, promoting Tregulatory cells
- Strengthen epithelial tight junctions and promote production of mucous layer

Secondary bile acids\(^5\):
The gastrointestinal microbiota is also involved with deconjugating/dehydroxylating primary to secondary bile acids. Bacteria such as *Clostridium hiranonis* are particularly important for this. The ratio of primary to secondary bile acids also has several important effects, including:

- Immune regulation
- Inhibition of germination of *C. difficile* spores
- Modulating glucose and insulin homeostasis

Dysbiosis:
Dysbiosis represents changes in the composition of the microbiome and is often characterized by a reduction in microbial species diversity\(^1\). Dysbiosis has been noted in various disease states, and even following certain medications.

Antibiotics:
Antibiotics have been shown in numerous studies to have an impact on the gastrointestinal microbiota. In a study investigating the impact of one week of tylosin treatment in dogs, tylosin-exposed dogs were noted to have decreased bacterial diversity characterized by decreases in anaerobes, with changes that did not consistently resolve following discontinuation of tylosin\(^6\). Additionally, dogs administered metronidazole have been shown to have altered bacterial
composition, with changes that did not fully resolve 4 weeks after administration\(^7,8\). Therefore, the impact of antibiotics on the microbiota cannot be overstated.

**Disease states- chronic enteropathies**

Dysbiosis has been associated with chronic enteropathies in numerous studies in both people and dogs. Common trends that are observed include increases in Proteobacteria, and decreases in Firmicutes, Bacteroidetes, and Fusobsacteria (in dogs)\(^3,9–12\). It is unknown whether these changes to the microbiota are cause or consequences of disease. However, in rodent models with IL-10 or IL-2 deletion, significant intestinal inflammation develops only in the presence of bacteria, not in germ free animals\(^10,13–15\).

**Inflammatory bowel disease and chronic enteropathies**

Inflammatory bowel disease represents one of the most frequent causes of chronic vomiting, anorexia and diarrhea in canine patients\(^16–19\). There are numerous factors suspected in the pathogenesis, as shown below:

![Figure 1: Factors involved in the pathogenesis of IBD, including the interactions of a genetic predisposition, dysbiosis, dysregulated immune response, and environmental factors such as dietary antigens.](image)

Inflammatory bowel disease (IBD) is diagnosed after exclusion of extra-gastrointestinal, infectious or parasitic disease, and intestinal disease of other causes (such as a gastrointestinal foreign body). It is also implies that trials with diet and deworming have failed, and that inflammation has been demonstrated histologically. In contrast, the term chronic enteropathy is utilized in animals where gastrointestinal biopsies have not been taken, and it does not imply which treatment will be needed to control the clinical signs\(^20\).
Despite treatment, many patients unfortunately continue to have clinical signs. A multimodal treatment of diet, immunomodulatory therapy, and treatments to address dysbiosis are often pursued, as demonstrated below.

**Figure 2:** Different aspects of treatment targets in IBD

There are different ways manipulation of the microbiota can be achieved, including through:
- Diet
- Probiotics/Prebiotics
- Antibiotics
- Fecal microbiota transplantation

**Fecal Microbiota Transplantation**

Fecal microbial transplantation (FMT) is defined as the ‘the transfer of feces from a healthy donor into the intestinal tract of a diseased recipient’\(^1\). One of the key goals of FMT is to restore diversity and microbial compositions comparable to that observed in healthy individuals\(^2\). There are numerous potential benefits to FMT, including (but not limited to)\(^2\):\(^3\):  
- Increase in richness of the microbiome  
- Re-establishing prominence of secondary bile acids in the feces  
- Restoring the integrity of the intestinal barrier  
- Transplantation of bacteriophages

Prior to performing FMT, several aspects should be considered, including:
- Recipient selection  
- Donor screening  
- FMT preparation  
- FMT administration

In selecting a patient to administer FMT, it may help in any patient with diarrhea, although a proper work-up is recommended to identify the underlying etiology, as otherwise the diarrhea may recur if the underlying cause is not addressed. FMT has been administered to patients with
acute diarrhea, as part of the treatment for IBD, in some infectious processes such as parvovirus infection, and even in some people with immune mediated disease\textsuperscript{24–27}.

Donor selection:
A properly selected and screened donor is an essential part of FMT. A donor for FMT should be healthy, fed a balanced (non-raw food diet), have no medical concerns or history of chronic gastrointestinal disease, and have not received antibiotics in the last 12 months. Criteria were recently outlined in a review of FMT in veterinary medicine\textsuperscript{23}. These patients should ideally have a normal CBC and biochemistry, have a negative fecal flotation, be negative for giardia oocysts (and consider performing a giardia ELISA), and should potentially be tested for fecal pathogens such as \textit{Salmonella} and \textit{Campylobacter}.

Preparation and Administration
Different methods of preparation and administration have been performed. In a recent review of FMT in veterinary medicine\textsuperscript{23}, it was recommended to use 2.5-5g feces/kg body weight of the recipient, suspend this in saline using a blender, sieve filter this solution, and then freeze the final product (at -20C or -80C). This solution can then be administered rectally via a 12-14Fr red rubber catheter into the colon, with sedation administered as needed based on patient comfort and tolerance. It is then recommended to not feed and to restrict activity for 4-6 hours following the FMT.

In a recent study using FMT in dogs with IBD as a preliminary study, a ratio of 1 part feces to 5 parts saline was blended, filtered, and stored in 60mL syringes at -20C for up to 3 months. This mixture was then administered at a dosage of 10mL/kg as a retention enema\textsuperscript{28}.

There are limited studies on the use of FMT in veterinary medicine. Thereby, further information is necessitated to better determine the optimal protocols, including whether to utilize fresh vs frozen feces, pooled vs non-pooled donor feces, the optimal method of administration, the optimal FMT recipient and donor, and whether to perform single vs multiple FMT infusions.


DIFFERENTIATING INFECTIOUS FROM NON-INFECTIOUS RESPIRATORY DISEASE IN THE CAT AND DOG

Jinelle A. Webb, DVM, MSc, DVSc, Diplomate ACVIM (Small Animal Internal Medicine)
VetLink Mobile Imaging, Oakville, Ontario
Tori Brown, DVM, Small Animal Internal Medicine Resident, VCA Canada Mississauga-Oakville Veterinary Emergency Hospital, Oakville, Ontario

Introduction

In cats and dogs from Ontario presenting with acute or chronic respiratory symptoms, there are an array of possible diagnoses. Infectious causes can include bacterial disease, viral disease, fungal / rickettsial disease, and parasitic disease, whereas non-infectious causes may include inflammatory or immune mediated disease, neoplastic disease, anatomic malformation, foreign body, smoke inhalation or aspiration of caustic material, cardiac disease, non cardiogenic pulmonary edema, trauma associated changes (pulmonary contusions, pneumothorax) and idiopathic disease. It can also be a challenge to determine if the disease affects the upper respiratory tract, lower respiratory tract including bronchial and interstitial locations, and pleural / mediastinal space, or a combination of these anatomic areas. The treatment and prognosis vary widely with the underlying disease process, making it important to pursue a definitive diagnosis. For infectious causes, this talk will focus on pathogens in Ontario.

Presentation

Cats and dogs with respiratory disease may present with coughing, dyspnea, tachypnea, sneezing, abnormal respiratory sounds, or abnormal respiratory patterns. Respiratory signs may be constant, or occur intermittently. Many cats, and some dogs, may present for feeling unwell, without obvious respiratory signs. Pets with nasal disease most commonly present with sneezing, nasal discharge, epistaxis, stertor, and increased nasal noise.

Although not always the case, loud or harsh expiratory noise is more commonly heard with lower respiratory tract disease, whereas loud or harsh inspiratory noise is heard more commonly with upper respiratory tract disease. Cases with pleural space disease may result in quieter lung sounds, depending on the amount of effusion (or air, in the case of pneumothorax) present. However, tachypnea may be noted. Tachypnea may also be noted in cases with significant interstitial or alveolar infiltrates. Coughing can be seen in both dogs and cats, but seems to be more common in dogs. In both cats and dogs, presence of a heart murmur may indicate the presence of heart disease leading to respiratory symptoms. However, pets with respiratory symptoms may have an unrelated, asymptomatic heart murmur. In addition, both cats and dogs may have symptomatic heart disease without a detectable heart murmur.

Infectious Causes

Bacterial

Common bacterial pneumonia (such as *Mycoplasma* spp., *Neisseria animaloris*, *Pasteurella* spp., *Bordetella bronchiseptica*, *Streptococcus* spp., *Mycobacterium* spp., *E. coli*, *Salmonella* spp., and *Yersinia pestis*)

Nocardiosis / Actinomycosis – pneumonia and pyothorax in dogs and cats, often associated with aspiration of plant material
Chlamydioidis – common cause of upper respiratory tract disease in cats

*Viral*

Canine infectious respiratory disease complex or kennel cough (canine adenovirus 2, canine parainfluenza virus, canine respiratory coronavirus, canine influenza virus, canine herpesvirus, canine distemper virus) – common cause of upper and lower respiratory disease in dogs

Feline upper respiratory tract disease (feline herpesvirus-1, feline calicivirus, rarely influenza virus, also includes chlamydioidis, *Mycoplasma* spp., *Bordetella bronchiseptica*, *Streptococcus equi subsp. zooepidemicus*)

Feline infectious peritonitis – uncommon cause of pleural effusion in cats

*Fungal / Rickettsial*

Blastomycosis – lower respiratory tract disease in dogs; extremely rare in cats

Histoplasmosis – lower respiratory tract disease in cats and dogs

Aspergillosis – nasal disease, occasionally involving lower respiratory tract or systemic spread; much more common in dogs than cats

Cryptococcus – nasal disease in cats, rarely involves hilar lymph nodes and pulmonary tissue

*Parasitic*

Lungworm (*Aelurostrongylus abstrusus, Eucoleus aerophilus, Crenosoma vulpis, Filaroides hirthi, Oslerus (Filaroides) osteri*)

Lung fluke (*Paragonimus kellicotti*)

Heartworm (*Dirofilaria immitis*)

Nasal mites (*Pneumonyssoides caninum*)

*Non-Infectious Causes*

Inflammatory / Immune Mediated Neoplastic

Cardiac Anatomic

Foreign Body Smoke Inhalation

Aspiration of Caustic Material Non Cardiogenic Pulmonary Edema

Trauma (Pneumothorax, Pulmonary Contusions) Idiopathic
Diagnostic Options

The differential diagnoses list will depend on the signalment, and also the history and physical examination. This will in turn dictate the recommended diagnostic steps. A thorough physical examination is imperative to help further evaluate the respiratory system. Some pets with significant respiratory compromise may decompensate with even a physical examination, therefore consider use of sedation and allowing the pet time with oxygen prior to performing diagnostics – and in some cases this may be needed even for a thorough physical examination.

Most dogs and cats presenting with lower respiratory signs will warrant thoracic radiographs. There are several different radiographic patterns that can be present, and some pets will have more than one pattern. A summary of the patterns is provided below.

Radiographic Appearance

<table>
<thead>
<tr>
<th>Location</th>
<th>Appearance</th>
<th>Common Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alveolar</td>
<td>Focal uniform soft tissue opacity, usually with distinct margins; air bronchograms</td>
<td>Aspiration pneumonia; bronchopneumonia; cardiogenic and noncardiogenic pulmonary edema; neoplasia; hemorrhage</td>
</tr>
<tr>
<td>Bronchial</td>
<td>Rings and lines through lung field, including periphery</td>
<td>Chronic bronchitis, asthma, eosinophilic bronchopneumopathy, heartworm disease</td>
</tr>
<tr>
<td>Interstitial—nodules or miliary pattern</td>
<td>Multiple diffuse small to variably sized nodules</td>
<td>Lymphoma, metastatic neoplasia, fungal disease, parasitic disease, eosinophilic bronchopneumopathy, pyogranulomatous pneumonias</td>
</tr>
<tr>
<td>Interstitial-unstructured</td>
<td>Diffuse increased opacity</td>
<td>Toxin exposure, lymphoma, edema, hemorrhage, numerous infectious etiologies</td>
</tr>
<tr>
<td>Vascular</td>
<td>Increased pulmonary arteries and/or veins</td>
<td>Heartworm disease, cor pulmonale, left-sided heart failure, PDA, VSD, ASD</td>
</tr>
</tbody>
</table>

Once radiographs are performed, the appearance may then lead to additional imaging modalities, infectious disease testing, or sampling of airways, lung parenchyma, pleural effusion, or masses.

Dogs and cats with a presentation and/or physical examination suggestive of tracheal or laryngeal disease may also warrant thoracic radiographs, with particular attention paid to the larynx and trachea. Fluoroscopy can be beneficial for assessing dynamic movement of the trachea in cases where tracheal collapse is suspected.

Additional Imaging Modalities

Thoracic ultrasound is indicated if there is the presence of pleural effusion, pericardial effusion, mediastinal mass, larger pulmonary masses, or thoracic wall masses. In addition, it can be used to assess for laryngeal or tracheal masses, and there are reports of utilization for assessing tracheal collapse. Ultrasound allows confirmation of fluid or masses, and also guided sampling, depending on the location and operator experience. Echocardiography is indicated if cardiac disease is suspected, and may be performed prior to thoracic radiographs in some cases.
CT scan has become more available in referral settings, and is indicated for some respiratory cases. CT scan removes the superimposition seen with traditional radiographs, and may allow guided sampling. CT scan does require heavy sedation or general anesthesia in most cases. CT scan is the primary imaging modality for nasal disease in dogs and cats. Nasal radiographs rarely provide enough information to warrant inclusion, although they can be diagnostic for metallic foreign objects.

Bronchoscopy and bronchoalveolar lavage are helpful for visualizing the trachea, and portions of the bronchi depending on pet size. It is useful for foreign body location and removal, obtaining airway samples, and potentially sampling airway masses. Given the potential for anesthetically unstable respiratory cases, these are typically performed in referral practice. Rhinoscopy has been almost entirely replaced by CT scan for nasal imaging, although it can be beneficial in some cases of suspected fungal rhinitis based on CT scan, to allow direct visualization and sampling of fungal plaques.

**Laboratory Testing for Respiratory Disease**

Cytology – if fluid or material is obtained, whether from airways, a mass or cystic structure, consolidated lung, or fluid from the pleural or pericardial space, cytology can be performed. This may reveal a diagnosis, such as with fungal disease, septic regions, parasitic disease, or neoplasia. In some cases, an infectious disease is confirmed, but additional testing may be required to determine the organism, such as the requirement of bacterial culture to determine the organism present. Cytologic results may be strongly supportive of a diagnosis, such as with eosinophilic bronchopneumopathy, asthma or bronchitis, and feline infectious peritonitis, but additional testing may be indicated to rule out other causes, or reach a definitive diagnosis. In many cases, cytology will reveal changes that can be consistent with several different disease states, such as a result of serosanguinous pleural effusion with no cause noted.

Histopathology – it is uncommon to obtain material for histopathology in dogs and cats with respiratory disease. Biopsies are commonly obtained from the nasal cavity, although they are more commonly diagnostic in neoplastic rhinitis rather than fungal rhinitis. Respiratory system masses (tracheal, laryngeal, mediastinal, pleural, pulmonary, cardiac, nasal) should be submitted for histopathology. If a consolidated lung lobe or abscess is removed, it should be submitted for infectious disease testing as well as histopathology.

Culture – as with cytology, if fluid or material is obtained from airways, a mass or cystic structure, consolidated lung, or pleural / pericardial fluid, culture can be performed. The clinician needs to have a suspicion of what type of infection may be present, in order to select an appropriate type of culture. Aerobic bacterial culture is most commonly selected, however some cases may warrant anaerobic bacterial culture in addition, and/or fungal culture. As commensal or opportunistic organisms can be present, caution should be taken with interpretation of an unexpected organism; for example, bacterial contamination from the oral cavity or skin is possible, depending on how the sample is obtained. It is possible to have a false negative culture, and some cultures can take a prolonged period to obtain results, such as with fungal cultures.

Parasitic analysis – larvae and parasitic eggs can be present in feces or in respiratory secretions, sometimes requiring a Baermann technique. It is important to remember that shedding is intermittent, and therefore a negative parasitic analysis does not rule out parasitic
disease. Some parasitic diseases have PCR testing available, which is useful in cases with a low worm burden.

Antigen and Antibody tests – There are several antigen and antibody tests available for respiratory infectious disease. The most commonly used outside of respiratory panels include:

- Heartworm antigen and antibody testing
- Urine immunoassay for *Blastomyces dermatitidis*
- Serologic testing for fungal disease such as *Blastomyces dermatitidis, Aspergillus* sp.

PCR testing and Respiratory Panels – PCR testing and respiratory panels that incorporate PCR testing and sometimes other forms of testing have become commonplace at veterinary laboratories. These panels can be very helpful for pets presenting with respiratory symptoms, as long as they are utilized appropriately. Some viruses can cause false positives results due to previous vaccination, and negative results do not rule out the infectious disease definitively. In addition, the presence of an infectious organism does not always mean that it is the cause of the symptoms. Therefore, results of PCR testing and panels must be taken in context with the patient’s signalment and presentation. For example, although upper respiratory disease in the cat is frequently due to viral disease, viral pneumonia is uncommon. Respiratory panels are very commonly utilised for suspected cases of feline upper respiratory tract disease and canine infectious respiratory disease complex.

Typical components of respiratory panels:
*Canine Infectious Respiratory Disease Complex:*
*Bordetella bronchiseptica, canine adenovirus type 2, canine distemper virus, canine herpesvirus type 1, canine parainfluenza virus, canine pneumovirus, canine respiratory coronavirus, H3N2 canine influenza virus, influenza type A virus (H1N1, H3N8), Mycoplasma cynos, Streptococcus equi* subsp. *zoopneumoniae*

*Feline Upper Respiratory Tract Disease:*
*Bordetella bronchiseptica, Chlamydia felis, feline calicivirus, feline herpesvirus type 1, influenza virus H7N2, influenza type A virus (H7N2, H3N2, H1N1, H3N8), Mycoplasma felis*

**Biomarkers**
Pro-BNP, Cardiac troponin I – used to evaluate for cardiac disease in dogs and cats

**Summary**

Cats and dogs can present with a variety of respiratory symptoms, and the presentation may be acute or chronic, and vary from mild to severe. The signalment, geography/travel history, history, and physical examination will suggest which diagnostic testing is indicated. In many cases, thoracic radiographs can provide basic information about respiratory changes, which may narrow the differential diagnoses list. A therapeutic trial may be indicated prior to reaching a definitive diagnosis. More critical cases may not have the ability to wait for diagnostic testing results, or may not be stable enough to perform some testing. There is a wide variety of testing available, and owner finances will often restrict the clinician’s ability to perform exhaustive testing. Knowledge about local infectious disease, and typical history and clinical signs associated with different causes of respiratory disease in the dog and cat, is imperative to aid in the diagnostic process.

References available from the authors upon request.
Equine Program
The Elite athletic horse: what makes a winner
Laurent Couetil, DVM, PhD, DACVIM(LAIM)
Purdue University College of Veterinary Medicine, West Lafayette, Indiana 47907

Investigation of the causes of decreased performance in athletic horses progressed due to the increasing use of high-speed treadmills and more recently, the availability of over ground endoscopy.

Factors involved in athletic performance

• Components:
  Athletic performance is dependent on multiple factors including physical aptitude, adequate energy production (ATP) and well trained musculoskeletal and cardiovascular systems. An additional factor, all too familiar to horsemen and difficult to control, is the mental factor or "will to win".

• Energy supply:
  Necessary energy is produced in 2 major ways:
  - Aerobic metabolism which requires oxygen as well as a "combustible" (i.e. glucose, glycogen, fat).
  - Anaerobic metabolism which only requires glycogen or glucose.

• Energy partitioning:
  The source of energy used depends on the intensity of exercise as well as on its duration. Anaerobic energy production is very rapid but does not last very long. Aerobic energy is produced at a slower pace, but yields 13 times more ATP and lasts as long as there is a combustible (oxygen and glycogen, fat). It is estimated that Quarter Horses running 1/4 mile races generate about 60% of their energy needs via anaerobic metabolism and the remainder via aerobic metabolism. In racehorses running a mile, 80% of energy needs are derived from aerobic metabolism.

• Aerobic energy delivery:
  Several factors are involved such as the oxygen transport chain, hemoglobin concentration, heart rate, stroke volume and oxygen extraction by muscles. The oxygen transport chain includes oxygen concentration in air, oxygen transport via conducting airways, oxygen diffusion across alveolar capillary barrier, oxygen binding to hemoglobin and distribution to muscle via circulation and finally oxygen diffusion from capillary to myocyte mitochondria.

Limitations to athletic performance

• Factors limiting performance:
  - In healthy horses: Various biomechanical problems and fatigue represent the major factors limiting athletic performance. In short duration, high intensity exercise (Quarter Horse, Thoroughbred, Standardbred), fatigue results from failure to produce energy. In long duration, low intensity exercise (endurance, 3D-event), lack of a combustible, excessive body temperature and dehydration are major limiting factors.
- Diseases: performance may be impaired by various problems such as musculoskeletal (lameness, tying up), respiratory (lung disease, upper airway obstruction), cardiovascular (arrhythmia, valvular disease), and neurological (EPM, wobbler) diseases.

• A retrospective study was performed on 163 horses presented for poor performance evaluation at Purdue’s Equine Sports Medicine Center. The breakdown per breed was: 52% Standardbreds, 27% Thoroughbreds, 18% Quarter Horses and 3% other breeds. The problems diagnosed among these 163 horses can be categorized as follows: respiratory disease 50%, lameness 35%, muscle problems 15%, and other disorders (cardiovascular, neurological, unknown origin) 22.5%. Approximately 20% of horses had a combination of problems, underlining the importance of performing comprehensive testing. Without it, these undetected problems are likely to hinder or shorten an athletic horse’s career.

Poor performance evaluation

• History:
  The horses’ history should establish the duration and severity of decreased performance. It should also help determine if the decreased performance is more likely due to respiratory, musculoskeletal or other type of problems. Previous health problems need to be investigated (exercise-induced pulmonary hemorrhage, respiratory infection, etc.).

• Clinical examination:
  Clinical investigation should focus on 3 major systems: respiratory, musculoskeletal and cardiovascular. In some cases, poor performance maybe secondary to neurologic disease such as wobblers, equine protozoal myeloencephalitis (EPM) and equine degenerative myelopathy (EDM).

  - Respiratory system
    a. inspection: nasal discharge (uni- or bilateral, sero-mucoid, mucopurulent); lymph nodes (submandibular, retropharyngeal, cranial cervical); cough; breathing pattern (e.g. excessive abdominal lift during expiration consistent with chronic obstructive pulmonary disease).
    b. palpation/percussion: lymph nodes; laryngeal muscles (e.g. crico-arytenoidus dorsalis atrophy in "roarers"); slap test; chest percussion.
    c. auscultation: bronchial sounds normally heard symmetrically; it is helpful to increase tidal volume using rebreathing bag or closing off nostrils for 1 minute in order to increase respiratory sound intensity; wheezes (continuous sounds) and crackles (non-continuous sounds) are abnormal respiratory sounds.

  - Musculoskeletal system
    a. inspection: look for symmetry of the musculature; limb conformation; hoof balance; shoeing; swelling or effusion.
    b. palpation: limbs; axial skeleton (dorsal spinal processes, dorsolumbar and lumbosacral muscles); joints; tendons; bones (e.g. splint bones, dorsal aspect of cannon bone); hoof tester.
    c. Lameness examination: walk, trot, (galop) on straight line and on circle; flexion tests.
- Cardiovascular system
  a. inspection: mucous membranes color and capillary refill time; jugular pulse (i.e. pulse wave should not go higher than lower third of the neck when head and neck are in normal position).
  b. palpation: pulse quality and frequency (e.g. facial artery).
  c. auscultation: horse's heart is large and beats slowly therefore in addition to S1 and S2, third (S3) and fourth (S4) heart sounds are often audible; cardiac rhythm (athletic horses often have second degree A-V block which disappears upon mild exercise); cardiac murmurs (high incidence of "innocent" or physiologic murmurs in athletic horses i.e. non-associated with valvular lesion).

- Neurological examination:
  Look for muscle atrophy; gait abnormalities should be graded on a scale of 0 to 4 in each of four categories: ataxia, paresis, hypometria, hypermetria; EPM can cause focal muscle atrophy and cause lameness.

Diagnostic tools
- Blood work: Hematology and serum biochemistry are part of the basic data that are needed. Anemia will impair oxygen transport and may cause decreased performance. Alteration in white blood cell count may indicate subclinical infections. Some serum enzymes are specific to certain tissues therefore, a significant increase these enzyme activities suggest tissue damage (e.g. CPK with muscle problems). Serum electrolytes, especially when compared with their level in urine (fractional clearances), can give indication about body status.

- Endoscopy: Endoscopy of the upper respiratory tract should always be part of poor performance evaluation. Abnormalities often occur during strenuous exercise and in these instances, dynamic endoscopy on a treadmill or over ground are recommended. Horses that exhibit abnormal respiratory noise during exercise, or that slow down abruptly toward the end of a race are good candidate for dynamic endoscopy.

- Cytology of the airways: Microscopic evaluation of BAL fluid correlates well with the type of lung problem (histopathology). Tracheal wash is a less sensitive and specific method to characterize lung problems, however it is the preferred method to collect bacteriological cultures.

- Imaging:
  - Radiography: poorly sensitive for the diagnosis of equine asthma or exercise-induced pulmonary hemorrhage (EIPH).
  - Ultrasonography: Used to image musculoskeletal and cardiovascular systems. To date, cardiac measurements have not been shown to predict horses' performance. Sensitive but not specific for the diagnosis of equine asthma or EIPH.
  - Nuclear scintigraphy: complicated lameness are best evaluated with the help of nuclear imaging.

- Exercise testing
  - Field testing: recently, several protocols have been published to perform standardized exercise test in racehorses and sports horses (Allen et al. 2016). The most important
data to be recorded during those tests are speed (using GPS technology), heart rate and blood lactate.

- Indications for treadmill exercise testing: to determine the causes of poor performance when no abnormalities are detected on physical examination or to evaluate the effect of abnormalities on performance (e.g. lameness, upper respiratory abnormality). Standardization of treadmill testing allows cross-sectional comparison between horses, as well as longitudinal comparison of a horse's progress after therapy. Athletic potential and fitness level can also be estimated by standardized treadmill testing.

- Treadmill acclimatization: About 30 minutes are necessary to acclimate a horse to mild exercise on a treadmill. The procedure typically requires 2 to 3 technicians. First, the horse needs to be equipped with appropriate gears and limb protection. Then, the horse is acclimated to the surroundings and the noise made by cooling fans and the treadmill in motion. The horse is walked a few times on the treadmill without the belt running. Finally, the treadmill is started while the horse is on it. Ideally, two technicians, one positioned on each side of the treadmill, hold the horse, with a thick cotton lead rope preferably, while a third technician stands behind the horse with a whip to encourage forward motion. Most horses will require at least 2 acclimatization runs before exercise testing.

- Protocols: Warm up takes place before any treadmill testing. The protocol used at Purdue’s Equine Sports Medicine Center (ESMC) consists of 4 minutes at a walk (2 m/s) and 4 minutes at a trot (4 m/s). Various incremental step test protocols have been used by researchers and clinicians. Most protocols are based on 1-minute increments starting exercise at a speed of approximately 4 m/s and increasing the speed until the horse can no longer keep pace with the treadmill despite encouragement to run. The protocol used at Purdue’s ESMC consists of an initial 2 minute step at 4 m/s followed by 1-minute steps at 6, 8, 10, 11 and 12 m/s. Treadmill incline is 5% for standardbreds and 10% for thoroughbreds. The slope allows horses to work harder but at a slower speed, therefore decreasing risks of injury.

• Measurements taken during exercise testing
  - Heart rate: Heart rate monitoring consists of 2 contact electrodes positioned on the left side of the chest, one at heart base level (point of shoulder) and a second on the dorsal third of the thorax. Electrodes are connected to a wireless transmitter attached to the horse's girth or harness. The receiver, fixed on the treadmill rail, transmits a signal to the computer and wall display panel. Heart rate (HR) monitoring gives an estimate of exercise intensity and cardiac output. Horses exercising at maximal intensity exhibit HR between 210-240 beats/minute. A horse's speed when HR is 200 beats/minute (V200) represents a useful parameter when comparing progress over time. The speed at which HR is at its maximum increases with fitness level (VHRmax).

  - Blood/plasma lactate: Arterial or venous blood is collected via a catheter placed into transverse facial artery or jugular vein. Blood samples are collected during the last 15 seconds of each incremental step while the horse is running on the treadmill. If blood will be processed rapidly after collection, heparinized samples can be kept on ice for up to 30 minutes without affected measurements. Otherwise, blood should be collected in tubes containing
fluoride/oxalate as an anticoagulant (gray top). Lactate is produced by exercising muscles during anaerobic metabolism. A horse's speed relative to a plasma lactate level of 4 mmol/L (V_LA4) is a reflection of fitness and exercise capacity.

- Blood gases: Arterial blood is collected via catheter placed into the transverse facial artery or carotid artery. Heparinized blood is immediately placed on ice and samples should be analyzed within 15 minutes of collection. Blood temperature is recorded for each sample via thermocouple probe placed in the arterial catheter which allows for correction of blood gas values which are determined at 37°C in the blood gas machine. Horse's blood temperature can reach 42°C during strenuous exercise, corresponding to a correction of more than 10 torr (positive correction for PaO2, negative correction for PaCO2). A study conducted at Purdue showed that measurement of blood gases constitute a sensitive way to detect lower airway disease in racehorses (Couetil and DeNicola, 1999). The study included 36 horses (20 Standardbreds and 16 Thoroughbreds) including 10 controls, 13 with SAID, and 13 with EIPH. The results of the study showed that horses with lung disease (EIPH or SAID) were significantly more hypoxemic than healthy control horses during a standardized treadmill test. Such lung diseases often go undetected for some time because the only manifestation is decreased level of performance.

- Dynamic endoscopy: A one-meter flexible video endoscope is inserted through the nasal passage into the nasopharynx and secured in place with velcro. Nasopharyngeal and laryngeal movement are recorded to allow later play-back of images in slow motion. Dynamic endoscopy is a useful test to perform if there is a suspicion of upper-airway obstruction such as laryngeal hemiparesis, soft palate displacement and epiglottic entrapment. This test can now be done in the field using “over ground” endoscope systems such as Advanced Monitors, Optomed, and Fritz.

Reference:
Role of imaging in diagnosing respiratory disease

- **Endoscopy of the respiratory tract**

  o Nasal passages: The nares consist of the nasal opening, alar folds, nasal diverticulum and the rostral aspect of the nasal septum. Nasal cavities are lined with a richly vascularized mucosa to allow warming and humidification of inhaled air. Vasomotor tone is under control of the autonomic nervous system with excitation of sympathetic and parasympathetic nerves leading to nasal vasoconstriction and vasodilation, respectively. Exercise is associated with sympathetic excitation and increased nasal volume. Despite these mechanisms designed to increase airway patency during exercise, the nasal passages still represent one of the bottleneck regions of the equine respiratory tract. Narrowing of the nasal passages secondary to sinus disease is difficult to appreciate by endoscopy. Other imaging modalities, in particular CT, are best to assess nasal passages and sinuses. The nasomaxillary opening is the communication between the nasal passage and the maxillary sinus. It is located in the middle meatus in the area of the ethmoid turbinate and it can be visualized with the endoscope.

  o Nasopharynx: The nasopharynx contains the guttural pouch openings, dorsal pharyngeal recess, soft palate, palatopharyngeal arch, larynx, and esophageal opening. In young horses the lymphoid tissue is active and can lead to the visual appearance of pharyngeal lymphoid hyperplasia. Pharyngeal lymphoid hyperplasia may be graded from 1 to 4.

  o Guttural pouches: The guttural pouch (GP) is a diverticulum of the eustachian tube. The guttural pouches are paired structures divided by a medial septum. Each pouch has a capacity of approximately 300 ml, although each pouch can accommodate up to 1 liter of fluid due to the distensible nature of the walls. The GP communicates with the pharynx through the pharyngeal orifice. A fibrocartilagenous flap closes over the orifice. The stylohyoid bone separates the interior of each pouch into the medial and lateral compartments. The medial compartment is approximately 2 times larger than the lateral compartment. The medial compartment contains the internal carotid artery, cranial cervical ganglion, cervical sympathetic trunk, vagus nerve (CN X), glossopharyngeal nerve (CN IX), hypoglossal nerve (CN XII), and spinal accessory nerve (CN XI) along the roof of the guttural pouch and traversing the caudal wall. The lateral compartment contains the external carotid artery along the ventral surface where it is in close association with the glossopharyngeal and hypoglossal nerves. Dorsally the external carotid artery continues as the maxillary artery. Also within this compartment is the maxillary vein, chorda tympani nerve, and branches of the mandibular nerve. The facial nerve (CN VII) courses over the caudal dorsal aspect.

**Figure 1**: Endoscopic view of the medial compartment of the right guttural pouch. Maxillary artery (MA), stylohyoid bone (SB), Internal Carotid artery (ICA), Glossopharyngeal and Hypoglossal nerves (white arrows), and longus capitus muscle (LC).
Soft palate: The soft palate begins rostrally at its intersection with the hard palate and ends caudally as palatopharyngeal arches which encircle the larynx. The soft palate at this level has a “button hole” appearance with the larynx acting as the “button”. Normally the soft palate rests beneath the epiglottis. This anatomical feature makes the horse an obligate nose breather.

Larynx: The larynx consists of four cartilaginous structures, the epiglottic, thyroid, cricoid, and paired arytenoid cartilages. The epiglottis is attached to the arytenoid cartilages by the aryepiglottic fold. Normally, the epiglottis rests dorsal to the soft palate except during deglutition when it covers the rima glottidis. The vocal cord is a fibroelastic structure which originates on the ventral surface of the larynx and inserts on the vocal process of the arytenoid cartilage. Axial to the vocal cord is the laryngeal ventricle.

Figure 2: Endoscopic view of a normal larynx at rest. Right (R), Left (L), Corniculate processes of the arytenoid cartilages (C), Aryepiglottic fold (AEF), Laryngeal ventricle (LV), vocal process of left arytenoid cartilage (large white arrow), and vocal cord (small white arrow).
Dynamic endoscopy:

Cervical trachea: The trachea is a flexible, non-collapsing tube which extends 70-80 cm (45-55 cm in ponies) from the cricoid cartilage of the larynx to the hilus of the lung. The trachea spans the distance from the first or second cervical vertebrae to the sixth intercostal space. Distally the trachea bifurcates into right and left principal bronchi. The cervical portion of the trachea extends from the larynx to the thoracic inlet and the thoracic portion extends from the thoracic inlet to the tracheal bifurcation. The thoracic portion of the trachea wall is subjected to pleural pressure but the lumen is near atmospheric pressure. Proximally, the trachea is superficial and is covered ventrally by the cutaneous colli and sternothyrohyoideus muscles. Distally, towards the thoracic inlet, the trachea is covered ventrally by the sternoccephalicus muscle.

The horse’s trachea has 48-60 concentric, incomplete hyaline cartilage rings. The cartilaginous rings are rigid and enclosed in a fibrous membrane which prevents collapse, and overlap dorsally to provide flexibility. Because these rings overlap dorsally, the trachea can be collapsed laterally by manual pressure or pressure caused by enlarged external structures adjacent to the trachea. Dorsally the trachealis muscle is attached to the inner surface of the tracheal rings and helps to maintain the patency of the tracheal lumen. The tracheal lumen is lined by a mucous membrane consisting of ciliated pseudostratified columnar epithelial cells and mucus-producing goblet cells. A blanket of translucent mucus coats the respiratory mucosa giving it a shiny, wet appearance (Fig. 3). The mucus layer aids in removal of particulate foreign material from the respiratory tract. The submucosa, deep to the mucous membrane lining, contains elastic tissue, which adds to the flexibility of the trachea.

Figure 3: Endoscopic view of the trachea in a healthy horse.
Intrathoracic airways: The ventral aspect is the trachea is lowest where it enters the thoracic inlet (Fig. 4A). As a result, excess mucous tends to pool at this level and fluid infused during tracheal wash will be easily aspirated by advancing the catheter tip in the tracheal puddle (Fig. 4B). The intrathoracic trachea is usually narrower proximal to the carina due to the aorta pushing the left side of the tracheal wall inwards. The trachea ends above the heart base at the level of the left atrium slightly right of midline. The trachea then gives way to the left and right principal bronchi at the carina (Fig. 5). After the carina, airways are divided into lobar, segmental and sub-segmental bronchi, bronchioles, and finally terminal bronchioles (Fig. 6). The right cranial lobar bronchus originates from the right principal bronchus at approximately 10 o’clock position (Fig. 5). The accessory lobar bronchus originates from the right principal bronchus at the 4 o’clock position next to the first bronchus of the right caudal lung lobe at 7 o’clock. The right principal bronchus then becomes the right caudal lobar bronchus. The left principal bronchus becomes the left caudal lobar bronchus after supplying the left cranial lobar bronchus at 2 o’clock (Fig. 5).

Figure 4: the white arrow indicates the lowest point of the trachea where mucus tends to accumulate (A). Endoscopy view of the lower third of the trachea with muco-purulent secretions pooling at the thoracic inlet (2B)
Figure 5: Endoscopic view of the carina. AccLB: accessory lobe bronchus; LCaLB1: first left caudal lobar bronchus; LCRLB: left cranial lobar bronchus; RCaLB1: first right caudal lobar bronchus; RCRLB: right cranial lobar bronchus

Figure 6: Ventral view of the horse trachea-bronchial tree (adapted from Nakakuki et al. 1993).
**Ultrasonography of the thorax:**
The procedure is performed with transducers ranging from in frequency from 4-10 MHz depending on the depth and resolution needed. Higher frequency (>7 MHz) will provide better details of the chest wall and lung surface. It is helpful to follow the same scanning protocol for all examinations in order to be thorough. This clinician scans the chest from caudal to cranial and dorsal to ventral starting at the most dorsal aspect of the last intercostal space (ICS17) sliding the probe longitudinally and ventrally between ribs. It is essential to prepare the skin and hair coat to maximize contact and conduction of the ultrasonic waves. For most horses, soaking the hair with isopropyl alcohol immediately prior to placing the probe is adequate. Horses with thick haircoat or obese horses will benefit from clipping the chest prior to using alcohol or coupling gel. Both sides of the chest should be scanned from ICS17 to ICS3. Cranial lung may be visualized cranial to the heart by placing the probe over the triceps muscle.

The lungs of the horse are divided into cranial and diaphragmatic lobes and in addition, there is an accessory lobe on the right side located ventrally and medially to the diaphragmatic lobe (Fig. 6). The heart can be visualized on ultrasonography on both left (ICS 3-6) and right (ICS 3-4) side and the level of the cardiac notch separating cranial and diaphragmatic lobes. The lung interface between pleural surface and aerated lung can be seen as a bright hyperechoic (i.e. white), horizontal line. The echogenicity is due to ultrasonic waves being reflected from the gas interface. This hyperechoic line is accompanied by additional hyperechoic parallel and equidistant lines located deeper to the surface that are artifacts of reverberation of the first interface (“A-lines”). During each tidal breath, the lung surface can be seen gliding along the chest wall. If the line is not seen gliding with each breath, it is likely that the horse has a pneumothorax. Another common finding on chest ultrasonography are comet-tail artifacts (“B-lines”) also called ring-down artefact that are due to change in acoustic properties on the lung surface (Fig. 7A). These artifacts originate from the pleural surface, have a strong ray-like appearance and move with each lung movement. Comet tail artefacts may be seen in normal lungs (usually few B-lines per field of view), especially in the dependent parts and often change with position of the horse (or foal). Pathologic ring-down artifacts result from fluid and inflammatory infiltrates causing a change in lung echogenicity resulting in a cluster of B-lines (Fig. 7B). Pleural effusion may be detected as hypoechoic (dark) fluid between chest wall and lung. Atelectasis and lung consolidation over the lung periphery are seen as hypoechoic areas next to hyperechoic aerated lung.

**Figure 7:** Thoracic ultrasound examination showing common artifacts. A) Ring-down artifact in a healthy horse (arrows). Normal lung interface is seem a horizontal hyperechoic line (white line; arrow heads) immediately below the chest wall (CW); B) Multiple ring-down artifacts in a horse with pneumonia.
How to maximize yield from respiratory samples

- When to choose TW or BAL?

Mucociliary clearance constantly moves mucus, cells and potential contaminants from peripheral lung regions towards the carina and up the trachea before being expectorated. Secretions are usually collected by tracheal wash (TW) or by bronchoalveolar lavage (BAL). Cytological and microbiological evaluation of respiratory samples provides invaluable information concerning the disease process. In cases of infectious lung disease (e.g. bacterial pneumonia), affected areas are often adjacent to unaffected areas. Secretions originating from affected lungs segments will eventually collect in the trachea. In these cases, cytologic examination and microbiological culture of TW fluid is likely to yield an etiologic diagnosis. In contrast, fluid collected by BAL is only representative of the lung region distal to the bronchus where the tube or endoscope was wedged. Infectious lung diseases such as pneumonia lead to focal or multifocal disease usually affecting cranio-ventral lung regions. Even with endoscopic guidance it is difficult to follow airways down to an affected lung segment. As a result, it is common to harvest cytologically normal BAL samples from horses with pulmonary infection. Yet, in cases of diffuse lung disease (e.g. recurrent airway obstruction, inflammatory airway disease) BAL fluid cytology correlates well with histology whereas TW cytology does not. This is probably due to contamination of the trachea by inhaled materials or nasopharyngeal secretions.

In general, TW is the preferred method for suspected localized infectious cases and BAL is preferred to characterize diffuse lung disease. In cases of uncertain etiology, it is best to perform both tests in order to maximize diagnostic yield. If both diagnostic techniques are used TW is performed first to avoid cross contamination between procedures. BAL fluid should be kept cool (on ice or in refrigerator) until processing to maintain cell morphology. Placing BALF in an EDTA tube will help prevent cell clumping. Do not add formalin or alcohol as it changes cell morphology dramatically. BALF should be processed within 24-48 h and the sooner the better. If delay is expecting in BALF processing, add serum from the horse (~ 2% or 0.2 ml to 10 ml in EDTA tube).

- Tracheal wash cytology:

Normal TW fluid cytology should contain scant amount of mucus, a predominance of pulmonary alveolar macrophages (PAMs; 40-80%) and epithelial cells with (20-50%) with lower numbers of neutrophils (< 20%), lymphocytes (< 10%), and eosinophils (<1%). Mast cells are typically not detected in TW cytology.

Cytology specimen demonstrating predominance of neutrophils with various degrees of degenerative changes (karyolysis and cytoplasmic vacuolation) and presence of intracellular or extracellular bacteria are consistent with infection and should prompt microbiologic culture of the fluid. Coughing during TW procedure may lead to oropharyngeal contamination recognized by presence of squamous epithelial cells laden with bacteria. Improper handling of TW fluid may also lead to degenerative changes of neutrophils in cases of non-infectious purulent
inflammation. The absence of bacteria or degenerative neutrophils does not rule out the possibility of an infectious disease. Aerobic and anaerobic cultures are recommended in febrile horses or in animals with a history suggestive of infectious disease. A Gram stain may help guide antibiotic therapy while waiting for culture results. Culture of TW fluid from infectious cases resulting in no growth may be due to an insufficient number of bacteria, prior antimicrobial therapy, inappropriate culture medium (e.g. mycoplasma and fungal infections), or viral infection without secondary bacterial infection. Alternatively, a bacterial isolate should be considered significant if it is a known pathogen, fluid cytology is consistent with infection and clinical examination is suggestive of an infectious process.

- **Bronchoalveolar lavage (BAL):**
  Normal BAL fluid cytology should have mainly lymphocytes (20-40 %) and pulmonary alveolar macrophages (40-70 %), with < 5 % neutrophils, < 2 % mast cells and < 1 % eosinophils.

A volume of 250-500 ml of warm sterile saline solution is infused (in one or two boluses) under pressure followed by immediate but gentle aspiration of the fluid using 60-ml syringes or a suction pump. It is important to use always the same technique because the volume of fluid used as well as the number of boluses administered have a significant effect of cell count and differential. Infusion of at least 250 ml is required to collect an appropriate BAL sample. On average, 50-70% of the infused volume can be aspirated. Smaller volumes are retrieved from horses with collapsible airways such as moderate to severe asthma. Fluid samples should be processed within 1 to 2 hours or stored on ice or at 4°C if sample shipping to the laboratory is to be delayed. Normal BAL fluid should appear slightly turbid with a layer of white foam on the surface (Fig. 8) and contain <400 cells/µl.

**Figure 8:** Appearance of normal BAL fluid.

- **Airway and lung biopsy:**
  Collection of tissue samples from the respiratory tract is a valuable diagnostic and prognostic tool allowing histopathology but also more advanced testing such as immunohistochemistry. The least invasive technique consists in endobronchial sampling of airway tissue under bronchoscopic guidance. The technique is safe but only small and superficial layers of airway mucosa are sampled. Parenchymal tissue may be obtained using closed or open lung biopsy techniques. Closed techniques include percutaneous and thoracoscopic lung biopsy.

  - Endobronchial biopsy
Transendoscopic biopsy of the airway may be done from nasal passages to peripheral airways as small as 2 mm in diameter (Fig. 4d-1). The forceps used may be cupped, fenestrated or alligator type. In general, larger forceps allow larger amount of tissue to be collected and provide better chances of obtaining diagnostic samples. Alligator forceps usually sample more tissue than cupped forceps. The horse should be sedated and restrained like for any endoscopy. Horses with increased breathing efforts due to bronchoconstriction such as in heaves should be premedicated with a bronchodilator (e.g. 500 µg of inhaled albuterol). Nasal oxygen supplementation (5-8 l/min) should be considered in horses with increased breathing efforts associated with hypoxemia. Topical anesthesia is recommended when sampling upper airways or to decrease coughing when sampling lower airways. A solution of diluted lidocaine (1 vol. of 2% lidocaine / 6 vol. sterile saline) is prepared in a 20 ml syringe and sprayed on the airway surface either directly through the endoscope working channel or via a catheter.

In the lungs, samples are taken at airway bifurcation from the main carina to subsegmental airways. A minimum of 5 samples is recommended and up to 20 bronchial biopsies can be obtained safely from one adult horse.

- Percutaneous lung biopsy
The procedure is performed with the horse restrained in stocks and sedated. A 5x5 cm of skin is clipped between the 7th-10th intercostal spaces, approximately 8-10 cm above the shoulder joint or over any lung area identified for sampling by ultrasonography or radiography. The skin is aseptically prepared and subcutaneous tissue and intercostal muscles are infiltrated with 20 ml of 2% lidocaine hydrochloride followed by a surgical scrub. Spring-loaded biopsy needles (14g x 20 cm, Temno, products group international, Lyons, Colorado) are preferred to Tru-Cut biopsy needles because they result in fewer complications (airway bleeding, hematoma). The biopsy needle is carefully inserted through a small skin incision just cranial to the rib and advanced through the chest wall (Fig. 4d-2). The needle is then inserted rapidly into the lung periphery at the end of inspiration and retrieved immediately after triggering sample collection. Multiple biopsies (2-4) should be taken to improve diagnostic value since samples are small. Lung tissue should be carefully lifted from the biopsy needle using a sterile needle and placed in 10% neutral buffered formalin. A tissue sample may be saved for microbiologic culture if indicated. Percutaneous lung biopsy is a relatively safe diagnostic method. However, significant complications may occur and warrant warning clients, including the possibility of death from hemorrhage or pneumothorax in approximately 3% of cases. Other complications such as coughing, airway bleeding, epistaxis, and hematoma at the biopsy site are common but usually self limiting. Some horses receiving insufficient local anesthesia may show signs related to pain from the biopsy site such as depression with intermittent signs of mild colic, collapse inside the stock or when walking out, or ataxia.

- Thoracocentesis
Thoracocentesis is performed for diagnostic and therapeutic purposes in cases of pleural effusion. The procedure is easily done on standing horses restrained in stocks and with sedation if appropriate. Ideally, thoracic ultrasonography will be conducted to identify the extent of pleural effusion and important landmarks such as heart and lung in order to optimize positioning of the thoracocentesis needle. Alternatively, the extent of the fluid line may be determined by auscultation and percussion. The equine mediastinum is fenestrated cranio-ventrally allowing fluid to migrate from one side to another. However, communications between both hemithoraces
are often sealed in inflammatory processes and collection of pleural fluid from both left and right side is recommended to maximize diagnostic ability.

The puncture site on the right side is located in the 6th – 7th intercostal spaces and approximately one hand width (10 cm) above the olecranon (Fig. 9). The site on the left is in 6th – 9th intercostal spaces and 5 cm above the olecranon. The site is clipped and surgically prepared. The subcutaneous tissue and intercostal muscle are infiltrated with 5-10 ml of 2% lidocaine solution while carefully avoiding damage to blood vessels and nerve running immediately caudal to each rib. Infiltration with lidocaine should also extend caudally over the adjacent rib over 2-3 cm. A stab incision is then made 2-3 cm caudal to the intercostal space. A sterile teat cannula is preferred for thoracocentesis because the blunt tip will avoid lung laceration. The cannula is first connected to extension tubing and three-way stop cock in closed position. The cannula is then carefully inserted through a small stab incision at a shallow angle while pointing the tip cranially and advancing under the skin until reaching the cranial aspect of the rib. This gap between skin incision and point of entry in the pleural space will prevent pneumothorax upon removal of the cannula. The needle is then rotated perpendicularly to the chest wall and pushed through the chest wall. Passage into the pleural space will be felt as sudden loss of resistance to advancement of the cannula. At that point, the three-way stop cock is opened to allow drainage of pleural fluid. If fluid doesn’t drain spontaneously, gentle suction may be applied with a syringe. Aliquots of the fluid are placed in EDTA tube (2 ml) for cytology and dry sterile tube for aerobic and anaerobic bacterial culture. Ultrasound guidance may be useful if fluid doesn’t drain readily.

**Figure 9**: Location for thoracocentesis on the right side of the thorax.
Laurent Couetil, DVM, PhD, DACVIM(LAIM)
Purdue University College of Veterinary Medicine, West Lafayette, Indiana 47907

**Latest information on causes and diagnosis of equine asthma**

Recurrent airway obstruction (RAO), also known as heaves and inflammatory airway disease (IAD) are the most common chronic inflammatory airway diseases of horses. Although clinical manifestation is usually different between RAO and IAD there are similarities in etiology, pathophysiology and therapy. Furthermore, because of similarities between these conditions and human asthma, the term **equine asthma** has been coined recently to describe horses with IAD (i.e. mild-moderate asthma) to RAO (i.e. severe asthma).\(^1\) Differentiating moderate to severe from mild equine asthma and infectious respiratory diseases may also be challenging in some cases. Criteria have been proposed to define each disease based on historical, clinical and functional tests. The purpose of this presentation is to review the current definition and understanding of equine asthma etiology and risk factors. The goal is to provide equine practitioners with evidence-based information to enable them to diagnosis equine asthma in the field and understand the main risk factors.

**Terminology:**
Respiratory disease terminology is confusing in the literature. Earlier studies have used the term COPD to describe horses that were affected by heaves or RAO but subsequent works have applied the term COPD to young racehorses that clearly were affected by IAD and not RAO while others have used COPD for horses affected by either IAD or RAO or summer pasture-associated RAO. Some older publications refer to emphysema when describing horses with the heaves phenotype because lungs do not collapse at necropsy. Lung emphysema may be present in a small number of chronically affected RAO horses however, clinical signs are still reversible in these animals. Lungs fail to collapse at necropsy because of air trapped behind obstructed airways and not because of true alveolar emphysema. Therefore, emphysematous lesions are not the main cause of clinical signs in RAO-affected horses.

Human asthmatics typically have a history of chronic, waxing, and waning respiratory symptoms, such as coughing and difficulty breathing that are associated with chronic airway inflammation and expiratory airflow limitation of variable severity. RAO (heaves) and IAD represent a range of chronic inflammatory disease of the airways in horses resembling human asthma in many respects including pathophysiology and therapy. In addition, horse owners understand that human asthma is an allergic disease caused by exposure to airborne particulates and have a better understanding of management and therapy recommendations for the management of affected horses when told it is equivalent to asthma in people. Therefore, the term equine asthma is now recommended to describe horses with mild (IAD) to severe (RAO) airway disease. The term equine asthma will be used in the remaining of the presentation.

**Disease definition:**
Early efforts led to the recommendation of diagnostic criteria for heaves and standardization of bronchoalveolar lavage (BAL) techniques.\(^2\) The purpose was to encourage researchers to use common criteria and techniques when conducting investigations in order to allow comparison between studies and also help practitioners manage affected horses based on scientific evidence. More recently, diagnostic criteria were refined to include mild to moderate asthma (IAD).\(^1\)
Horses with mild to moderate asthma are typically young, although the condition can be seen in horses of any age presenting with a complaint of chronic (> 3 weeks), occasional coughing and poor performance but are otherwise healthy (i.e. no fever or other signs of systemic illness). Because there are many potential causes of poor performance, it is important to rule out other respiratory or non-respiratory causes. Diagnosis may be confirmed by documented evidence of increased mucus upon trachea-bronchial endoscopy. This is best accomplished by examining the horses approximately 1 hour following exercise and mucus scores of 2 out of 5 or greater in racehorses and 3 or greater in sports horses have been associated with decreased performance. The most effective way to confirm a diagnosis mild asthma is by performing a BAL and documenting increased proportions of neutrophils (>5%), mast cells (>2%), eosinophils (>1%) or any combination thereof.

Severe asthma affects horses older than 7 years of age and is associated with exposure to airborne dust rich in molds and endotoxins. Such exposure is usually the result of feeding moldy hay. Some horses present identical clinical signs while being on pasture during the summer. This syndrome is called severe, equine summer asthma (summer pasture-associated RAO) and is indistinguishable from severe asthma except for its etiology. Finally, a small subset of horses is affected by both forms of severe asthma. Clinical signs of severe asthma are characterized by cough, increased respiratory efforts, increased mucus in airways and exercise intolerance. These signs are due to lower airway inflammation and obstruction as a result of bronchospasm, mucus plugging of small airways and airway hyperresponsiveness. During disease exacerbation, horses with severe asthma exhibit neutrophilic airway inflammation based on BAL fluid cytology (> 25% neutrophils) and increased breathing efforts resulting in maximum changes in transpulmonary pressure (∆Pmax) > 15 cmH2O during lung function testing. The latter can be rapidly improved by administration of bronchodilators such as Buscopan® IV or inhaled albuterol. Long term clinical improvement occurs after decreasing dust exposure (e.g. pasture) or providing therapy with corticosteroids and bronchodilators.

Causes of equine asthma:

Mild-moderate equine asthma:
Proposed causes of mild asthma include inhaled environmental particles, noxious gases and pollutants, and infectious agents (bacteria, viruses) with a modulatory role played by factors such as the horse’s immune response and genetic make-up. There is strong evidence to support the role of exposure to dust and molds in the pathogenesis of mild asthma. Healthy yearlings fed hay demonstrate increased BAL fluid neutrophil count and percentage, and more severe airway inflammation when housed in a stable than when kept on pasture. Exposure of healthy horses to moldy hay or endotoxins results in BAL fluid neutrophilia and airway hyperresponsiveness. Also, horses in training kept on straw bedding experience episodes of mild asthma that last longer than in horses bedded on shredded paper. More recently, the importance of small particulate (respirable dust) exposure was demonstrated in racehorses showing elevation in BALF inflammatory cells in response to increasing exposure (eosinophils in 1-2 years old; neutrophils or mast cells is 3 year old and older). Exposure to atmospheric pollutants such as ozone, nitrogen dioxide, carbon monoxide, sulphur dioxide in addition to noxious gases present in stables such as ammonia, hydrogen sulfide and methane have the potential to contribute to airway inflammation in horses. Many of these compounds have been shown to cause airway disease in humans and animals but data in horses are scarce.
The role of bacterial infection is controversial. Tracheal inflammation and infection is prevalent in horses, in particular racehorses and the likelihood of isolating bacteria from tracheal wash samples collected from racehorses in training is strongly associated with inflammation severity. However, bacteria may in fact be present in the trachea because of contamination during sampling or represent transient colonization of the proximal airways. No bacteria are cultured from tracheal wash samples in up to 54% of horses with mild asthma.\textsuperscript{10} Also, the trachea is not a sterile environment as recent evidence suggest that horses with mild asthma exhibit a unique microbiota that may be influenced by corticosteroid therapy.\textsuperscript{11} In racehorses, tracheal inflammation is associated with cough however, it is not associated with decreased performance.\textsuperscript{3}

Respiratory viruses do not appear to play an important role in mild asthma. Several reports have shown no evidence of viral infections based on serology or virus isolation aimed at detecting equine herpes, influenza, adenovirus, and rhinoviruses. Using detection of viral particles by quantitative PCR, association of mild asthma in racehorses with equine herpesvirus type-2 and equine rhinitis virus type B as been reported.\textsuperscript{12} An association between mild equine asthma and exercise-induced pulmonary hemorrhage has been found in some studies but not in others.

**Severe equine asthma:**
Severe asthma is associated with exposure to high levels of organic molds particularly abundant in moldy hay and poorly ventilated stables. Clinical signs may develop within hours to days of exposure. Even good quality hay contains molds that may trigger clinical signs in susceptible horses. Hay from round bales has been associated with worse clinical presentation presumably because they are usually kept on the field where they are exposed to rain. However, small bales of hay harvested insufficiently dry and stored indoors will also lead to mold growth to reach a maximum for hay baled at 35-50% moisture. Approximately 70 species of fungi and actinomycetes have been identified in hay dust and among them thermophilic molds such as Aspergillus fumigatus, Faenia rectivirgula and Thermoactinomyces vulgaris are particularly abundant in moldy hay. These spores have a small diameter (\textless 5 μm) allowing them to be inhaled deep in peripheral airways (respirable particles) where they may trigger an inflammatory reaction. The severity of the inflammatory response is dose-dependent. Endotoxins are present in large quantities in horses’ environment and potentiate the inflammatory response to inhaled molds. Outdoors airborne pollen and molds appear to also play a role in summer-RAO and some cases or RAO.

**Diagnostic approach for equine asthma:**
A detailed history, physical examination, and diagnostic tests of horses presenting with clinical signs including cough, increased respiratory secretions, and poor performance should help eliminate differential diagnoses. Presence of a fever suggests infectious respiratory diseases such as viral diseases, bronchopneumonia, pleuroneumonia, and pulmonary abscess. Hematology findings may be helpful to rule out bacterial infections. Leukocytosis with neutrophilia is commonly found with bacterial respiratory infections and during the acute phase of a bacterial infection, increased numbers of immature neutrophils (“left shift”) may be observed. Neutrophilia may also accompany non-infectious inflammatory diseases (e.g. exposure to toxins), neoplasia, mycotic, and parasitic infections. Hematologic changes during the early phase of a viral respiratory infection (e.g. influenza) are often characterized by normocytic, normochromic anemia, lymphopenia or lymphocytosis, and sometimes neutropenia.
Neutrophilia may follow within a week of initial clinical signs, particularly in cases of secondary bacterial infection. Monocytosis may develop during the recovery phase of a viral infection. Occasional coughing is chronic (> 3 weeks) in horses with mild asthma. Main differentials are infectious tracheobronchitis, horses with severe asthma during periods of remission, parasitic pneumonitis and other causes of decreased performance. Horses with severe asthma typically display severe exercise intolerance and increased respiratory efforts during periods of disease exacerbation, however these signs may be subtle during periods of disease remission. In those cases, pulmonary function testing or response to therapy will help reach a definitive diagnosis. Cytological analysis of BALF allows recognition of 3 types of inflammatory profiles in mild/moderate. The most commonly encountered profile is characterized by an increased total nucleated cell count with mild neutrophilia (5-20 % cells). Two other cytologic profiles characterized by increased percentages of mast cells (> 2 %) and eosinophils (> 0.1 %) are also observed in some horses. The severity of BALF neutrophilia is usually more pronounced with severe equine asthma (>25%), however there is significant overlap between diseases.

Eosinophilic inflammation may be also be associated with parasitic pneumonitis (*Parascaris equorum*, *Dictyocaulus arnfieldi*), hypersensitivity pneumonitis, fungal pneumonia, and cutaneous habronemiasis. Fecal flotation (Baermann technique) is often not diagnostic of *P. equorum* infection in foals because migration through the lungs occurs during the prepatent period. *Dictyocaulus arnfieldi* follows a complete cycle in donkeys, mules, and asses, however, the infection is usually not patent in horses. Therefore, the Baermann fecal flotation is not useful either. A presumptive diagnosis of parasitic pneumonitis may be reached when respiratory secretions reveal eosinophilic inflammation with evidence of parasite eggs or larvae, exposure to donkeys exists, or anthelmintic therapy results in clinical improvement. Increased metachromatic cells (mast cells, basophils) has been described in horses with equine asthma but has not been associated with other types of respiratory disease.

Direct quantification of airway obstruction requires sophisticated equipment and expertise only available at specialized referral centers. However, transpulmonary pressure may be measured easily in the field using an esophageal balloon catheter made from a foal nasogastric tube and condom sealed at the tip that is connected proximally to a pressure gauge. Using this rudimentary device, transpulmonary pressure can be measured before and after bronchodilation (e.g. using Buscopan®). Horses with severe asthma will experience a decrease in transpulmonary pressure by at least 20% following bronchodilation.

References:

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How to best Manage and treat equine asthma:

Treatment of equine asthma should combine environmental changes and medical therapy. The goals of medical therapy are to control airway inflammation and relieve airflow obstruction using mainly corticosteroids and bronchodilators or antimicrobials when infection is suspected. There are few peer-reviewed clinical trials published regarding therapy of mild equine asthma. Most of the drugs and dosages recommended are based on studies performed on horses with severe asthma (RAO), however good clinical response has been observed using those guidelines. Both systemic and aerosolized drugs are effective, however the potential for adverse effects and prolonged elimination times is greater with systemic administration. The advantages of aerosol therapy are ease of administration and safety. The disadvantages are cost of delivery devices and drugs (metered-dose inhalers). Non-steroidal anti-inflammatory and anti-histamine drugs are ineffective for the treatment of equine asthma.

Environmental management:
Inhaled dust particles play an important role in the pathophysiology of equine asthma and treatment should always include recommendation to decrease exposure to environmental irritants to airways. Two main approaches help reduce exposure of the horse’s airways to respirable particles. The first approach is to use feedstuff and bedding that generate low dust levels. The second approach is to increase removal of airborne particles by improving ventilation in the barn or stall. The ideal environment for horses with severe asthma (RAO) is pasture because exposure to dust is significantly less than in stalls. If for practical reasons the horse cannot be kept on pasture, ventilation in the barn and stall, the type of bedding, feedstuff, and general management should be scrutinized in order to minimize allergen exposure. Most horses improve clinically 1 to 2 weeks after being turned outside on pasture. Horses that only improve partially after being placed on pasture would benefit from medical therapy. Once the horse becomes free of clinical disease (remission), medication can be discontinued. Because of the nature of the disease, susceptible horses may suffer another bout of the disease when exposed to allergens. Horses with summer pasture-associated asthma are generally affected between June and September. The recommended environment for these horses during the summer is low-dust indoor housing. The management of affected horses may be complicated by the fact that some animals suffer both from classic and summer pasture-associated asthma. Studies have demonstrated effective reduction in dust exposure when horses where fed haylage or soaked hay instead of dry hay.\(^1\) Supplementation of the diet with omega-3 polyunsaturated fatty acids in combination with low-dust feed is further effective in resolving clinical signs over a shorter period of time in horses with both moderate and severe asthma.\(^2\)

Medical therapy:
Antibiotics
A large proportion of racehorses suspected of respiratory disease because of poor performance, cough, or nasal discharge are treated with antimicrobials usually without supportive evidence of bacterial infection. However, there is currently no controlled study reporting the efficacy of antimicrobials in the treatment of mild asthma. Available data shows no association between
response to treatment and the type of antimicrobial used, duration of treatment or time elapsed between repeated examinations. Only 50% of racehorses diagnosed with mild asthma based on excess tracheal mucus respond favorably to a single course of antibiotic therapy. Therefore, there is currently no evidence-based data supporting the use of antimicrobials in horses with mild asthma.

Some horses with asthma that develop exacerbation of clinical signs secondary to bacterial infection documented based on tracheal wash cytology and culture would benefit from antimicrobial therapy. But complete resolution would require following up with appropriate therapy aimed at controlling airway inflammation once infection is resolved.

**Systemic corticosteroids**

Corticosteroids are potent inflammation inhibitors proven to be effective for the treatment of severe asthma. Recommended dosages are summarized in Table 1. Triamcinolone acetonide is a long-acting corticosteroid that may improve lung function for 2 to 4 weeks after administration of a single dose but administration should not be repeated at less than 3 months interval because of the risk of complications such as laminitis. Dexamethasone induces a marked improvement in clinical within hours of treatment but maximal benefit may take a week. A reduction in BALF neutrophilia is evident within 3 days of therapy. Dexamethasone has good oral bioavailability however, it may be impaired by feeding. Oral administration of the drug may require high dosages (0.16 mg/kg) and be given after fasting to achieve consistent results. Treatment of severely asthmatic horses with dexamethasone 21-isonicotinate reduces airway obstruction within 3 days after treatment initiation with a maximum effect obtained after 7 days. Isoflupredone acetate has similar efficacy as dexamethasone.

Prednisone is poorly absorbed after oral administration of tablets or liquid forms. Conversely, both liquid and tablet forms of prednisolone are well absorbed in the horse with a bioavailability > 50%. Recommended dose is 0.5-1 mg/kg, once a day.

Deleterious side effects associated with corticosteroid therapy depend on drug potency, dose used, and treatment duration. Adrenal suppression may be minimized by giving medication in the morning when using alternate-day therapy. Long acting and potent corticosteroids (e.g. triamcinolone, dexamethasone) are more likely to cause adverse effects such as immune suppression, iatrogenic Cushing’s disease, adrenal cortex suppression and laminitis. Discontinuation of dexamethasone after an extended treatment period (> 3 weeks) should be done carefully to avoid acute adrenocortical insufficiency. Dexamethasone results in adrenal suppression for up to 3 days as compared to < 24 hours for prednisolone. Therefore, discontinuation of prolonged dexamethasone therapy should be performed by slowly and gradually decreasing the dose until the least suppressive amount (0.01 mg/kg) is given every fourth day for a minimum of 2 weeks. Alternatively, dexamethasone therapy may be replaced by an equipotent dose of prednisolone (1 mg dexamethasone ≈ 8 mg prednisolone) that will be tapered down to alternate day treatment. Before treatment is discontinued, an ACTH stimulation test should be performed to assess the adrenocortical reserve necessary for the horse to cope with stress.

**Inhaled corticosteroids**

Administration of therapeutic substances via inhalation has the advantage of delivering high concentration of the drug directly into the lungs while minimizing the amount absorbed.
systemically and therefore, reducing the risk of adverse effects. In addition, systemic side effects and drug residue are decreased. At least five different inhaled corticosteroids are available to treat inflammatory lung diseases in humans: Beclomethasone dipropionate, budesonide, flunisolide, fluticasone propionate, and triamcinolone acetonide. A common test of potency for inhaled corticosteroids called the McKenzie skin blanching test allows relative ranking of the compounds from least to most potent (relative to dexamethasone potency = 1): flunisolide = triamcinolone acetonide (330) < beclomethasone dipropionate (600) < budesonide (980) < fluticasone propionate (1200). Recommended dosages for these off-label inhaled corticosteroids are summarized in Table 2. The latest and only inhaled corticosteroid approved for the treatment of equine asthma is ciclesonide (Aservo Equihaler; Boehringer Ingelheim).11-12 Clinical experience suggests that a combination of environmental measures aimed at decreasing dust exposure coupled with inhalation therapy using the same drugs effective against severe asthma is also beneficial for mild disease. Currently available aerosol delivery devices used to administer drugs packaged in metered-dose inhalers (MDI) provide similar clinical efficacy (AeroHippus™ and Equine Haler™).4 The Aservo Equihaler includes both the canister with inhalable drug and the aerosol delivery device in one handheld device. Improved clinical signs, decreased airway hyperresponsiveness, and reduced pulmonary inflammation are usually detectable within 2 weeks of therapy. Therapy with inhaled corticosteroids results in faster improvement of clinical signs and lung function as compared to environmental management alone.5,6

**Systemic bronchodilators**

Bronchodilators are indicated to relax airway smooth muscle and relieve airflow obstruction, but they should not be used alone for extended periods of time because they have no anti-inflammatory properties and do not reduce airway hyperresponsiveness. In addition, prolonged use of certain type of bronchodilators (e.g. beta2-agonists) as solo medication induces airway receptor down regulation and renders the drug less effective.7 This phenomenon is prevented by combined use of beta2-agonists with corticosteroids. The three classes of substances available as systemic bronchodilators are anticholinergic, beta2-agonist, and methylxanthine drugs (Table 1). Atropine is an anticholinergic drug that provides rapid and marked improvement in lung function however, potentially serious side effects such as ileus and abdominal pain can develop when high doses are used (22 – 88 mg) or with repeated administration. Another anticholinergic drug, glycopyrrolate, has similar efficacy as atropine but without deleterious effects on gut motility. These drugs may be used as a single dose for the rapid relief of severe airway obstruction and for diagnostic purposes. Clenbuterol hydrochloride syrup (Ventipulmin®) is approved for the treatment of severe equine asthma. Treatment should be initiated at the low end of the dose and increased progressively if no clinical response is noted. Some horses (25 %) may not respond to clenbuterol even at the high end of the dose. Clenbuterol has also anti-inflammatory properties and may help airway mucociliary clearance. Mild side effects such as sweating, muscle tremors, and excitement occur in less than 10 % of horses treated with oral clenbuterol. Albuterol (beta2-receptor agonist) is poorly absorbed orally however, inhalation therapy results in rapid but short lived improvement in lung function (~1 hour).4 Methylxanthine and derivatives may be beneficial in horses with asthma, however plasma levels necessary for bronchodilation vary widely between horses and the range between effective and toxic concentration is narrow. Aminophylline and theophylline administered every 12 hours
improve lung function and clinical signs in up to 50% of affected horses. Common side effects are hyperesthesia, hyperexcitability, and muscle tremors. Pentoxifylline administered to horses with severe asthma results in significant improvement in lung function and is not associated with adverse effects.

**Inhaled bronchodilators**

Two main classes of inhaled bronchodilators have been used in the horse: beta2-agonist and anticholinergic drugs (Table 2). Beta2-agonists induce airway smooth muscle relaxation regardless of bronchoconstriction mechanism and also inhibit mast cell degranulation. Albuterol, levalbuterol, pirbuterol, and fenoterol are short acting bronchodilators (1-2 hours) with rapid onset of action (5 minutes). Some horses may benefit from the effects of albuterol for up to 7 hours. Salmeterol and formoterol are long-acting beta2-agonists (6-8 hours) suitable for twice daily dosing but with slow onset of action (15 minutes). Salmeterol is currently only available as a dry powder “diskus” that cannot be administered to horses. Albuterol results in maximal bronchodilation with 540 μg from metered-dose inhaler (~ 1 μg/kg) using an AeroHippus or Equine Haler delivery device but only 150 μg (~ 0.3 μg/kg) when administered with ultrasonic nebulizer (e.g. Sahoma or Flexineb).³

Ipratropium bromide is an anticholinergic drug chemically derived from atropine but devoid of side effects when administered by inhalation. Nebulization of 2 μg/kg causes bronchodilation for approximately 6 hours with a maximum effect obtained one hour after administration. The effects of anticholinergic drugs on airway smooth muscle are additive to beta2-agonists.

**Other therapies**

Supplementation of diet with omega-3 polyunsaturated fatty acids (e.g. DHA and EPA; Aleira®) in combination with reducing exposure to dust results in improved clinical signs and lung function, as well as reduced airway inflammation.⁴ Sodium cromoglycate (cromolyn) improves clinical signs and decreases bronchial hyperresponsiveness when administered to horses with mild asthma characterized by a high mast cell count in BAL fluid.⁵ However, it is ineffective for the treatment of mild asthma with other inflammatory profiles.

Oral administration of low-dose (50 – 150 U q24 hours) interferon alpha (IFNα) for five days reduces neutrophil, macrophage, lymphocyte, and total nucleated cell counts in the BALF of racehorses with mild asthma as well as cough for at least two weeks.¹⁰ Higher doses of IFNα (450 U) appear to be less effective. Endoscopic scores based on respiratory exudate, cough, and pharyngeal lymphoid hyperplasia were significantly reduced after one week of therapy but were not different from placebo at 2 weeks.

![Table 1: Medications used systemically for severe equine asthma therapy.](image-url)
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose delivered per actuation</th>
<th>Propellant</th>
<th>Device</th>
<th>Dose</th>
<th>Duration of action</th>
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</thead>
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<td><strong>Corticosteroids</strong></td>
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<tr>
<td>Beclomethasone</td>
<td>80 μg</td>
<td>HFA</td>
<td>EADD a</td>
<td>1-3 μg/kg, q12 hours</td>
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<tr>
<td>Ciclesonide</td>
<td>343 μg</td>
<td>Soft Mist™</td>
<td>Aservo Equihaler®</td>
<td>2744 μg q12h for 5 days; 4116 μg q24h for 5 days</td>
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<td>HFA</td>
<td>Aeromask b</td>
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<td><strong>Bronchodilators</strong></td>
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<td>EADD</td>
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<td>Ipratropium</td>
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<td>CFC</td>
<td>Aeromask</td>
<td>0.2-0.4 μg/kg</td>
<td>4-6 hours</td>
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</tbody>
</table>

Table 2: Medications used for aerosoltherapy of RAO in the horse. a Equine aerosol delivery device (not commercially available). b Aeromask and AeroHippus (Trudell Medical International, Ontario, Canada). c Equine Haler (Jorgensen Laboratories). d EquiPoudre (Agritronics Int., Meux, Belgium).
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Device</th>
<th>Route</th>
<th>Duration</th>
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</thead>
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<td>200 μg/capsule</td>
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<td>EquiPoudre</td>
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<td>0.02% solution for nebulization</td>
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<td>CFC</td>
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<td>1-2 mg</td>
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<td>EADD</td>
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<td>Salmeterol</td>
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<td>CFC</td>
<td>Aeromask</td>
<td>210 μg</td>
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</table>

**Cromones**

<table>
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<th>Drug</th>
<th>Dosage</th>
<th>Device</th>
<th>Route</th>
<th>Duration</th>
</tr>
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<tbody>
<tr>
<td>0.02% solution for nebulization</td>
<td>2 ml vials</td>
<td>Jet nebulizer</td>
<td>200 mg, q12 h</td>
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<td>Ultrasonic nebulizer</td>
<td>80 mg q24 h</td>
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**References:**
BROODMARE EMERGENCIES IN THE FIELD
Sarah Shaw, DVM, DACVIM

Broodmares present with some of the most critical emergencies in equine practice. A timely response by an equipped veterinarian can prove lifesaving in many cases.

Uterine Torsion

Uterine torsion is most commonly seen in the early part of the third trimester. A study by Spoormakers et al (2016) found the mean gestation at diagnosis was 283 days, with a range from 153-369 days. Mares generally present with mild and intermittent colic signs though some mares present with severe pain. Diagnosis is made by rectal palpation with a tight band coursing across the abdomen. The foal will be very difficult to palpate if a uterine torsion is present. Mares with a uterine torsion should be referred to a surgical facility. Surgical correction can be performed by standing flank laparotomy or ventral midline incision. Historically, mare survival rates were between 60 – 84% and foal survival rates were between 30 – 54%. Spoormakers et al (2016) reported 90.5% survival in mares and 82.3% survival in foals to discharge. Standing flank laparotomy was reported with a higher survival rate of foals (88.7%) compared with 35% foal survival with other approaches. Survival rates of mares and foals were significantly higher when uterine torsion was identified prior to 320 days gestation. This study also evaluated future fertility of the mare. Fertility did not differ between mares treated by standing flank laparotomy compared to other techniques and 93.5% became pregnant again.

Dystocia

Equine dystocias are true emergencies. It is ideal to discuss foaling emergencies with your clients in advance of foaling season. No device can replace in-person foal watch (either on camera or stall-side, as the devices on the market all have the potential to fail. Foaling attendants should also not rely solely on milk analysis kits, as they are not 100% accurate and late-term abortions can also result in dystocia. Inexperienced foaling attendants should be shown a photo of a red bag and a pair of scissors should be either part of the foaling kit or attached to the foaling stall door. Stage 2 of labor in mares should take less than 30 minutes and failure to progress warrants an immediate call to a veterinarian.

As a veterinarian, dystocia and foal resuscitation supplies should be easily accessible and stocked. A foaling kit should contain sterile sleeves, straps, handles, a head snare, an ambubag, an endotracheal tube, epinephrine, sedation for the mare, small needles and syringes, and potentially other emergency medications (Solu-medrol, atropine, etc). Towels, light lube, a stomach tube, and pump should also be readily accessible. Some veterinarians may elect to carry fetotomy supplies.

Adequate sedation (detomidine and butorphanol) is an essential first step once a dystocia has been identified. These mares require a significant amount of pain control. A small volume epidural (4-6 mL 2% lidocaine) also provides pain relief without ataxia. Aseptic technique reduces the risk of metritis. The use of light lube mixed with warm water and pumped around the fetus helps to create space and lubrication for manipulation the foal. If significant progress in correcting the dystocia cannot be made in 10-15 minutes, either referral to a surgical facility if possible or general anesthesia of the mare is recommended. Anesthesia of the mare and lifting of her hindquarters using a pulley system and ropes around the hind fetlocks allows more room for manipulation of the foal (controlled vaginal delivery).
Following any significant dystocia (requiring assisted vaginal delivery, controlled vaginal delivery, cesarean section, or fetotomy) the mare should be covered with broad-spectrum, injectable antibiotics (ie. penicillin and gentamicin), provided pain-management, and monitored carefully for fever, lethargy, and inappetence. Large-volume uterine lavage is recommended 4-6 hours post-dystocia, with a brief abdominal ultrasound pre- and post-lavage to identify any excess peritoneal fluid suggestive of either a uterine tear or uterine artery hemorrhage. Twice daily uterine lavage should be performed until the fluid is clear or mare is considered stable. Low doses of oxytocin (10 IU either IV or IM) can be given every 2-4 hours to aid uterine clearance. Once the mare is stable, free of fevers, and uterine flushes are clear (3 to 5 days), a 7-10 day course of trimethoprim/sulfa medication is recommended.

**Retained Fetal Membranes**

The placenta is considered retained if it has not been passed 3 hours post-foaling. Managers and foaling attendants should be informed that this is considered an emergency. Oxytocin should be part of every foaling kit so that small doses (10 IU IV or IM) can be given at 3 hours if the placenta has not passed. Mares with retained fetal membranes (RFM) should be treated prophylactically with systemic antimicrobials (ie. penicillin and gentamicin) and flunixin (0.25 – 0.5 mg/kg TID). Prophylactic icing of the mare’s feet is recommended if possible to prevent laminitis. Frequent, large volume uterine lavage with isotonic saline should be performed 2-3 times daily. More recently, a technique using umbilical vessel water infusion has been described as a very effective treatment for RFM in mares. A detailed description of this procedure is available at [https://www.ivis.org/library/aaep/aaep-annual-convention-las-vegas-2015/how-to-use-umbilical-vessel-water-infusion-to-treat-retained-fetal-membranes-mares](https://www.ivis.org/library/aaep/aaep-annual-convention-las-vegas-2015/how-to-use-umbilical-vessel-water-infusion-to-treat-retained-fetal-membranes-mares).

Some mares will have concurrent hypocalcemia with RFM and supplementation with IV calcium borogluconate diluted in IV fluids may be beneficial. This can be combined with slow IV infusion of 20 IU of oxytocin diluted in 1 L saline to induce uterine contractions.

**Peripartum Colic**

Colic in peripartum mares is an emergency. Large colon volvulus and uterine artery hemorrhage can occur pre- or post-partum and both cause clinical signs of severe pain. Uterine tears are generally seen postpartum and are associated with mild-moderate pain, fever, and inappetence.

Large colon volvulus (LCV) is a common cause of severe colic in postpartum mares. Differentiating a large colon volvulus can usually be done with a physical exam, palpation per rectum, and point-of-care ultrasound (POCUS). Clinical signs specific to LCV include a mare with severe colic signs, a poor response to sedation, and a gas-distended colon on palpation per rectum (though 720-degree volvulus will often not palpate distended). Heart rate may be normal despite pain due to a vagal response. Abdominal ultrasound may reveal a thickened colon wall, colon vessels visible in the right ventral flank, and no cellular free fluid (as would be noted in periparturient hemorrhage). Immediate referral to a surgical facility is required to correct this intestinal event. Mares will likely require IM sedation for the trailer ride, but this should be discussed with the surgeon receiving the case if possible.

Uterine artery hemorrhage is most commonly seen postpartum. Mares present in moderate to severe pain, with pale to white mucus membranes, and an elevated heart rate and respiratory rate. Mares may also sweat profusely or have blood passing from their vulva. Peripheral blood lactate will often be elevated. Abdominal ultrasound should be performed in order to identify a
bleed into the abdomen (echogenic, swirling fluid). Palpation per rectum should be performed carefully, without the use of a nose twitch, to identify a bleed within the broad ligaments. Treatment includes sedation to manage pain (detomidine and butorphanol), antimicrobial treatment (ie. penicillin and gentamicin), and aminocaproic acid to aid in stable clot formation (an initial loading dose of 40 mg/kg diluted in 1 L saline over 30-60 minutes followed by 20 mg/kg diluted in 1 L saline over 30-60 minutes QID). Naloxone has been used anecdotally in mares with hemorrhagic shock to release platelets (12-20 mg IV bolus). Mares with uterine artery bleeds are commonly azotemic, so conservative treatment with NSAIDs should be administered and monitoring of serum biochemistry is recommended.

Uterine tears in postpartum mares are best identified via abdominal ultrasound. An increase in anechoic to echogenic free abdominal fluid is seen in conjunction with a fever and colic signs, consistent with peritonitis. The author’s practice protocol is to scan post-partum mare abdomens pre- and post-lavage to rule out free peritoneal fluid accumulation. Referral to a surgical facility for abdominal lavage (medical or surgical) is recommended in mares with a suspected or identified uterine tear. A retrospective study by Javsicas et al. reported 75% survival in mares with uterine tears identified within 7 days of foaling. They found no significant difference in outcomes between medical and surgical management. In that study, uterine tears occurred more commonly in the uterine horns and more so in the right horn. While medical management yielded no difference in survival outcomes, medical therapy can be as costly as surgical treatment.

**Metritis**

Metritis in mares is considered life-threatening and handled similarly to retained fetal membranes. Mares typically present with a fever (up to 41°C) and can be dull or colicky. Mucus membranes are often severely injected and bounding digital pulses may be noted early in disease. A careful examination of the vaginal walls should be performed to rule out any tears communicating with the abdomen. It is difficult to assess cervical integrity post-partum.

The most essential treatment is frequent uterine lavage, which should be performed 2-3 times daily and each time until fluid is clear. If fluid return is poor, oxytocin (10 IU IV) can be given during the lavage to improve the volume returned. Mares should be covered with broad-spectrum, injectable antibiotics (ie. penicillin and gentamicin), flunixin (0.25-0.5 mg/kg IV TID; enough to control pyrexia), oxytocin (10-20 IU IM q2-4 hours), and ideally placed in ice boots. Intra-uterine treatment is not considered beneficial. If the mare has a nursing foal by her side, the foal may require supplementation with milk replacer.

**Selected References**


Spoormakers TJP, Graat EAM, Ter Braake F, Stout TAE, and Bergman HJ. Mare and foal survival and subsequent fertility of mares treated for uterine torsion. 2016 *Eq Vet J.* 48: 172-175
FIELD MANAGEMENT OF SICK NEONATAL FOALS
Sarah Shaw, DVM, DACVIM

Foal emergencies are dreaded by many veterinarians as they are stressful and often critical. Neonatal maladjustment syndrome (NMS), or dummy foal syndrome, may be caused by sepsis, hypoxic ischemic encephalopathy, increased circulating neurosteroids, or a combination of the above. Colic is also common in neonates, and in those less than 5 days of age we need to differentiate between enteritis, meconium impaction, and a ruptured bladder.

Initial assessment of the patient’s mentation, recumbency, maturity, hydration status, suckle reflex, and TPR should be performed on a sick neonate. A systematic approach to a foal examination differs slightly from that of a mature horse. Hydration status can be assessed through a skin tent on the superior eyelid, the color and CRT of mucus membranes, pulse quality (including that in distal limbs), temperature of extremities, and the position of the globe. Particular attention should be paid to the respiratory pattern, palpation of the ribs, airflow from the nostrils, the abdominal contour, the umbilicus, range of motion of joints, and history or evidence of urination and passing manure. Evidence of entropion, corneal ulceration, and icteric mucus membranes are often present in sick neonates. The hard palate integrity can be assessed by palpation but a cleft of the soft palate is typically diagnosed endoscopically. An examination of the mare should include mucus membranes, udder, and colostrum quality.

Table 1. Normal Physical Examination Parameters by Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart Rate (bpm)</th>
<th>Respiratory Rate (brpm)</th>
<th>Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>60 – 80</td>
<td>Gasping</td>
<td>37.2 – 39.1</td>
</tr>
<tr>
<td>0 – 2 hours</td>
<td>120 - 150</td>
<td>40 – 60</td>
<td>37.2 – 39.1</td>
</tr>
<tr>
<td>12 hours</td>
<td>80 – 120</td>
<td>30 – 40</td>
<td>37.2 – 39.1</td>
</tr>
<tr>
<td>24 hours</td>
<td>80 - 100</td>
<td>30 - 35</td>
<td>37.2 – 39.1</td>
</tr>
</tbody>
</table>

Table 2. Foal Milestones Timetable

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Concern</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sternal</td>
<td>5 min</td>
<td>5 – 10 min</td>
<td>&gt; 10 min</td>
</tr>
<tr>
<td>Suckle</td>
<td>5 – 10 min</td>
<td>10 – 15 min</td>
<td>&gt; 15 min</td>
</tr>
<tr>
<td>Standing</td>
<td>&lt; 1 hr</td>
<td>1 – 2 hr</td>
<td>&gt; 2 hr</td>
</tr>
<tr>
<td>Nursing</td>
<td>&lt; 2 hr</td>
<td>3 – 4 hr</td>
<td>&gt; 4 hr</td>
</tr>
</tbody>
</table>

Stall-side Tests

Stall-side tests can guide treatment in equine neonates. IgG testing should be performed on all routine new foal exams. Recheck of a previously-normal IgG may reveal a significant decrease in cases of sepsis. The Gamma-Check E (qualitative test with high sensitivity) and Foal Snap Test (semi-quantitative test with good sensitivity and specificity) are both available commercially. Complete failure of passive transfer (FPT) is defined as an IgG less than 400 mg/dL and partial FPT is defined as an IgG between 400 and 800 mg/dL. Some insurance companies will require demonstration of an IgG > 800 mg/dL. The administration of commercial plasma products is common to address FPT in foals. One liter of commercial plasma is expected to increase the IgG by 200-300 mg/dL if no ongoing losses (sepsis) are occurring. Plasma administration rates should begin at 0.5 mL/kg bwt/h and gradually increased if no adverse effects are noted. Plasma reactions can manifest as pale or white mucus membranes, increased heart and respiratory rate, muscle tremors, or colic, and plasma administration should be paused if these signs are noted. If signs recur once plasma administration is re-started, the
procedure should be discontinued completely. Flunixin, Solu-medrol, and epinephrine should be kept on-hand for all plasma administrations. Foals that have received milk replacer have been anecdotally reported to be at an increased risk of plasma reactions.

Lactometers can be used to further assess hydration and gauge response to ongoing fluid therapy. A normal blood lactate in neonate is less than 2 mmol/L. The Lactate Pro or Lactate Plus has been shown to display good reliability and accuracy when compared to a laboratory-based analyzer (Tanner et al.). Point-of-care glucose monitoring and SAA measurements may also be helpful in guiding treatment when immediate blood gas analysis or serum biochemistry results are not available.

**Laboratory Tests**

Complete blood count (CBC) and serum biochemistry should be evaluated on all sick foals. Leukopenia is strongly suggestive of sepsis. Anemia may indicate neonatal isoerythrolysis. An elevated creatinine may not indicate primary renal disease. Rather, creatinine is cleared by the placenta and may remain elevated post-partum in foals born to mares with placental insufficiency. Placental creatinine increases should decrease by 33-50% following 24 hours of IV fluid administration.

Blood culture collection can be performed on-farm while placing a sterile catheter, or aseptically using a needle and syringe. This is beneficial even if referral is elected, as antimicrobial therapy can be initiated prior to shipping and the culture vial can be sent to the referral facility with the foal.

**Sedation of the Neonate**

Sedation may be required in foals in order to place an IV catheter or perform diagnostic imaging. Foals with HIE can be spastic and difficult to restrain. In compromised foals, especially those with any history of seizure-like activity, diazepam (0.1 mg/kg IV) is the author’s sedation of choice. Butorphanol (0.05 mg/kg IV) results in more profound sedation but is also safe in compromised foals. The use of alpha-2 agonists increases the risk of reverse shunting through a patent foramen ovale or patent ductus arteriosus.

**Intravenous Fluids**

Fluid therapy is recommended to treat hypovolemia and dehydration. Correction of deficits is best performed using boluses of 20 mL/kg bwt (1 L in an average-sized foal). Available replacement fluids include Normosol, lactated Ringers solution (LRS), and Plasmalyte. This can be repeated every 20 minutes, up to 3 times, with careful assessment of the foal’s hydration parameters following each bolus. Attention to the foal’s pulse quality, warmth of distal limbs, and mentation will help guide the number of boluses required. Supplemental glucose is not recommended in resuscitation fluids as it may cause hyperglycemia.

Maintenance fluids can be given at a rate of 2-4 mL/kg bwt. If glucose supplementation is elected, blood glucose should be monitored using a glucometer to maintain blood glucose levels between 4 – 5.5 mmol/L. 5% glucose can be provided by IV infusion at a rate of 4 mg/kg bwt/min. The replacement fluids listed above are less-suitable for long-term administration due to their high sodium concentrations and the foal’s limited ability to excrete sodium. The exception is foals with diarrhea that require ongoing replacement of electrolytes. Ideal
maintenance fluids that restrict sodium include 5% dextrose, normosol-M, or a combination of 5% dextrose and LRS.

**Enteral Nutrition**

The placement of an indwelling feeding tube can be very helpful on farms with experienced personnel. A practical approach is outlined by Austin (2016).

**Ancillary Diagnostics**

Ultrasoundography of the abdomen, umbilicus, and thorax can be helpful in determining potential comorbidities or a source of systemic sepsis. Radiographs of the carpus and tarsus (DPs and laterals) should be performed to look for incomplete ossification of cuboidal bones. Thoracic radiographs may also be performed on-farm on neonates to identify atelectasis or pneumonia.

**Neonatal Sepsis**

Neonatal sepsis is the leading cause of morbidity and mortality in foals under 7 days of age. The sepsis scoring system can be used, where a score of over 11 is strongly suggestive of sepsis. The most common route of bacterial entry in foals is through the gastrointestinal tract. Clinical manifestations of sepsis include fever or hypothermia, lethargy, weakness, tachycardia, and tachypnea. A decrease in nursing, dehydration, entropion, injected sclera and mucus membranes, iters, uveitis, corneal ulcers, and coagulopathy can also be identified. Pneumonia or synovial distension may be associated with septic arthritis, osteomyelitis, septic physis, and tenosynovitis. Less commonly, septic foals can develop meningitis which can mimic the clinical signs of HIE (depressed, seizures, and rigid neck).

**Table 3. Common antimicrobial drugs used in neonates.**

<table>
<thead>
<tr>
<th>Antimicrobial</th>
<th>Dose</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>20–30 mg/kg bw t i.v. or i.m.</td>
<td>May cause renal damage, Gram -ve coverage only</td>
</tr>
<tr>
<td>Ampicillin Na</td>
<td>20 mg/kg bw t l.v. q. 6 h</td>
<td>Usually used with aminoglycoside</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>40 mg/kg i.v. q. 6 h</td>
<td>Used only when resistance to commonly used antibiotics is identified</td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>40–50 mg/kg bw t l.v. q. 6 h</td>
<td>Used only when resistance to commonly used antibiotics is identified</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>5–10 mg/kg bw t l.v. or i.m. q. 6–12 h</td>
<td>Used alone or in combination with aminoglycoside</td>
</tr>
<tr>
<td>Chloramphenicol*</td>
<td>40 mg/kg bw t i.v. q. 6 h</td>
<td>Wear gloves as there is a human health risk</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>8–15 mg/kg i.v. or i.m. q. 24 h</td>
<td>May cause renal damage, Gram -ve coverage only</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>10 mg/kg bw t per os. i.v., or per rectum q. 12 h</td>
<td>Used most commonly for suspected clostridial infections</td>
</tr>
<tr>
<td>Oxytetracycline</td>
<td>2 g/50 kg foal</td>
<td>For treatment of contracted tendons. Do not give to dehydrated foals as may cause renal injury. Repeat q. 24-48 h.</td>
</tr>
<tr>
<td>Potassium penicillin</td>
<td>22,000 units/kg bw t l.v. q. 6 h</td>
<td>Used in combination with an aminoglycoside</td>
</tr>
<tr>
<td>Ticarcillin/clavulanic acid</td>
<td>40–50 mg/kg bw t l.v. or i.m. q. 8 h</td>
<td>For use in β-lactamase producing bacteria</td>
</tr>
<tr>
<td>Trimethoprim-sulfonamide</td>
<td>15 mg/kg bw t per os q. 12 h</td>
<td>Used for long-term treatment in foals with demonstrated susceptibility</td>
</tr>
</tbody>
</table>

*Use of this antibiotic may be restricted or prohibited in some countries.

Source: Austin, SM Management and treatment of the sick equine neonate in ambulatory practice

**Hypoxic Ischemic Encephalopathy**

Hypoxic ischemic encephalopathy is most commonly the result of placental insufficiency rather than a foaling event. These dummy foals often present with seizures. Diazepan can be given at
5 mg IV for a 50 kg foal PRN to control seizure activity. Due to the overlap of HIE with neonatal sepsis, foals suspected to have HIE should be treated with broad-spectrum antimicrobials. Generally these foals require referral for 24-hour fluids, oxygen, and supportive care.

### Table 4. Medications used for treatment neonatal encephalopathy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>0.11–0.44 mg/kg bwt, i.v., repeat as needed</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.11–0.44 mg/kg bwt i.v.</td>
</tr>
<tr>
<td></td>
<td>0.4 mg/kg bwt i.v. as constant rate infusion</td>
</tr>
<tr>
<td>Phenobarbital loading</td>
<td>Loading dose: 10 mg/kg bwt in 30 ml sterile water, give slowly</td>
</tr>
<tr>
<td></td>
<td>Maintenance dose: 5 mg/kg bwt i.v. q. 12 h slowly</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Loading dose: 10 mg/kg bwt per os</td>
</tr>
<tr>
<td></td>
<td>Maintenance dose: 3.0 mg/kg bwt per os, q, 24 h</td>
</tr>
<tr>
<td>Dimethyl sulfoxide</td>
<td>1.0 g/kg bwt as 20% solution i.v.</td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>Loading dose: 50 mg/kg bwt as 1% solution slow i.v.</td>
</tr>
<tr>
<td></td>
<td>Maintenance dose: 24 mg/kg bwt/h for 24 h</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>44 mg/kg bwt per os within 4 h of birth</td>
</tr>
<tr>
<td>Thiamine</td>
<td>1–20 mg/kg bwt q. 12 h i.m.</td>
</tr>
<tr>
<td>Ascorbic acid</td>
<td>50–100 mg/kg bwt/day</td>
</tr>
<tr>
<td>α-Tocopherol</td>
<td>500–1000 iu, per os, q. 24 h</td>
</tr>
</tbody>
</table>

Source: Austin, SM Management and treatment of the sick equine neonate in ambulatory practice

### Maladjustment Syndrome with Abnormal Neurosteroids

Neonatal maladjustment syndrome has also been associated with increased circulating neurosteroids. These foals may also have other comorbidities, such as sepsis and HIE, so careful examination is recommended to determine an appropriate treatment plan. The Madigan foal squeeze was developed in order to mimic the act of foaling and has been beneficial in treatment of this subset of foals. It can also be used as a method of medication-free restraint when giving plasma or performing ultrasounds on healthy foals less than 3 days of age. Contraindications for the foal squeeze include: foal never stood, rib fractures, respiratory distress, shock, sepsis, signs of prematurity, and congenital abnormalities. A detailed description of the method may be found at: https://compneuro.vetmed.ucdavis.edu/sites/g/files/dgvnks5376/files/inline-files/mfsm_instructions_0.pdf

### Colic in the Neonate

Equine neonates with colic with colic signs should be worked up with a thorough physical examination and point of care ultrasound (POCUS). Common causes include meconium impaction, ruptured bladder, and enteritis.
Meconium Impaction

These foals can be colicky within hours of birth and up to 48 hours later. They often stand with their back arched straining to pass manure. There should be no significant increase in free fluid on abdominal ultrasound and the small intestinal motility is generally within normal limits. Echogenic material may be visualized in the small colon.

Treatment with a fleet or Microlax enema can be performed once. If no meconium is passed, a soapy water enema and a single dose of flunixin (0.25 – 0.5 mg/kg IV or PO) can be administered. If colic signs persist and meconium impaction is still identified, an acetylcysteine retention enema can be performed while administering IV crystalloid fluids. For this procedure, the foal is sedated using diazepam or butorphanol (as above) and a 28 or 30 Fr foley catheter is placed per rectum and the bulb inflated. The foal should receive 200 mL of a 4% acetylcysteine solution, the foley catheter clamped for 20 minutes, and then released. Co-administration of 1 L IV fluids in these foals is also beneficial.

Ruptured Bladder

Foals with a ruptured bladder will often posture to urinate by stretching out with a flat back. Free, anechoic, peritoneal fluid can be identified on abdominal ultrasound. Referral to a surgical facility for electrolyte correction and surgical intervention is recommended. Rarely, foals can have tears in either their ureters (resulting in uroabdomen) or urethras (in males this results in subcutaneous accumulation of urine and subsequent tissue sloughing).

Enteritis

Enteritis in foals is managed similarly to sepsis with IV fluids, antimicrobial drugs, and careful enteral nutrition. At least 50% of foals with diarrhea have systemic sepsis identified on blood culture. Intravenous replacement fluids are recommended and, practically, can be given as q2 – q4 hr boluses on farm through a long-term indwelling IV catheter. Enteritis in neonates < 7 days of age is commonly associated with Clostridial disease. Metronidazole can be given IV (10 mg/kg, slowly, IV, alongside isotonic IV fluids) and is affordable in foals. Partial or complete GI rest often benefits these foals and limiting nursing in conjunction with IV fluid administration is possible on-farm with good personnel. Probiotics, gastroprotectants, and diatomaceous earth are optional adjunctive therapies.

Selected References


Rhodococcus equi

*Rhodococcus equi* is a significant cause of morbidity and mortality in foals. The most common manifestation of disease is pulmonary consolidations or abscesses in foals aged 3 weeks to 6 months. Sanz *et al* (2013) performed a challenge study looking at different doses of bacterial inoculum at different ages. Foals challenged at 2- and 3-weeks of age developed pneumonia at all doses. Conversely, foals challenged at 3- and 6-weeks of age with the low doses did not develop clinical evidence of pneumonia. This study supports the hypothesis that susceptibility to *R. equi* infection diminishes early in foals. Exposure is therefore believed to occur in the first 1-3 weeks of life. Due to the slow-growing nature of the pyogranulomatous lesions, clinical disease generally occurs weeks to months after exposure.

The administration of *R. equi* hyperimmune plasma in the first 48 hours of life continues to be the standard-of-care for foals when economically feasible. This expensive and labor-intensive procedure is variably effective with conflicting efficacy in different studies. Ultrasound screening programs have proven beneficial in the field to identify foals with subclinical disease. A common approach is to perform thoracic ultrasound every 2 weeks, beginning at 3-4 weeks of age, until at least 8 weeks of age. A number of studies have revealed that many subclinical lesions will resolve spontaneously, without any antimicrobial treatment (Venner *et al* and Chaffin *et al*). In these studies, total maximal diameter (TMD) was measured and foals with lesions measuring over 10 cm (Venner *et al*) or 20 cm (Chaffin *et al*) were treated. On farms in Southern Ontario, foals sometimes present with clinical disease despite having a smaller TMD of lesions. Definitive diagnosis of *R. equi* pneumonia requires a tracheobronchial aspirate with evidence of septic suppurative inflammation and growth of *R equi*. However, empirical treatment is often administered based on farm history, clinical signs, and thoracic ultrasound. Treatment is recommended in all foals with a combination of clinical signs (fever, cough, diarrhea, lethargy) and evidence of pulmonary lesions. Treatment with a macrolide antibiotic (clarithromycin or azithromycin) in combination with rifampin remains the treatment of choice unless rifampin or macrolide resistance is suspected or identified. A recent study by Erol *et al* (2022) evaluated dual therapy with clarithromycin and either doxycycline or minocycline. These combinations resulted in significantly lower mutant prevention combinations. Further research is warranted on these combinations in order to minimize the continued emergence of antimicrobial resistance in *R. equi* isolates.

Extra-pulmonary disorders (EPDs) are also common with *R. equi* infection. A retrospective study by Reuss *et al*. identified EPDs in 74% of foals with *R. equi* and this likely underestimates the true prevalence of EPDs. Extrapulmonary disorders include abdominal abscesses, uveitis, omphalophlebitis, polysynovitis and polytenosynovitis, pyogranulomatous hepatitis, ulcerative typhlocolitis and pyogranulomatous nephritis. Reuss et al. reported that intra-abdominal abscessation was associated with non-survival and only 40% of the foals with intra-abdominal abscesses presented with diarrhea. Foals with persistent fevers or clinical signs outside of pneumonia should be evaluated for EPDs. May foals with EPDs of *R. equi* respond favourably to systemic treatment with a macrolide and rifampin.
Other Foal Pneumonias

*Streptococcus equi* subsp. *zooepidemicus* is the most common cause of bacterial pneumonia in foals and weanlings. As it is a normal inhabitant of the upper airway, it may opportunistically invade the lower airway following viral respiratory infections. Anecdotally, the author finds that foals greater than 16 weeks of age presenting with clinical signs and sonographic findings consistent with pneumonia are more likely to culture *S. zooepidemicus* than *R. equi*. Strep pneumonias also respond exceptionally well to macrolide antibiotics but as foals age, they are at an increased risk of antimicrobial-associated diarrhea. Ceftiofur sodium (Excenel®), ceftiofur crystalline free acid (Excede®), penicillin and doxycycline are all antimicrobial options for the treatment of pneumonia caused by *S. zooepidemicus*. While isolates are often susceptible to trimethoprim/sulfa combinations *in vitro*, they may be resistant *in vivo*.

Viral pneumonias including equine herpesviruses (EHV) 1 & 4, equine influenza (EI), and equine arteritis virus often affect weanling foals. Respiratory PCR panels can be helpful when trying to determine the cause a respiratory disease outbreak in foals and weanlings. Identifying EHV or EI may also motivate owners to vaccinate their mares and foals against these pathogens.

Umbilical Infection

Umbilical infections are relatively common in foals less than 8 weeks of age and can affect any of the following structures: the external umbilicus, the umbilical vein, the urachus, and/or the umbilical arteries. Omphalophlebitis is a differential for fevers in foals and can be identified sonographically. A linear rectal probe is an ideal shape and provides sufficient depth and detail for ultrasonography of the umbilical structures. Adequate restraint is recommended in order to prevent foal kicks to the ultrasonographer’s shins. The umbilical vein should measure no greater than 1 cm diameter just cranial to the urachus and become smaller as it courses cranially into the liver. The urachus and arteries measured together should be 2.5 cm or less and the umbilical arteries should measure less than 1 cm as they insert on the bladder. A detailed description of umbilical ultrasound in neonatal foals is available at: https://www.ivis.org/sites/default/files/library/aaep/2002/9101020000261.PDF.

Medical management is often successful. However, surgical removal of the umbilical remnants was associated with high survival in a 2019 study (Reig Codina *et al.* in foals without concurrent septic arthritis/physitis or failure of passive transfer. Omphalophlebitis is a risk factor for the development of septic arthritis, septic tenosynovitis, and septic physis and this should be discussed with the client prior to pursuing medical management. The urachus is the most commonly infected umbilical structure. If the urachus is mildly enlarged with no involvement of the umbilical vein or arteries, or if only the external umbilicus is involved, the author will often prescribe trimethoprim/sulfa with or without rifampin. Treatment with chloramphenicol (44 – 50 mg/kg PO TID) is reserved for more severe infections and allows broad-spectrum coverage and good penetration. Umbilical infections involving the cranial aspect of the umbilical vein can lead to liver abscesses and are difficult to address surgically. Treatment with broad-spectrum injectable antibiotics (penicillin or ceftiofur sodium + amikacin) is recommended. Repeat ultrasound should be performed every 3-5 days to ensure response to treatment.

Septic Arthritis and Physitis

Polysynovitis/tenosynovitis can either be immune-mediated secondary to a primary infection or septic in nature. Non-septic polysynovitis, as is seen with *R. equi* infections, results in joint or...
tendon sheath effusion but no apparent lameness. Foals with joint or tendon sheath effusion and lameness should be approached as a septic process. These foals often have a fever and elevated white blood cell count. If referral to a surgical facility is not an option, joint fluid collection and needle lavage can be performed on-farm. Brief, general anesthesia in foals can be achieved by administering xylazine (0.5 – 1 mg/kg IV) and ketamine (2.2 mg/kg IV) in the same syringe without paradoxical excitement. Septic physitis cases may be anesthetized and treated with IV regional limb perfusion. Systemic treatment with injectable antimicrobials is also recommended (ie. procaine penicillin, 22,000 IU/kg IM BID and amikacin, 21-25 mg/kg IV SID for 3-5 days) prior to switching to oral medications. Chloramphenicol (44-50 mg/kg PO TID) has good bone penetration and can be effective for the treatment of septic physitis. NSAIDs are valuable for pain control but should be used with caution and in conjunction with monitoring renal values on serum biochemistry.

Lawsonia intracellularis Infection

Lawsonia infection most commonly affects weanlings and short-yearlings in the late-fall and early winter, though older yearlings and rarely older horses can also present with clinical disease. Affected individuals often present after stressful events (ie. weaning, shipping, or prepping for sales) with a fever and lethargy. Bloodwork reveals panhypoproteinemia, which can be severe. Diarrhea, distal limb edema, and throatlash edema are seen in more advanced cases. Further evidence of Lawsonia infection includes thickened small intestinal walls on abdominal ultrasound. Diagnosis is made based on clinical signs, abdominal ultrasound, and Lawsonia intracellularis identified on fecal PCR. Paired serologic testing using an immunoperoxidase monolayer assay can be helpful retrospectively when looking at exposure on-farm. Once a novel case is identified on a farm, serum total protein should be monitored weekly in all similar-aged youngstock. Some farm managers may be comfortable pulling blood and measuring total protein on a refractometer.

Lawsonia intracellularis is susceptible to a number of commonly-used antimicrobials in horses. Oxytetracycline (6.6 mg/kg IV, SID) for 3 days, followed by 10-14 days of treatment with doxycycline (5 mg/kg PO BID) is sufficient for eliminating the bacterial infection. Macrolide antibiotics and chloramphenicol are also effective in treating Lawsonia infection. However, due to the severity of this protein-losing enteropathy, recovery is slow and foals/weanlings will often lose a significant amount of weight. Initially after treatment a decrease in serum proteins is often noted before they gradually begin to increase.

A live, avirulent, porcine vaccine (Enterisol, Boehringer Ingelheim) has been used off-label, per rectum, in foals for the prevention of disease caused by Lawsonia intracellularis. A small experimental infection study showed complete protection of vaccinated foals against L. intracellularis. In southern Ontario, the author’s practice has widely-employed the vaccine and has not had a clinical case in a vaccinated foal to-date.

References


The term ‘deformity’ is used when the anatomical structures are normal but are abnormally flexed, extended or deviated. In addition, differential diagnoses to neonatal limb deformities will be discussed, including gait deficits from structural dysfunction, and growth plate fractures.

**Musculotendinous unit laxity**

**Etiology**
Many foals are born with some degree of tendon and ligament laxity.¹

**Clinical signs**
This is more common in the hindlimbs than the front limbs. Visual inspection shows dorsiflexion of the metacarpo/metatarsophalangeal joint and distal interphalangeal joints. In severe cases, the heel bulbs or fetlocks may touch the ground.¹

**Diagnosis**
History and clinical signs.

**Treatment**
Mild cases usually improve within a few days without any intervention. Controlled exercise by keeping the mare and foal on stall rest, and walking the mare can also help. Bandaging should only be employed if it is to protect the palmar/plantar structures of the limb from being traumatised by contact with the ground. In more severe cases, application of a heel extension can support the distal limb.¹

**Prognosis**
The prognosis is good. Most foals are better after up to 5 days of treatment.²

**Congenital flexural limb deformity**

**Etiology**
Some 20% of fetuses have a form of limb contraction.³ Congenital flexural deformities can be centred at the level of the carpus, fetlock, or distal interphalangeal joint. Carpi deformities are quite common, and usually bilateral, though to varying degrees. Fetlock deformities will frequently occur alongside those of the carpus, and occasionally as a sole entity. Congenital flexural deformity of the distal interphalangeal joint present less frequently. Flexural deformities of the distal interphalangeal joint occur from a shorter musculotendinous unit of the deep digital flexor tendon (DDFT) than what would ideally match the longer bone length. Due to the attachment of the DDFT to the distal phalanx, and thus the close proximity to the distal interphalangeal joint, a flexural deformity of the joint presents itself.

A contracture of the fetlock has been associated with the musculotendinous unit of the superficial digital flexor tendon. There is, however, no pathologic condition of the tendon per se, as the common name for the condition, ‘contracted tendons’, would suggest. Furthermore, tendons do not even have contractile units: any contraction must be initiated by the muscle proximal to the tendon and its insertion. The association is therefore in a mismatch of growth between the bone and soft tissue, rather than a problem with the musculotendinous unit itself.

Causes of contracted tendons have not been fully established, but have been theorized to be associated with in utero malpositioning, bone or joint malformations, ingestion of toxic plants or other teratogens by the mare, genetic factors.⁴
Clinical Signs
Various forms can be seen depending on the unit that has a mismatch of growth. When the unit involves the DDFT, the foal appears to walk on its toes, in a ballerina stance. When the SDFT is involved alone, an upright pastern is seen. When a combination of the 2 units is involved, both appearances are seen. Carpal conformation should be viewed from the side. Any cranial deviation from the vertical line composed of the antebrachium, carpus, and metacarpus is considered a flexural limb deformity. 5

Diagnosis
Diagnosis is based on clinical signs observed statically and dynamically. Foals that are unable to walk should be palpated, and the limbs and joints stretched, while assessing range of motion and identification of the structures on the flexor aspect of the limb that are taut. If there is no range of motion, then radiographs are required.

Treatment
Pharmacologic
Oxytetracyline
Goal of using this medication is to promote relaxation of the muscle attaching to the flexor tendon, to allow a greater opportunity to stretch the flexor surface through stretching, physiotherapy and exercise. Intravenous oxytetracyline (44mg/kg or 1-3g in 1L saline, 1 to 3 times in the first week of life) is reported to reduce muscle contractility by chelation of circulating calcium. 5 Administration is not without risk, and severe rhabdomyolysis with acute renal injury has been reported, and renal damage alone has been reported observed at the authors institution. It should not be administered to dehydrated foals, and clinicians advocate analysing renal parameters prior to administration, however, the risk is still present in a normal foal. 6

Sedatives
Sedatives, including alpha-2 agonists and diazepam, can induce some short-term relaxation, and analgesia that can be useful while other procedures such as splinting or physical manipulation are carried out.

Non-steroidal anti-inflammatory drugs
These need to be used carefully in young foals. However, with some moderate to severe contractures, foals will not use their limbs adequately to allow external coaptation or controlled exercise to help stretch the tendon. The use of analgesia can help these treatments to work, and prevent prolonged recumbency from pain which will only make the contracture worse and create a vicious cycle of pain worsening the contracture. Gastroprotectants should be considered in parallel to this use.

Non pharmacologic
Physiotherapy
This can be useful on mild forms that can already be physically manipulated into a correct position. Several techniques can be used. If the foal will stand, with the leg weightbearing, pressure can be exerted by one hand onto the affected joint and help there until the point of maximal resistance and when the foal begins to resent the manipulation. Slow and steady stretching seems to be more beneficial. This can be done for 5-15 minutes, 3-6 times a day. 5
**Controlled exercise**

In general, the mare and foal are placed on stall rest, with controlled exercise if the foal can tolerate it. This can be tailored to the individual foal. Care must to taken not to make the foal more painful than it already is through exercise, as this will exacerbate the problem.

**External Coaptation**

This can take place in the form of splints or casts. In the authors clinic, casts are generally reserved for severe cases that do not respond to initial treatment, and this is therefore rarely in the neonate. Splints are used to support the foal that otherwise would not be able to ambulate or stand for long enough to nurse. Splints can be fashioned out of PVC piping material or made from cast material to mould onto the dorsal or palmar aspect of the leg. Both are placed over a well padded leg with bandage material. A typical protocol would be to place the splints on for 6 to 12 hours and then remove them for 6 to 12 hours, to prevent pressure sores developing. These can be severe in foals, and the resulting pain can make the contracture more challenging to treat.

**Surgical options**

These are rarely required in the neonate and are reserved for foals that do not respond to the above therapies. Inferior check ligament desmotomy may allow for improvement in the distal interphalangeal joint; proximal check ligament desmotomy may allow for improvement in the fetlock joint, sometimes the distal check ligament needs to be transected also. It has been reported that flexural deformities of the carpus may respond to proximal check ligament desmotomy, and/or transection of the ligaments of the ulnaris lateralis and flexor carpi ulnaris.\(^7\)

**Prognosis**

Carpal contracture to an angle of less than 100 degrees that cannot be manually straightened to an angle of 180 degrees, carries a poor prognosis, and euthanasia may be warranted.\(^8\)

When limb prognosis can be initially corrected, the prognosis for return to athletic expectations is favorable.\(^5\)

**Arthrogryposis**

**Etiology**

This is characterized by severe permanent fusion of joints in abnormal positions. There are no known genetic factors, and it may be from a malposition in utero. Some cases have concurrent defects such as wry nose, cleft palate. Foals with hypothyroid goiter have been affected.

**Clinical Signs**

This is a cause of severe dystocia. Mainly carpi, fetlock and hocks are ankylosed, showing complete loss of joint mobility.\(^2\)

**Diagnosis**

Diagnosis is based on clinical examination.

**Treatment**

None. Surgical transection of supporting soft tissue structures does not make enough, if any difference. Foals may be able to tolerate fetlock fusion but cannot be used for any work.\(^2\)
Prognosis
Hopeless. It is reported that some mares consistently produce foals with this deformity.²

Angular limb deformity
Etiology
Only 3% of Thoroughbred foals have correct carpal conformation in their first week of life.⁹ This statistic is not unexpected when considering that neonatal foals have been in utero in a flexed position, and are born have varying degrees of weakness, muscle tone, and periarticular ligament laxity.⁹ Hormonal balances including hypothyroidism have been blamed for some cases. There is also thought to be a relationship between overnutrition of the mare resulting in intrauterine malpositioning. Teratogens, plant toxins, colic, placentitis and mineral imbalances have all been implicated. Premature or dysmature foals are more commonly affected than full term foals. If incomplete ossification of the cuboidal bones is present, they may develop a deformity from uneven pressure on the cartilaginous precursor to the bone, which can be crushed.¹¹

Clinical Signs
Carpal valgus of 4 degrees is considered within normal limits for neonatal foals.¹⁰ Deviations may be obvious but can be complicated but concurrent rotational and flexural deformities.²

Diagnosis
Diagnosis should be made with the foal ambulating and walking toward and away from observer.¹ The dorsal and palmar/plantar aspects of the long bones of the foal should be observed, rather than the front and the back of the foal. Rotation of the limb can make the limb appear as though there is an angular limb deformity when in fact it is normal. Radiographs can be taken when the angular deformity is severe.

Treatment
Foals with periarticular ligament laxity should be treated conservatively with controlled exercise. Resolution is usually seen within the first month of life.¹ Surgical intervention is rarely needed in the first month of life, although foals with severe fetlock deformities should be monitored closely. Glue on shoes can be used if the foal has difficulty ambulating.

Prognosis
This is good with appropriate treatment.

Growth plate fractures
Etiology
Traumatic in origin.

Clinical signs
This will vary depending on the type of fracture. Neonatal foals must be examined for systemic status also, including shock, blood loss, dehydration and hypoglycemia.

Diagnosis
Radiographs. Limbs should be inspected for wounds.
Treatment

Prognosis
Varies depending on age, location, and timeliness of referral

Gait deficits from structural dysfunction
Rupture of common and lateral digital extensor tendon

Etiology
Tendon rupture can occur as a primary problem or secondary to a flexural limb deformity.\(^\text{12}\)

Clinical signs
Focal soft tissue swelling over the carpus, with a resulting knuckling forward of the carpus and fetlock. As a result, the foal is unable to advance forward. In contrast to a contractual deformity, the limb will remain straight when manually placed under the foal.

Diagnosis
Physical examination. Ultrasoundographic evaluation will show a fluid filled swelling within the tendon sheath dorsal to the carpus. The ruptured ends of the tendon can usually be identified.

Treatment
Restricted exercise is essential. Bandaging, including casting and splinting is necessary depending on the severity of the clinical signs.

Prognosis
It is thought of as good but there is little follow-up of this condition in the scientific literature.\(^\text{12}\)

Rupture of gastrocnemius muscle

Etiology
Loss of support along the tension flexor surface of the bone with resultant loss of support of structures.

Clinical signs
Inability to rise, disruption of the reciprocal apparatus, swelling in the caudal aspect of the thigh, instability of the stifle joint, stifle joint effusion.\(^\text{13}\)

Diagnosis
Clinical signs.

Treatment
For mild gastrocnemius injury, exercise restriction via forced recumbency, with minimal or no bandaging, may be sufficient treatment. For more severe disruption of the muscle, limb stabilization via splinting and intensive nursing and monitoring are necessary.\(^\text{13}\)
Prognosis

Moderate to severe gastrocnemius muscle injury is difficult to treat successfully, and the long-term prognosis for athletic function should be regarded as guarded.¹³

5. Earl M. Gaughan, Flexural Limb Deformities of the Carpus and Fetlock in Foals, Veterinary Clinics of North America: Equine Practice, Volume 33, Issue 2, 2017, Pages 331-342,
POST-CASTRATION COMPLICATIONS
Nicola Cribb, MA, VetMB, DVSc, Diplomate ACVS
Assistant Professor, Large Animal Surgery, Ontario Veterinary College

1. ACUTE COMPLICATIONS

Surgical decision-making.
There are several choices that can be made prior to starting a castration surgery, and they may or may not impact on the complication rate. They include:

i) Standing vs GA. An obvious advantage of a standing castration is it obviates the risks of general anesthesia and recover. However, studies have shown an increased occurrence of complications following standing castration.1,2 One recent study of standing castration found 60% of Thoroughbred racehorses undergoing a standing open castration had complications.3

ii) Use of antimicrobials or not. Use is based on clinician preference. Two studies have shown that most respondents 90% and 97% use antimicrobial prophylaxis.4,5 A study in 1995 of 23,000 castrations showed that most complications were as a result of inflammatory and/or infectious processes and significantly more infections occurred in horses that were not given antimicrobials (4% vs 2.9% respectively).6

iii) Use of emasculators. The serra emasculators, which crush and cut at the same time, and the reimer emasculators, which crush only, are the 2 most commonly used. A higher rate of hemorrhage was reported by respondents of a survey following use of the Reimer emasculators.6 However, an ex vivo study showed that during an open castration the contrary was seen: the Reimer emasculator resisted higher pressure.7 No differences between the Reimer and Serra emasculators were seen during a closed castration. The Henderson equine castrating instrument has a similar complication rate to traditional emasculators of 10%, as seen in one study.8

iv) Use of ligatures. One did not find any advantage in using ligatures as part of the castration surgery.9 Another study that did not use ligatures in 95% of horses found that that overall rate of complications from hemorrhage was low, and the authors suggested that ligatures may not be necessary to prevent post-operative hemorrhage. This author places one transfixating ligature of No.2 polyglactin 910 on all castrations.

Intra-operative complications
Most complications occur in the post-operative period. Inability to identify a testicle can arise from cryptorchidism, or poor positioning of the genital structures. If both testicles cannot be identified while the horse is standing and sedated, sometimes they will become apparent once the horse is anesthetised in dorsal or lateral recumbency. A testicle that is in the low inguinal region can sometimes be more easily exteriorised into the scrotum following injection of 10ml of lidocaine into the body of the testicle. Care must be taken not to confuse a testicle for a penis, and cause iatrogenic penile damage.10 Testicles that cannot be found in the inguinal or low scrotal region need an alternative approach for retrieval. In the event that no testicle can be found despite more invasive and or laparoscopic techniques, castration of the descended testicle and testosterone testing as soon as 24 hours post-operatively can determine whether the horse is a true monorchid.

Post-operative complications
a) Hemorrhage. Some slow dripping of blood can occur for several hours following castration from minor vessels in scrotal skin or subcutaneous tissues, or tunic. More significant bleeding can occur from vascular structures in spermatic cord that were not adequately crushed/ligated. If bleeding severe fluids and whole blood therapy may be required. Occasionally bleeding distal tip of spermatic cord can be identified in standing horse. If so, apply clamp and leave in place for 24 hours. Occasionally bleeding can be controlled by packing scrotum with gauze and closing scrotal skin with towel clamps or suture for 24 hours. Usually general anesthesia and exploration required to ligate cord. Make incision over superficial inguinal ring if cannot identify cord.

This author has used formalin in horses where repeat surgery is not an option. 10ml of a 4% formaldehyde solution diluted in 1L of saline IV. 11 Horses can also bleed into the peritoneal cavity of the transected cord has passed though internal inguinal ring into the abdomen. Ultrasonographic monitoring of fluid levels in the abdomen, and monitoring of the hemodynamic status of the horse can help determine the need to transfuse blood. Up to 75% of red blood cells lost into a body cavity (eg peritoneum) are autotransfused back into the body within 24 to 72 hours. 12
b) Omental prolapse. Omentum from abdomen may pass through vaginal ring and scrotum. Inspect omentum and perform rectal examination to ensure that bowel has not passed through also. If only small amount of omentum options are to emasculate with the horse standing, stall rest/cross-tie for 48 hours, antibiotics to prevent ascending infection. If significant omentum is present, refer to surgical facility. Close scrotum with towel clamps or suture so further omentum does not prolapse. The horse placed in dorsal recumbency, and may need an incision over superficial inguinal ring to ligate the omentum, and close the superficial inguinal ring.

c) Evisceration. This is a rare but potentially fatal complication. It can occur immediately after surgery or up to 7 days post-surgery. Emergency treatment involves preventing further evisceration: sedate horse, place intestine within scrotum and close with towel clamps or suture. Antibiotics, and NSAIDS are required and referral to a surgical facility. A 50% prognosis for survival depending on degree of contamination and viability of bowel is given.

2. DELAYED COMPLICATIONS
a) Excessive swelling. This is a common complication. Mild swelling can be normal and should resolve within 10-14 days of surgery. Swelling that does not resolve, is excessive, associated with discharge or foul-smell or lameness requires treatment. It is usually due to lack of drainage and requires the incision to be opened up, antibiotics, NSAIDs, exercise, hydrotherapy at a low pressure so to not push any bacteria into deeper tissues.

b) Infected cord – scirrhous cord. This is characterized by a lack of drainage, purulent discharge, swelling. It can occur months to years after castration. Small multi-focal abscesses can appear in cord creating a lot of fibrous tissue surrounding it. It is usually caused by *Staphylococcus* and *Streptococcus* species.
If caught early, antibiotics, NSAIDs and ventral drainage can be effective treatment. If there is a chronic infection, surgical resection of affected portion of cord is required.

c) Unaltered behaviour. Stallion-like behaviour can persist if it is a learned behaviour (more likely in older horses or those that have been used for breeding). It occurs in 20% of these horses. It can also occur if there is remaining testicular tissue – only usually occurs if epididymis is mistaken for hypoplastic testicle during cryptorchid castration, and testicle is not removed.

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Practice Management Program
Inclusivity Above All

Kemba Marshall, MPH, DVM, DABVP (Avian), SHRM-CP
Marshall Recruiting Consortium
St. Louis, MO

Whether we look around at conferences or association meetings or read labor and statistics reports it is clear that veterinary medicine is one of the least diverse professions. The data also support the benefits of diversity beyond simply being the right thing to do, benefits such as increased team morale and increased profitability. As we consider ways to increase diversity within our workplace, we must first pause to ensure our workplace is an inclusive, welcoming environment. While striving for diverse teams ensures employees with a wide array of backgrounds. Inclusive environments strive to ensure every feel equally welcomed and valued. The Kellogg School of Management at Northwestern University explains it this way: “Inclusion is about welcoming, developing and advancing a diverse mix of individuals. It’s about making people feel valued, including changing practices that might unfairly benefit any one group and making sure that everyone feels they have the same opportunity to advance and make an impact. Creating that environment is where the real challenge lies.” No matter what your role is within the company you can impact inclusivity in a positive manner. Before we discuss your role, let’s examine the look of inclusive environments.

What do inclusive veterinary environments have?
1. Educated leaders who appropriately set the tone. Leadership around any practice starts at the top. Inclusive veterinary spaces have leaders who understand the importance of inclusivity and make a habit of being inclusive. Leaders spend time with team members across all levels of the organization and have transparent dialogue about all aspects of the business including financial performance and company culture. Inclusive leaders solicit the employee opinions and are active listeners. Inclusive leadership combines both informal and scheduled touch bases. Inclusive leaders explain why inclusivity is important to both internal stakeholders (staff) and stakeholders (vendors and clients).
2. Inclusion councils where leadership works with managers or supervisors who are committed to inclusion to set recruitment, hiring, retaining and advancement goals. These councils should be diverse and represent various ages, genders, roles and employment tenure.
3. Cultures of celebrating employee uniques. International and non-Christian holidays are observed. Employees should be given floating holidays to accommodate significant days when the work site is operational, but the employee chooses to take the day as a personal holiday.
4. Active teams who feel heard, valued and respected. Managers have one view of work but employees have another view. Both are valid and should be listened to. Employees who feel included feel safe enough to provide candid feedback. This feedback should be received with gratitude as a means for continual improvement. There should also be an anonymous way to provide feedback to management.
5. Assess the physical environment to identify ways to increase inclusion. A mother’s room and prayer/meditation room along with gender neutral restrooms can increase inclusion of the physical space. Accessible Canada Act improvements will also demonstrate inclusivity.
6. Partner with a Human Resources professional to look for ways to make company policies and procedures more inclusive. Patient forms and client handouts being available in multiple languages is a way to show inclusion. Looking around the physical and online worksite can reveal that imagery can be updated to reflect the larger society. Policies and procedures can be evaluated for inclusion. This can be as simple as what times meetings are held or if company meetings can be hybrid.
7. Inclusive practices communicate goals and measure progress.

18-Question Workplace Inclusivity Self-Assessment this assessment can be used at anytime during your effort to make your work site more inclusive. Ask yourself each of the following questions. You are not going into great depth; you only want yes or no answers. You will see some of these items are easy to address. Take pronoun clips as an example. Pronoun clips can be attached to stethoscopes and signal inclusivity. It is important to note that pronouns are not to be described as “preferred”. Pronouns should be referred to as the individual intends them to be. Just as you would not intentionally mispronounce someone’s name as you welcomed them to an event you should
make every effort to always use their pronoun of choice. There are immediate and low-cost things that can be done to increase inclusion in the physical environment and on the walls. Other items will require a larger investment of time or money, so it is helpful to identify short, mid and long term goals to improve inclusivity. No matter where your worksite is today there is always room for improvement.

1. Does your work site have publicly posted anti-harassment and anti-discrimination policies?
2. Does your work site provide anti-harassment and anti-discrimination training?
3. Do you know what to do if you observe harassment or discrimination at your work site?
4. Does your work site celebrate diverse holidays?
5. Do pictures on the walls of your work site mirror a diverse society?
6. Do pictures on your company website mirror a diverse society?
7. Does your work site have gender neutral restrooms?
8. Does your work site provide client materials in languages beyond English?
9. Are team members encouraged to display the languages they are fluent in?
10. Are client forms inclusive?
11. Are client forms gender neutral?
12. Is feedback used and received as a gift in your workplace?
13. Is mentoring available for all team members?
14. Are applicants recruited from diverse sources?
15. Are inclusive symbols displayed throughout your workplace?
16. Is your work site Accessible Canada Act (ACA) compliant?
17. Does your work site have Employee/Associate Resource Groups (ERGs/ARGs)?
18. Does your work site offer medical benefits for same sex spouse or domestic partners?

Does the work of inclusivity vary by your role in the organization? Yes, it does. But every role in the organization can play an active role in creating inclusive spaces.

Managerial ways to be more inclusive.

1. Hire for talent not a resume. Inclusive managers are well aware of their own conscious bias. Unconscious bias is that nanosecond decision making ability that allows us to do many common things without thinking. We close our garages, turn off our alarms, we even commute to work without really being aware that we did. Unconscious bias also causes a preference for sameness. Unconscious bias can cause us to value a profession, university or geographic area as better or worse when we center ourselves as the norm or ideal. In a hiring situation unconscious bias can assume that a degree from a prestigious university makes a candidate more capable than a person with a degree from a lesser-known university. This is not to be confused with a lowering of standards, this is an expansion of talent by looking into untapped talent marketplaces. Inclusive managers bring creativity and innovation to the talent hiring process before they even meet their candidates.

2. All creativity is unleashed. Traditional leaders can be very rank and file, with preconceived notions of the proper way to nurture creativity. There may be various channels the creative idea has to be routed to which means many potential stops where the creative idea can be nixed. Inclusive leaders require innovation and creativity across all levels by not providing rigidity. Inclusive leaders hold to the mission and vision of the organization and invite dialogue about everything else. Inclusive leaders see no correlation between the employee’s title and the value the employee brings.

3. Use opportunity as your primary development tool. Inclusive leaders create cultures where anyone can contribute to important outcomes. Inclusive leaders have a core belief that the people they hire can and should do anything. Inclusive leaders believe that team members should develop in new directions throughout their careers.

4. Foster competition and collaboration-being intensive and nurturing. We tend to be competitive by nature so we can use that to foster inclusivity. Employees can have contests to develop internal inclusive onboarding or outreach programs. Veterinary medicine is a common career choice for young people; career exploration activities are a great way for veterinary professionals to share our career journey with diverse audiences. Such competition paired with collaboration also generates a cohort effect when it comes to talent: The more you help people become better, the more they help one another get better. By mobilizing both collaboration and competition, inclusive leaders create an ecosystem in which ideas collide, prompting new ideas to arise. Embracing and integrating these four practices might require that you unlearn old habits. While we
often praise leaders for how much they know cultures of inclusion require humility. Inclusive leaders declare that they do not know everything. Inclusive workplaces are on a continuum. There is not a time when leaders can declare the workplace inclusive and stop working at inclusion. Individuals who grasp inclusive leadership the quickest are those who display the greatest curiosity and courage (traits that others have regarded as integral to inclusive leadership). If you don’t feel that you possess an especially curious or courageous personality, never fear — you can learn these traits. One place to start is to make a conscious effort to part ways with the egotism that runs rampant in corporate environments. Give up your need to be “the expert.” Practice asking more questions (and intentional listening), even if you fear looking foolish.

Deloitte Australia polled inclusive leaders from Australia, New Zealand, Singapore, Hong Kong and the U.S. to assemble a list of six traits of inclusive leaders. Inclusive leaders can access a broader spectrum of perspectives and ideas which yield improved decision-making. This improved decision making allows for an ability to anticipate the future, handle uncertainty and innovate. These leaders not only promote diverse teams but also increase their capacity to innovate and deal with uncertainty. These leaders are able to effectively engage with a wide range of attitudinally, demographically and culturally diverse stakeholders.

Six Traits of Inclusive Leaders:

Cognizance. Personal and organizational bias is a leaders Achilles’ heel. These are the blinders we put on as we attempt to make the best decisions with the information, we have available. Blinders narrow our field of vision and often preclude us from making objective decisions. Inclusive leaders try to identify their own biases and learn to prevent these biases from influencing talent decisions. Inclusive leaders seek to implement policies, processes and structures to prevent organizational biases from stifling diversity and inclusion. Without continual efforts to remove unconscious bias, inclusive leaders understand that natural inclinations take us towards self-cloning. To operate successfully in today’s economy a different approach is required.

Collaboration. Because a diverse thinking team is greater than the sum of its parts inclusive leaders understand that team members must first be willing to share their perspectives. Leaders enable environments in which all individuals feel empowered and express their opinions freely with the group. Inclusive leaders realize that effective collaboration requires diversity of thinking. To this end they pay close attention to the team’s composition and processes. Inclusive leaders develop one team identity through shared goals. Inclusive leaders ensure team members value each other’s knowledge and capabilities.

Commitment. Cultivating a diverse, inclusive workforce requires time and energy. With all of the demands on our time and energy staying the course is hard. Inclusive leaders not only understand the business imperative of inclusion but also have a deep-seated sense of fairness rooted in personal experience. Inclusive leaders believe they create a welcoming culture and have a strong personal responsibility for change. The devotion of time, energy and resources to nurture inclusive workforces takes an investment in people. What does this investment look like, inspiring others to share their passion and goals. This investment signals true commitment.

Courage. Our associations, organizations and business norms have attitudes and practices that yield homogeneity. Inclusive leaders challenge these entrenched attitudes and are willing to be seen as culturally or politically unpopular. Inclusive leaders also demonstrate courage because talking about limitations and imperfections (which we all have) involves personal risk-taking. Inclusive leaders have the courage and humility to seek contributions from others to overcome limitations and imperfections. Some leaders find it difficult to admit they do not have all of the answers. In actuality this is stating the obvious, none of us have all the answers.

Cultural intelligence. Because not everyone sees the world through the same cultural frame inclusive leaders change their styles to fit cultural norms. This may mean that extroverts have to be more introverted with groups that value more modesty and humility. This may mean that introverts will have to channel extroverted energy when the occasion calls for an out loud, celebratory delivery of information. Inclusive leaders understand their own cultural norms and how those impact their worldview and expectation of others. Inclusive leaders understand cultural stereotyping and how to move past it.
Curiosity. Inclusive leaders are life learners, open-minded and have a desire for exposure to different ideas. Inclusive leaders understand that different ideas and experiences enable growth. They solicit various viewpoints to minimize their blind spots and improve decision making. Inclusive leaders engage in respectful questioning, actively listen to others and synthesize a wide range of ideas. Inclusive leaders ensure those around them feel represented, valued and respected. Because snap judgments can stifle ideation and are frequently biased inclusive leaders refrain from making fast judgments.

What about those of us who do not hold leadership positions? Yes we have our role in creating inclusive work environments as well. We often refer to ourselves as teams but what do inclusive team members do?

1. Teammates help each other out and provide instrumental assistance. Peers can provide information, make introductions to key contacts, verbalize support for someone else’s ideas in meetings and offer advice.
2. Teammates take care of each other emotionally. This looks like the laughing, socializing and bantering we do. This looks like the snacks we bring into the clinic when we know the schedule will be busy for the day. This looks like personal check ins or the reassurances we offer when teammates share the ebbs and flows of life they are experiencing.
3. Teammates show up for one another. These are subtle gestures of caring like walking out to the parking lot together after our shifts are over. During company meetings we sit next to one another and we go out of our way to welcome new team members into the group. Even on virtual meetings this looks like turning our cameras on and overlooking the pets and humans that inadvertently pop into view during meetings. We come by to put one more hand on the wiggly patient or grab that pen or tape that is just out of reach.

Peer to peer interpersonal inclusion is psychological as you feel noticed and valued by your teammate. It also boosts individual job performance, team effectiveness and overall morale. Each act strengthens the feeling of inclusion, demonstrates the physical presence of someone lending a hand and provides emotional support. These actions can and should be done by everyone on the team. It is also contagious, seeing someone lend a hand or showing support encourages you to do the same. Team members who are included are also motivated to come to work each day. Included individuals have high degrees of psychological safety and share information readily. This translates into faster problem solving. Once the problem has been solved we can all move onto the next problem. Inclusive practices by peers allow us to see the broad range of skill sets others possess. Employees can be presented with special projects and stretch assignments that tap into their unique skill sets. The sum total of peer inclusion is that employees are retained and allowed to grow professionally.

In inclusive environments both leaders and peers have the ability to become allies and upstanders. Allies use their position or power to advocate for others who do not hold the same or position or power. Upstanders are active participants in shaping the culture of any workplace. Upstanders assist their peers when something is awry as opposed to bystanders. Bystanders stand idly by and watch as things are not going well. Here are a few examples of what can go wrong in the workplace and how inclusive leaders and peers may address them. Just as we can all imagine inclusive acts we can all visualize exclusive actions. We have seen team members struggle to complete a task and no one provide assistance. We have seen the great divide in meetings where one person always seems to sit off to themselves. We have seen the one co-worker walk to their car alone after their shift because everyone else walked out together and forgot there was one more person in the building. We have seen in groups go out to lunch or order lunch in for a few. When lunch arrives it then becomes very obvious not everyone was asked if they wanted to order food.

Now that we have talked about the benefits of inclusion, we should discuss the dangers of exclusion. Without inclusion, team members have a false sense of security; like walking across a tight rope. Exclusion removes psychological safety. It is not uncommon for team members who are always off to themselves to be frequently left out of informal gatherings like lunch. Without a sense of inclusion these individuals withdraw and stop contributing to meetings, voicing their opinion during problem solving exercises or fully contributing in their roles. Diversity without inclusion can also risk tokenism. Tokenism occurs when an individual is brought into a work setting to “check the box” and say someone from a diverse background was hired. It is one thing to hire someone and a completely onboard that person so that they are welcomed and function with all rights and responsibilities as a team member. If
you have brought in an individual and that person is unable to fully contribute you miss out on the benefits of diverse team problem solving. When individuals do not feel valued, respected or included they leave. Increased staff turnover then causes a cascade effect where remaining employees have to do extra work while the role is backfilled. This constant churn of associates has detrimental effects on the overall morale and productivity of the business.

While diversity and inclusion are important and are best practices the first step is to really assess how well your current employees are included. If you want to know how your employees feel, ask them. This should be approached carefully so that employees are safe to share their opinions. Every business is looking for maximum productivity and efficiency from associates. Allowing employees to be radically candid in describing their work experience is how improvement begins. A true assessment will reveal what is working well and what can be improved. This is a great way to be inclusive. Often leaders think they have to be the ones to do all of the doing. Using an inclusion council (which has diverse representation of your current team) you can begin an assessment. Remember assessments should have multiple feedback modalities. Employees should be allowed to submit handwritten or electronic feedback. Feedback should be able to be anonymous.

The most important aspect of feedback is that results be broadly communicated. Once the results have been communicated there should be a plan for addressing the top three issues raised. There should also be a plan for continuing the top three issues that work well. Bringing the team together to make veterinary work settings more inclusive in and of itself furthers the work of inclusion. As the workplace continues its inclusion efforts, surveys, feedback and consequent actions can be part of the makeup of valuing, respecting and listening to associates.

In conclusion we will end as we began with hallmarks of inclusive workplaces and invite you and your team to begin or continue your journey with this list:

1. Leader education.
2. Form an inclusion council.
3. Celebrate team member uniques.
4. Listen to employees.
5. Look for environmentally inclusive projects.
6. Hold more effective meetings.
7. Communicate goals and measure progress.

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It’s one of the most commonly used terms in business and personal settings, innovation. It’s something to be strived for in the arts, in sports and in service delivery to our pets and our clients. But what exactly is innovation? Wikipedia defines innovation as the practical implementation of ideas that result in the introduction of new services or improvement in offering goods or services.

During this presentation we will narrow our focus to personal innovation and my term for that is InYouVation; innovation is in you. We will discuss the five building blocks of innovation which are very similar to business model innovation. The eight barriers to innovation will also be discussed. Using tips and tools for overcoming barriers we will complete actionable, personable roadmaps. We will use a business case, Research In Motion (RIM) and the iconic Blackberry to compare and contrast our personal innovation roadmap with business innovation best practices.

A brief history of RIM. In 1999 several events happened that forever changed the world. Europe introduced the Euro, SpongeBob SquarePants made its international debut, global heartbreak unfolded during the Columbine mass shooting and the Blackberry was introduced to corporations around the world. Blackberry was the first device that allowed a user to see emails on a pocket-sized device and calendars to be updated real time as meeting logistics changed. Blackberry provided an always on connection to Microsoft Outlook. Blackberry’s board of directors were friends of the company’s co-founders Mike Balsillie and Jim Lazardis and the board provided little to no technical expertise. Blackberry was originally available only to corporate and government enterprises. Blackberry sought long contract periods and coveted the corporate IT market who relied on server-based solutions. A darling of Wall Street Blackberry’s stock price in 1999 was $1.50/share. At its pinnacle in 2007 the stock price was $230.00/share. In 2007 Google came on strong in the personal consumer mobile phone market with the advantage of simplistic touchscreen technology and cameras built into phones. While Blackberry thought their unique offering was the technology of accessing email it turns out the end user wasn’t focused on the Blackberry operating system but rather the convenience of accessing email away from the office. Once mobile phone capability was merged with email access and cameras, Blackberry and its clickwheel was quickly outmaneuvered by iPhone and Android touch screen cell phones.

In less than ten years Blackberry went from the most admired to most irrelevant personal device on the market. Blackberry was led by co-CEOs who had once been praised for their diversity. The proprietary technology and business operations were championed in the C-suite by two very different individuals. The building blocks of business and personal innovation are: a unique purpose statement, awareness of your strengths, a degree of risk tolerance, collaboration and a dedication to life-long learning. We will look at the five building blocks of personal innovation below in greater detail and augment them with lessons from Blackberry’s rise and fall.

1. **Craft a purpose statement**- Just as businesses have purpose statements individuals should have a purpose statement. Purpose statements should be expansive in nature. Looking at our example of Blackberry who existed to allow corporate employees to have
mobile access to Microsoft Outlook, we do not want a purpose statement that is so narrow that we render ourselves obsolete. Blackberry’s mission statement was, “To be the world’s leading provider of end-to-end mobility solutions that are the most secure and trusted.” Blackberry focused on Microsoft Outlook not the ability to place a phone call. Immediately their competitors found a niche by reading Blackberry’s purpose statement and comparing that to their business strategy. Your purpose and the focus of your purpose statement should be personal to you. There are no right or wrong statements. These statements should also be fluid and reflect where you are now while giving yourself room to grow into your next phase. If you are an early career professional with 1-3 years of experience, your purpose now will look different than it will as you become an established professional with 7-15 years of experience. Your purpose may change as you reach an advanced career phase where you have more than three decades of professional experience. Your purpose and focus will also undoubtedly be impacted by your personal life. Are you starting a family with your partner, raising children, caring for aging parents or serving as a military spouse? Your purpose statement should be holistic, reflecting all of you while allowing for growth and development. I personally have found the Six Word Story Exercise to be very helpful as I contemplate my purpose statement. My six-word story is that “I Inspire Others to Inspire Others”. There are multiple ways to begin the work of your purpose statement; they all require quiet contemplation and self-awareness. The following questions may be helpful as you begin your introspection:

- Why do I exist?
- What matters most to me?
- What do I value?
- How do I want to be remembered?

2. **Identify your 3 Uniques** - Veterinary professionals tend to be high achievers. This can mean glancing over our strengths and dwelling on our “needs improvement areas”. Hard work, persistence and dedication could likely be listed as strengths by all who have undergone the rigors of advanced medical training. Now ask yourself how these strengths add value to the world around you? Veterinary professionals tend to be very aware of global issues and action oriented. This can make it daunting to try and understand how we are uniquely suited to impact the world. The reality is we may not impact the entire world. There is a world around us that we can impact. That world is comprised of your family, friends, peers and clients. How are you uniquely suited to impact those that you see daily? On the go email/calendar/Microsoft word access was incredibly unique when Blackberry first entered the market. The security of the Blackberry device earned RIM steady government purchase orders, that was incredibly unique in the early 2000s. The fact that Blackberry devices were initially only available to corporate and government employees made the devices rare and unique. Blackberry did lay the groundwork for the creation of the mobile device and that did in fact change the world.

3. **Take risks** - There have been many articles written about Blackberry, perhaps because RIM’s fiscal performance was such a rise and fall or because many who started their careers at RIM continued on in the business world and are frequently asked their opinions. There was a group at RIM who wanted to license the Blackberry software technology so that it could be used with non-Blackberry devices. There was another group that wanted to continue to couple the Blackberry devices and keep the software
proprietary to Blackberry to ensure additional hardware sales. Sometimes giving voice to your opinion can be seen as taking a risk. Innovation is an action word it is the process of implementing something new or improving an existing process. Innovation requires a level of risk tolerance. If you are going to do something new it means you have to stop doing something old. Risk tolerance is not the same as wantonly taking an ill-advised risk. Risk tolerance means gathering information from multiple sources and then demonstrably committing to your purpose. Risk tolerance means you have looked at the pros and the cons and in the long run the action you are about to take is worth the risk. There is no growth without change. Even if you are not questioning what you can be doing better, someone else is questioning that. A personal advisory board can serve as a source of information for you. Any advisor must be willing and able to help you question what you could be doing better and what risks you should be considering. Those of us who take innovation seriously must be willing to listen to what our advisors have to say.

4. **Collaborate diversely**-RIM drew inspiration from Sun Tzu’s “The Art of War” in their approach to business negotiations and treated partners like Sprint, AT&T and T Mobile as adversaries. RIM also believed the security of the Blackberry device was unparalleled. RIM would face serious economic challenges and had no partners to rely on for support or loyalty. When iOS and Android devices needed security software, they partnered with companies like Mobileiron, Airwatch and Good Technology to bring in additional expertise. While iOS and Android did not have in house security competencies they sought out partners who did have security as a core competency. Diverse collaborations have been proven again and again to pay off for all collaborators. When considering the diversity of your personal and professional networks you should ask yourself if everyone you are collaborating with looks like you, thinks like you and has a similar life experience. If the answer is no you are likely not approaching innovation holistically. In his book, The Diversity Bonus, Professor Scott Page uses models and data to prove that the collective performance of teams who think differently includes a diversity bonus, an extra amount. Diversity bonuses are enabled by policies and protocols including the individuals and teams you collaborate with. No one has all the pieces to the puzzle. Our job as innovators is to find the missing pieces to our innovative ideas in others through collaboration.

5. **Embrace lifelong learning** once the founders of RIM developed the first Blackberry they went from being innovators to fast followers. Lazaridis and Balsillie assumed that corporate users would reject the camera phone feature, only to eventually yield and include a camera feature on Blackberry devices. In response to the iPhone with less buttons and unnecessary features Blackberry included more buttons and features. RIM thought users wanted the Blackberry operating system when customers by their purchases signaled they wanted more options to text, send emails and make phone calls. Lifelong learning professionally and personally can come from any source. Professionally we likely have continuing education meetings that we attend annually. Perhaps it is time to shake up the routine and attend a new conference or learn a new skill that benefits your practice. Personally lifelong learning can come from your hobbies. If you enjoy museums, long Sunday drives, horseback riding, swimming or yoga be an active participant in these activities. Being a lifelong learner means making time for the personal you. For many of us lifelong learning is tied to our work-life balance. As stated earlier one of the most important building blocks for innovation is time.
devoted to it. Twenty minutes a day dedicated to learning something new or researching a
topic which interests you is one way to embrace lifelong learning.

Now that we have covered the building blocks of innovation let’s talk about what gets in the way
of personal innovation. There are 8 common barriers that face us all read on to see how we can
overcome these barriers.

Innovation is about a systemic, cultural dedication to the propagation of ideas generated by
creative individuals. Free space to put your potent mind to work. Culture directly influences an
individual’s potential for innovation. Gaia Grant, from the University of Sydney Business School
advises that psychological and environmental blockers have to be identified to encourage
innovation. She advises we ask ourselves two questions: “What is stopping me from
innovating?” and “What can I do to address the obstacle?”

1. Closed-mindedness - a reluctance to take in new perspectives is always a barrier to
innovation. To paraphrase a quote from Albert Einstein, problems can never be solved
in the same mind that created them.

How can closed-mindedness be addressed? -collaborate with others internally and
externally. 80% of innovations are sparked by someone whose primary expertise is
outside of the field of the breakthrough. According to the Price Waterhouse Global
Innovation Survey, the most innovative companies collaborate with external partners
three times (34%) more often that the least innovative companies (10%). Since
companies are made up of people it stands to reason that the most innovative people
are highly collaborative.

2. Isolated ideation - In addition to being able to see things from the point of view of others
one must be willing and able to fully explore other modes of thinking. Take into
consideration information from all sides.

How can isolated ideation be addressed? - Crowdsourcing and mind mapping-
collaboratives for idea generation. Seeing examples of ideas generated by others can
improve one’s own ideation outcomes. See the full range of creativity and diversity of
other ideas. Mapping mechanisms allow you to see similarities and link the ideas.
Harvard and Carnegie Mellon research shows that people presented with creative
elements generated more creative ideas than those who just saw random ideas. We
can immerse ourselves in hobbies and creative group activities to spur our innovation.
Many retailers have a practice of comparative or comp shopping where cross-functional
teams visit local stores to see merchandising displays. In veterinary settings this can be
approximated by having teams attend CE meetings and visit exhibitor or trade show
booths together.

3. Sensory overload - More work and more to-dos don’t make you more productive. You
have to have time to breathe and think. You can be piling too much on your plate (or
onto your employee’s plates).

How can sensory overload be addressed? -Organize and prioritize tasks. It is also
helpful to learn to say no and not feel guilty about doing so.

4. Lack of follow through. Lots of potentially innovative ideas stay stuck at the water
cooler because people lack the drive or motivation to see things through to the end.
Always follow up on any valuable ideas you may. The author frequently makes lists and
records voice memos to capture ideas when I am in the middle of doing something else.
How can lack of follow through be addressed? - Invite others to collaborate and provide feedback. People don’t like to be sold; they like to be invited. Strive to follow up immediately if a discussion or meeting sparks an innovative idea so that the conversation continues. Be patient and realize great ideas are rarely fully-formed in the eureka moment. Your idea may need time to mature. Incubate it with other innovations. A personal advisory board can be a safe space to share ideas and solicit feedback. Your advisory board should represent different thinking styles, different ages, different professions and different lived experiences.

5. Blind allegiance to tradition- “It’s the way we do things around here”. You have to be willing to let go of old company policies, processes or organizational structures that are not working anymore. How can blind allegiance to tradition be addressed? - By establishing a virtual collaboration environment. What can be relaxed, changed, updated or eliminated to allow ideas to flourish? With so many virtual meeting options, collaborations can take place internationally.

6. Goals that are too big or too distant- Real innovation requires a mix of short, medium and long term goals. Innovation often appears daunting because you are trying to solve very complex issues. Big, audacious, hairy goals (BHAGs) grab headlines but they can be impossible to execute. This can be discouraging for high achievers like veterinary professionals who are accustomed to success. Innovation can be managed by breaking it down into steps. How can goal setting be addressed? - SMART goals are the key. Goals should be specific, measurable, achievable, realistic and time bound. A big or distant goal may be “I am going to exercise every day for the next year”. A SMART goal may be “For the next thirty days I will exercise for thirty minutes three days each week.”

7. Do-it-yourself-ing. More often than not innovation requires teamwork which requires a support system that you communicate with. How can do-it-yourself-ing be addressed? - Here is your time to make an investment. Meeting new colleagues, mentoring and volunteer activities are a great way to meet others. We often feel compelled to go it alone because we don’t think others will understand our innovative idea or take it seriously. Meeting new people and spending time investing in those new relationships can be a great way to find a new innovation accountability partner. You can up your odds of success by meeting someone while you are taking a class or attending

8. Poor resource management- Innovation requires investments of time and energy. Additional investments may be technology and/or capital funding. How can poor resource management be addressed? - By taking an accurate count of the time and money you have to invest in innovation and deciding how to make the best use of your resources. You should also anticipate setbacks. Allow more time and energy than you think your innovation will require.

To some degree we all innovate. We identify problems and attempt to implement root cause solutions. Innovation, to some degree can be a learned mindset with a road map and practice.

How to Create an Innovation Road Map
1. What is the problem your innovation solves? Try and visualize the problem from multiple points of view. Also explain the problem to others. Explaining a problem aloud gives you and the listener a way to identify different points of view. Your solution doesn’t have to be unique, but it does have to be better.

2. What steps do you need to take in order to begin your innovation?

3. Prioritize the steps you need to take and assign deadlines.

4. What milestones will you use to monitor your strategy?

5. Develop a list of new aptitudes and attitudes your innovation will require you to acquire.

6. Build soft and interpersonal skills to complement your problem solving and analytical skills.

7. Visualize your action plan. Create a mental sketch of your life as your innovation begins.

8. Monitor your action plan, evaluate your progress and update your plan as needed.

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People, organizations and corporations love initiatives. Great you say, but what are initiatives? Initiatives are projects which are used to introduce a new value or achieve a key strategic goal. Product Management Institute defines a project as a temporary endeavor undertaken to create a unique product, service or result. Initiatives can be small or large. Notable examples are personalizing Coca Cola cans with individual names on them to personalize the look or building the London Bridge as a means to enable individuals to cross the river while ships sail the river. LinkedIn.com What is an Initiative and What is Initiative Management? Ali, M.A.A. March 22, 2020. What could go wrong with an initiative? It can go from temporary to never ending, it can lack a clear goal or business purpose. No matter the size, all initiatives take time and money and someone to do the doing. These means initiatives can wind up being very costly. Initiatives also have an emotional investment, someone has proposed a project idea and believes it to be a good idea. Being a good idea is not enough for a successful initiative. We will discuss the importance of prioritizing initiatives as a component of effective leadership.

Let us begin by discussing seven common reasons why prioritization fails:

**Impact blindness** - Leaders know the actual costs to develop their initiative(s). Leaders are often oblivious to the total number and cumulative impact of current initiatives. Managers and employees are expected to do this work without systems to identify, measure or manage the demands caused by initiatives. Even if the impact can be measured it rarely can be assessed across the organization due to the volume of the initiative, company size and/or company complexity. Managers and employees however do feel the impact of the pressure of expectations to deliver all initiatives. Initiatives are always in addition to tasks required to run the business from day to day.

**Multiplier effects** - Many organizations leave teams to plan their own departmental initiatives without a clear line of sight to other departmental initiatives. Departments who have evaluated the initiative’s impact internally rarely have the capability of measuring the impact of their initiative when combined with all other company initiatives being run. At a certain point there are simply too many things to do. Each initiative requires an additional step and those steps add up. The downstream impact of current initiatives in place is rarely measured at the leadership level.

**Political logrolling** - This termed was coined by the US Congressman Davy Crocket and is named for the tradition of neighbors traveling to each other’s homes to assist with the process of rolling their logs. In organizations it looks like one leader getting their initiative to continue by promising to help another leader keep their initiative rolling. These can lead to eternal projects because leaders have multiple funding sources and the influence to require that an initiative live on even if official project dollars or dedicated head count are no longer available.

**Insufficient Resources** - This is known also as an unfunded mandate in governmental terms. A project can be required but lack head count or funding for implementation. Managers always have important goals but may lack resources to accomplish those goals. In this situation the manager has to decide which initiatives and tasks get completed and which ones do not.
**Band-aid initiatives**-When patch work improvements are applied to complex situations the result may be, you guessed it, additional initiatives. None of these proposed projects may deal adequately with the root cause. Individuals assume they know what the problem is. Band-aid initiatives stem from lack of awareness about what the real problem is.

**Cost Myopia**-This is a partial, short-sighted fix. An initiative to reduce spending results in layoffs or hiring freezes. The additional workload is expected to be done by the remaining employees. The cost of turnover, burnout and performance strain is not accounted for. Only the cost savings from reducing headcount is factored into the equation. If strained employees now leave to work for other companies there will be more work left to be done by fewer employees.

**Initiative inertia**-Companies may lack both the means and will to stop existing initiatives. Mystery shoppers were once the retail standard to understanding the in-store customer experience. Currently, mystery shoppers are no longer essential to understand the customer experience in the face of digital shopper data, ecommerce reviews and ratings. It does take an additional skillset and likely additional software to capture, understand and value customer data gathered online. It may be easier to continue an outdated initiative than to invest in system upgrades and employee upskilling needed to leverage data in new forms.

Let us move on to discuss three reasons why effective prioritization is a critical skill for all leaders. Effective prioritization impacts employee and organizational health. Effective prioritization points the entire organization towards one north star. Effective prioritization takes companies from good to great.

How many hours will the average person spend at work over the course of their life? Assuming we work 40 hours a week for 52 weeks each year we will spend one-third of our lifetime at work. Yes, we will spend 90,000 hours at work. Annie Dilliard has a famous quote that says, “How we spend our days is of course how we spend our lives”. Leaders set the tone of the business and are responsible for directing the use of the company’s money, and employees' time spent on accomplishing strategic goals. Leaders set the culture of the workplace by determining employee’s workload and therefore work life balance. Work stress and productivity are direct results of prioritization. Without prioritization employees can feel that they are being asked to juggle day to day functions while needlessly taking on additional projects. There are numerous reports that tie work stress to poor health outcomes for individuals. Headaches, work related injuries, high blood pressure, decreased immune function and fatigue are just a few of the consequences of work stress. As employees take time off to manage these illnesses or worse yet become ill and unable to work, additional work falls to others. This sets up a vicious cycle where the employees that remain take on additional workloads and consequently additional work stress. Leaders must be effective at prioritization because leadership choices impact the health and well being of employees. For the amount of work companies need to have done by the number of staff to accomplish the work, leaders have to do everything in their power to ensure their employees are healthy. **The health of the organization is a direct reflection of the health of individuals that make up the organization. Leaders can positively impact the health of employees and the overall organization through effective prioritization.**

Employees have to understand the mission and vision of an organization. Employees should be able to articulate “what we do as a company and why it matters”. While strategies are common for organizations it is uncommon for every employee within the organization to be able to
communicate the strategy. Without this understanding employees may choose to implement or not implement initiatives which the business deems critical. In the 7 Habits of Highly Effective Individuals Stephen Covey describes effective leaders as those able to point everyone in the same direction. Effective prioritization demonstrates the one “wildly important goal” businesses are focused on by aligning initiatives to support that goal. Note that the most successful goals serve a greater purpose. Employees no longer only look for jobs that pay a wage or a salary. Individuals seek purpose driven work. Initiatives which may come down to employees through leaders are always made better when employees are allowed to review and provide candid feedback. Leaders never have the complete view of the business because they rarely are face to face with customers or end users. Employees and managers are in contact with customers and often have insights that are vital to understanding the root cause of any problem. Effective prioritization is critical for employee engagement and retention. Leaders can improve employee engagement by constantly communicating the mission and aligning all initiatives to support the mission.

Finally let us conclude by applying personal work scenarios to at least one prioritization model to create a customized prioritization roadmap including next steps for implementation.
Candor as Kindness
Kemba L. Marshall MPH, DVM, ABVP (Avian), SHRM-CP

Raise your hand if you believe communication is always part of any problem, and also always part of the solution. To any issue, at any point of your career. We agree! The purpose of this radically candid communication session is to provide strategies and resources for managing difficult conversations and helping upset clients and colleagues. Kim Scott’s Radical Candor model of caring deeply and challenging directly creates a firm foundation that will set program attendees up for successful, strong, and sustainable communication wins.

What makes conversations difficult? Conversations can be difficult when the objectives include: delivering bad news, admitting that you made a mistake or ending a relationship. Asking someone to make a change or even reviewing an estimate before proceeding with a patient’s clinical work up or treatment plan can all be difficult. Difficult conversations can occur when individuals are emotionally tied to an outcome, a process, a client or a patient. Difficult conversations can occur when there has been a policy or procedure violation that needs to be addressed or when a social media post now has implications at the workplace. Disappointment, frustration, distrust, anger and misunderstanding can all make conversations difficult.

Often, we have been told from a young age to remain silent if we what we have to say is not ‘good’. Additionally, we have been told to be professional at work and to “check our personal stuff at the door”. As Kim Scott author explains in her book, Radical Candor, difficult conversations can be those where, in order to spare someone’s feelings, we neglect to say the thing that must be said. We can also sugarcoat the thing that must be said to such a degree that it is no longer recognizable as an issue. Difficult conversations can also be those where we are passive aggressive and say nothing directly to an individual. Difficult conversations can be those where the speaker is using the conversation as a means of public humiliation for the listener.

When business revenue, team expectations, patient care or client satisfaction have been negatively impacted and must be discussed, staff conversations can be difficult. While there can be the temptation to not say anything or brush it under the rug, silence rarely makes anything better. We wind up letting things fester and emotions build up when individuals feel unseen, unheard or unappreciated. Preconceived notions can also make conversations difficult. “I did not want to hurt your feelings” is a common reason given when the question posed is, “Why didn’t you tell me?”. We have preconceived notions that telling someone the truth will hurt their feelings as we ask that people be honest with us at all times. Conversations can be difficult because interpersonal relationships are complicated.

As veterinary professionals we are taught to not just treat the symptoms but address the root cause. There are tools to help us address the root causes of difficulty in conversation to improve our professional (and personal) relationships. In our time together in this session we will get to the root cause of the difficulty in conversations. We will learn to use radical candor to manage difficult conversations. We will contrast three methods for working with upset clients and colleagues. We will conclude with learning the ‘what’s working well (WWW) and what could be even better yet (EBY) feedback model.

“Radical Candor” is what happens when you put “Care Personally” and “Challenge Directly” together.-Kim Scott
Kim Scott uses her experiences at Google, Apple, Twitter and even that of managing a diamond cutting facility in Moscow to develop her guiding principle. That principle is, as a boss you must be human and straightforward. The specific type of guidance Scott recommends is radical in that people should mean what they say. The candor highlights a sincere belief on the part of the individual in what they say and do.

Radical Candor emphasizes the quality of honest communication. In order to have honest communication, one must care personally while using transparent and candid communication to challenge directly. Using Radical Candor helps form legitimate bonds among team members. In order to achieve their best work, teams need genuine connection. There are ten principles that combine to make up the Radical Candor framework of communication.

1. Care Personally and Challenge Directly
2. Great Leaders Lead Through Guidance
3. Emotional Presence is Essential for Good Leadership
4. Put Deliberate Efforts into Understanding Your Colleagues
5. Be Open to Criticism
6. Learn Self-Compassion
7. Use the Get Stuff Done Wheel Effectively
8. You Can Heal from Traumatic Experiences
9. Resilience Prepares You for Hardships Later in Life
10. Take As Much As You Give

The practice of radical candor can be facilitated by having honest conversations. Systematic process review also facilitates radical candor. Finally, listening to ideas from different role players will make the practice of radical candor easier to implement. Now that the framework of radical candor has been listed above let us expand on the foundational principles; to care personally and challenge directly.

Care Personally and Challenge Directly advises that first employees are to be seen as human beings. Seeing someone as a human being required a personal connection and care for them on a personal level. In work settings leaders historically have been taught to not engage on a personal level and handle only the business of work during work hours. Managers can (and should) create a nurturing work environment alongside professional relationships.

Caring personally requires work, dedication and conscious time investment. Caring personally is a stark contrast to historical ideas of professionalism. The goal of caring personally is to understand the humans you work with and factors that might affect the team’s performance. Caring personally for members of the team strengthens the team overall. The work of caring personally is that leaders must bring their whole selves to work and engage employees on a level deeper than creating work goals, developing work processes and evaluating the progress made towards work goals. Caring personally is to care for each member of your team as a human being.

There are several barriers to caring personally. The first is arrogance. Holding a position of power or feelings of superiority will make it difficult to care personally. The second barrier is being unable or unwilling to show vulnerability. Belief that professionalism does not involve personal interaction is a barrier to caring personally.

Challenging directly or challenging in person balances caring personally. Challenging directly while caring personally allows you to connect with the human recipient of your communication. Radical candor results in transparent communication to help the organization reach its goals faster. Challenging directly means not only are you able to provide direct in the moment feedback or correction but that you are able to receive in the moment feedback or correction. Part of what makes conversations difficult is we have to hear things that upset us. Empathy requires us to understand this as both the deliverer and recipient of communication. Remember that the term organization is used in its broadest sense here. An organization is a group of human beings who have come together to do something.

Radical candor can be pictured as a 2x2 table. The spectrum of caring personally can be viewed as the y axis moving up from not caring at all to caring personally. The spectrum of challenging others can be viewed as the x axis and moves from silence at zero to challenging directly. There are four outcomes assigned to the four boxes. When you do not care at all and are silent this is termed manipulative insincerity™. When you care personally but remain silent this is called ruinous empathy™. Radical candor™ is the ideal when you personally care the most and challenge directly. When you challenge directly but do not care personally this is termed obnoxious aggression™. The elements of this 2x2 table are further explained below.

Manipulative insincerity is also referred to as being passive aggressive or political backstabbing. This feedback failure occurs because you do not wish to directly challenge the individual face to face. Hallmarks of this behavior are
flattery to someone's face and harsh criticism to someone's back. Manipulative insincerity can be seen in response to obnoxious aggression. Manipulative insincere guidance comes about when the boss has a desire to be liked and take advantage of it.

Ruinous empathy shows a high degree of personal care without honest criticism. In this quadrant unclear feedback is given to avoid hurting the recipient. Sugar coating feedback may spare the individuals feelings short term. Long term this will hinder the individual's growth because there is either no feedback or the feedback is false. Ruinous empathy is unhelpful and ultimately damages the professional relationship. Ruinous empathy occurs when bosses are attempting to reduce tension. Ruinous empathy causes bosses to insist on letting things go as opposed to resolving issues.

Radical candor is humble, helpful, immediate, in person (in public if it is praise and in private if it is criticism) and it does not personalize. Radical candor combines criticism with the goals of helping individuals see mistakes and correct them. Radical candor delivers contextualized and specific praise. Contextualized and specific praise allows individuals to feel seen and valued.

Obnoxious aggression is the unnecessary act of challenging someone directly. This is brutal honesty or front stabbing. This cultivates impersonal relationships and does not feel like sincere feedback. Obnoxious Aggression is displayed when the boss belittles and publicly embarrasses employees. While obnoxious aggression criticism can be effective it is very costly. Obnoxious aggression leaves a trail of dead bodies in its wake. Praising people aggressively or under the wrong circumstances may result in making individuals feel ashamed or underestimated. This is dangerous because individuals who do not feel seen or valued are at a significant risk of turnover.

While calmly reading the above explanations these likely resonate with many. In the moment of heated or difficult conversations often times we are not consciously thinking. We are either responding or deflecting blows. Whether working with colleagues or clients there are several methods to work through upsetting situations. Positions of leadership are tasked with managing needs and emotions of team members and clients. Leaders have the responsibility and privilege of building a culture of trust and psychological safety. Cultures of trust and psychological safety assure that the team trusts the team's foundation is solid and able to withstand the weight of open and honest dialogue.

As service providers veterinary clinics are balancing not only the medical needs of our veterinary patients but also the needs of our clients. While administering patient care has its own challenges, compassion fatigue being a significant one, the past three years have brought increased tension in to workplaces. There is a cost to continually working through the uncertainties of a pandemic, an increase in societal stress and increased burnout and turnover in many work sites. With more people working remotely or working in a hybrid model increased requests for appointments mean additional strain on the roughly 5100 Ontario veterinarians providing veterinary care for the province. According to the Canadian Animal Health Institute the country’s dog population grew from 7.6 million in 2016 to 8.3 million in 2018. 2020 reports indicated that 18% of current pet owners obtained a pet during the pandemic. In response to staffing challenges and shortages attributed to the ongoing pandemic, elder care and childcare more than 80% of veterinary clinics are at capacity and are forced to turn new clients away. The natural tendency when you receive criticism or negative feedback is to personalize the information.

There are three different ways you can manage difficult conversations (or negative feedback) with peers or clients. First, by depersonalizing this data you can move away from perceptions of danger or personal attack which cause you to dismiss, deflect or defend against negative feedback. Team members or clients who are angry are providing you with data and insights. As a leader you do not want to add judgment or excuses to the data. By accepting the fact that a person is frustrated you can lean into the anger with an intent to learn and move towards effective resolution. Ask for more information rather than suppressing or ignoring anger. You care for and acknowledge the individual when you ask for more information. By reframing the concept of anger you can navigate this emotion sensibly. If an individual wants to vent allow them to do so. You are the leader and in charge of setting the culture and tone of the workplace. An angry person does not have to trigger a defensive posture. It may be that there is valuable information shared that addresses the root cause for the anger and points you towards an improved outcome or process. You should acknowledge that you care deeply by affirming their position. Your conversation can begin with, “I know you are angry. My goal is to listen to you with the intent to learn (establishing you care about the individual). By caring enough about this situation to be angry, once you provide additional information, I invite you to provide ideas on how we can address this issue together (addressing the issue head on without beating around the bush).”
Secondly, inviting dialogue de-escalates emotions around difficult conversations. In learning about the source of anger you can channel frustration into a constructive outcome. Whether you are working with team members or clients you become more proactive when you pivot towards the challenge of benefitting others. Leaders should not assume they know everything and should invite dialogue to illuminate blind spots by listening to the perspective of others. Your ability to help others regulate and pivot their emotions not only helps people feel better but helps drive constructive dialogue around what should be changed and how that change can begin. By inviting dialogue you acknowledge the validity of their response to the situation. This does not mean that you agree with everything said, this means you agree that the person saying these things is someone you care about as a human being. That caring personally allows you to hear the direct challenge brought to you in a difficult conversation where you are hearing criticism. Your conversation can begin with, “While this may be difficult for you to say and difficult for me to hear this dialogue is necessary because we are going to have to find a way forward.” This acknowledges the situation for what it is (difficult) and acknowledges the foundation of trust in the necessity of the relationship. Challenging directly invites others to air out their grievances and be heard. Here you want to focus on data and not take this information sharing personally. The ultimate goal is an improved business outcome, and this is what is required to reach that goal.

The final approach to dealing with peer or client difficult conversations is acknowledging your role in conflict. This shows your authority and humility as a leader and builds trust. Your conversation here may begin with “Thank you for sharing this information with me. I can see that I have a role in this conflict. I am also in a position to address this and you have my commitment to do so.” Here you demonstrate that you care personally by acknowledging the reality of the situation. You challenge yourself directly by stating that you will work towards a solution and the individual is your accountability partner. The individual will have a tendency to continue to seek evidence to support their prior belief. Initially, flaws or missteps will be noticed more than improvements. To address this confirmation bias your role is to invite others to notice the good you are trying to do and develop the ability to give the benefit of the doubt. If you are able to own your developmental needs and invite your peers to consistently give you feedback and advice you are modeling radical candor.

Two of the key factors in a work setting that erode trust are lack of transparency and playing favorites. When trust erodes in any relationship conversations will become difficult because there is a loss of psychological safety. Psychological safety is created when individuals know that they are seen, valued and are contributing to decision making. If there are negative perceptions about you specifically you must be straightforward and hear the perceptions of others. We all have blind spots and what you do not know can hurt your performance as a leader. Your response to angry colleagues and clients can actually build trust and serve as fuel for greater future performance.

Putting it all together we can use a feedback system that allows us to depersonalize feedback and focus on data points. When individuals come together to accomplish a task they care enough to be there and care enough to provide feedback. Start by asking what went well (WWW). Do not add any more guardrails than this because you want unsolicited feedback on the entire goal that you gathered around in the first place. This causes individuals to pause and reflect on the beginning, middle and end of the process and builds trust. By asking for feedback you emphasize your personal concern and respect for the individuals you are working with. It is helpful to ask respondents to list the WWWs so that you capture this feedback. Here as a leader, you are an active listener.

The next question is, what would be even better yet (EBY)? You demonstrate that you invite the direct challenge of hearing something that may be criticism. Again, you are listening; not defending, dismissing or deflecting. Your commitment to the process is continual improvement so that the team’s overall performance is better due to the feedback. By demonstrating the positive nature of receiving criticism you are modeling to the team how to receive feedback. Because you know how it feels to receive criticism this allows you to be more empathetic when providing criticism. We started our conversation with old adages about professionalism meaning being work robots and silence being the best tactic if you do not have something good to say. Those are not true. What is true is that people do not care how much you know until they know how much you care. Candor is kindness and in the EBY portion of feedback you invite the kindness of fellow human beings to help you get better. Getting better can involve more effective meetings, better client communication, better patient care and better employee morale. EBY invites you to include the perspectives of others who may have skillsets and talents you have been unaware of up until now. EBY invites innovation in the everyday process of work that can be a culture of innovative EBY conversations about every aspect of the business. By continually soliciting feedback you establish this as a culture norm and disarm the recipient of the feedback. Instead of being seen as an attack, feedback is seen as a gift that can be used for the entire team’s benefit, growth and development.
Good leadership requires emotional presence, clear guidance, teamwork and results. No relationship thrives when role players bring bare minimum effort. In order to understand your colleagues, you have to understand how work fits into their life goals. In order to understand your clients, you have to understand how their pets fit into their life goals.

Difficult conversations are unavoidable. You can understand radical candor and still have conversations that did not go as planned. The framework of radical candor helps us recognize where we are in the conversation and points us towards the desired outcome.

Any team of people working together will always need someone to guide them. If you are fortunate enough to be chosen to lead a group, you have two approaches to leadership. The first is through power and authority where you tell workers what to do and display negative consequences when a worker does not do what they are told. This leadership style is ineffective and devoid of humanity. In small ways individuals can undermine this authoritative leadership style by doing just what you instructed but nothing more. There is a human centered approach to leadership which is much more effective because you invite individuals to come together and develop strong relationships to accomplish a task. This human centered approach is founded in a core of deep trust. Radical Candor has a two-dimensional view of trust. The two dimensions are to care personally and challenge directly.

Radical Candor identifies in order to achieve radical candor you must be willing and able to:

1. Care for subordinates on a personal level. Their role in the workplace has no bearing on their worth as a human being. They may report to you but they are not beneath you.
2. Challenge people face to face
3. Get to know your subordinates on a human level.
4. Build and maintain a culture of honesty and direct feedback.
5. Recognize that feedback does not have to involve hostility. You are receiving data not being personally attacked. Remember this is also true when you are delivering feedback.
6. Bring humanity (yours first) into the workplace in order to build strong personal relationships.

Kim Scott uses this business case for Radical Candor: “When radical candor is encouraged and supported by the boss, communication flows, resentments that have festered come up to the surface and get resolved, and people begin to love not just their work but whom they work with and where they work. When people love their job, the whole team is more successful. The resulting happiness is the success beyond success”.

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Everyday Leadership

Kemba L. Marshall, MPH, DVM, DABVP (Avian), SHRM-CP

Marshall Recruiting Consortium

Have you ever led a sports team, fundraising initiative, a team of employees or organized your family’s holiday plans? Welcome to leadership. What is everyday leadership? Everyday leadership is our unique ability to impact others through our everyday actions. In work settings, employees who exemplify everyday leadership are empowered to make decisions and are motivated to do their best. Managers are responsible for empowering employees. Employees are responsible for making sound business decisions and being motivated to do their best. A significant benefit of everyday leadership is the retention of committed, empowered employees.

Historically, leaders have been a homogenous population of cisgender, older White men. Our world demographics are showing ever increasing diversity and in order to be effective, leadership has to reflect the general population. Gender and ethnicity diversity as well as faith, personality and disability are dimensions of diversity that should be reflected in leadership in order to increase inclusion. Individuals across all ranks of an organization should be empowered to lead as critical, creative and ethical thinkers. Every role at every organization has an element of leadership. When well executed that leadership should be acknowledged in employees. After all, what you appreciate, appreciates.

Associates want to be seen, heard and contribute to important business decisions. Another benefit of recognizing everyday leadership is we encourage employees to find their voice. We can increase inclusion by encouraging individuals to think about their contributions to move the business forward. Everyday leaders can provide opinions about organizational culture, operations and output. Everyday leaders who are empowered to speak up share insight into what is working well and what would be even better yet in our workplaces.

If you want to improve your leadership skills, you must first see yourself as a leader. Leadership needs to be perceived as attainable. Recognizing everyday leadership demonstrates to others that they also are in fact leaders regardless of their work title or role. Recognizing everyday leadership in our teams directly challenge feelings of low self-esteem, self-doubt and uncertainty. When we all see ourselves as leaders, we realize we all have a responsibility to lead. Acknowledging everyday leadership enables us to have teams that are empowered to develop their own understanding of ethics and personal responsibility. There is an important, added benefit of everyday leadership. When we develop leaders from within our own organization, we have the ability to drastically improve retention.

Now that we understand a little bit more about what everyday leadership is we will move to how it’s done. We will start with looking at traits of everyday leadership.

Adaptability- Everyday leadership adapts to changing situations. Everyday leaders are not compelled to stick to a script, or the ways things have always been done. Everyday leaders think outside of the box and learn to embrace new ideas and concepts that may be outside of their comfort zone. Everyday leaders accept that challenges and obstacles are all part of the personal growth and development process.

Emotional intelligence (EQ) is the ability to recognize, understand and manage your own emotions. It is also important to learn to influence the emotions of others. Everyday leaders have a high EQ and are skilled at balancing the needs of their team and the company. Feelings and emotions are often mixed together in veterinary practices. EQ always everyday leaders to acknowledge feelings and emotions of employees and clients while moving towards desired business outcomes.

Vision- Everyday leaders perform day to day tasks while keeping an eye on the company’s vision. Day to day tasks may be updated or improved as everyday leaders evaluate the correlation between the task and the company’s vision. Everyday leaders feel safe questioning established policies and procedures with the goal of making recommendations to improve them.

Participation- Everyday leaders show up. They get their hands dirty to do what it takes to get the job done. Everyday leaders lead by example and participate in activities with their team members. Team participation boosts morale as employees are working shoulder to shoulder with everyday leaders. This team participation also allows for innovation.
and process improvement as team members participate in accomplishing tasks and can spot roadblocks or inefficiencies.

Coaching ability—Everyday leaders possess the talent to coach instead of simply telling others what to do. Combined with emotional intelligence, this coaching ability sees the individual. Knowledge of and appreciation for others allows the everyday leader to tailor how to inspire and encourage others. Everyday leaders know there is no one size fits all formula for motivating employees. Everyday leaders invest time in getting to know individuals and therefore can identify the most impactful motivation techniques. Everyday leaders are focused on outcomes not the task itself so if the steps vary that is allowed as long as at the end of the day expected outcomes were achieved.

Everyday leaders who are individual contributors can begin their leadership journey by utilizing the following six steps:

1. **Embrace conflict** it is normal and healthy. We are trying to discover the best solution, not prove that we are right when discussing alternate approaches to problems or challenges. Learn to listen fully. This means listening for understanding. This does not mean listening and waiting for your turn to respond. “Tell me more about your plan” is a powerful statement that invites others to share their innovative idea in a nurturing environment. Everyday leaders exercise great control and do not take conflict as a personal attack.

2. **Make space for different ideas.** Often the idea that proves to be the best one is a combination of several ideas shared during a brainstorming session. Making space for different ideas means understanding the diversity of personality types on your team and allowing them to contribute in a way that is welcoming for feedback. This may mean giving pre-reads and allowing individuals to pre-submit comments. This also means giving all individuals the ability to speak without being interrupted. Nurturing environments allow for individuals to share their “wow” of the idea without getting bogged down in the “how” of the idea.

3. **Reward and recognize others’ achievements.** Everyday leaders hold others accountable meaning that they praise publicly and correct privately. Everyday leaders lean into their EQ and understand how important recognition is to boosting overall team morale. There is also the healthy competition of encouraging other team members to do their best work to advance business outcomes. Rewarding and recognizing others achievements allows us to also identify unknown strengths of team members. These hidden skill sets are a great foundation for stretch assignments, special projects and other elements of individual leadership plans.

4. **Make friends.** Everyday leaders understand the importance of networking. Strong business relationships allow for cross-functional alignment and allow for further ideations. Networking allows for streamlined work processes and can improve efficiency. Making friends allows everyday leaders to have better visibility across the entire organization. Everyday leaders can then use their networks to improve networks of others. Everyday leaders are pivotal to have all individuals in the organization working towards one goal.

5. **Critique compassionately.** Everyday leaders understand the importance of maintaining trust and psychological safety to drive innovation. Critiques can be seen as rejection and this can result in people shutting down rather than risk further rejection. Everyday leaders understand that to get the best out of others praise should be shouted and critiques should be whispered. Most importantly everyday leaders invite others to give them criticism. By inviting criticism everyday leaders remove the preconceived notion that criticism is bad. They move towards the productivity of criticism in continual improvement.

While talking about leadership you have to also talk about personal accountability. Leadership is not something that is bestowed upon a person, true leadership is grown from within. True leadership also has to be agreed upon by others. Leadership is not a title it is a respected position of care, trust and authority that others place in you. Leadership is earned based on one’s habits. Four habits are critical to everyday leadership. Everyday leaders make a habit of being accountable to outcomes rather than tasks. Sticking to the plan completely without taking moments along the way to assess whether or not it’s working can be futile. The plan is known, safe and feels productive. Sticking to the plan no matter what can be impractical leading to unnecessary busywork, chaotic recovery and goals that seem out of reach.

Prioritizing outcomes rather than the plan can be scary as more time will be spent in the unknown. Outcomes are the north star you are headed towards. Adjustment is part of the plan. If something is not going well or a mistake has been made you can get back on the task of reaching outcomes.
Secondly, everyday leaders do not take breakdowns personally. Breakdowns happen as we are human. Rather than looking for perfection leaders can be present enough to quickly make an adjustment during a crisis and get back on track. Proactive recovery plans can be used to anticipate obstacles ahead of time. Team members should know they are all mutually responsible for desired outcomes. Brainstorming, problem solving and creating solutions together is a great way to develop shared ownership among the team taking pressure off the leader to be perfect.

Thirdly, everyday leaders own up to and learn from their mistakes. Learn from your mistakes because if you are not making mistakes, you likely are not taking enough chances to move the business forward. Keeping mishaps a secret when they affect others is a recipe for snowballing the negative effects of your mistake. Owning up to your mistakes gives your team a chance to support you in making it right. Owning your mistakes signals to your team they are not held to unreasonable goals of perfection. Team problem solving creates deep and lasting trust among team members. Reaching goals easier is when you are not watching your back and defending yourself. The most important thing about life including business is life-long learning.

Everyday leaders listen well and listen often. Being the leader does not mean being the best, the smartest or a dictator. Being a leader means listening to others. The people around you are your treasure bringing you diverse insights, ideas and experiences to make your organization stronger. One strong perspective is not enough to maintain a well-functioning team in such a diverse and creating world. Listening to others give you ideas and helps you create connections. Intently listening to others acknowledges they are important to you and that they are involved in making important organizational decision. Everyday leaders demonstrate inclusion by being active listeners.

Everyday leaders honor their word. They keep agreements as related to desired business and personal outcomes. Everyday leaders spend the time and energy needed to follow up and follow through. Honoring commitments builds group trust and cohesion.

Everyday leaders build a culture of communal problem solving—all the hard work does not have to fall on the shoulders of one person. Problem solving allows you to learn more about the unique skill sets of your team. Additionally, you learn more about the knowledge and creativity of that person. Teams who problem solve are more nimble and agile responding to change with a faster pace. Creative, communal solutions create trust and allow for more innovation and risk taking.

Everyday leaders trust their teams. Micromanaging creates cultures of low trust. High trust increases personal accountability as individuals understand their role on the team and in achieving the outcome. Leaders must allow the team to demonstrate trustworthiness. Everyday leaders realize there is more than one way to accomplish a goal and allow individual creativity in accomplishing duties and tasks.

Everyday leaders pass the praise and accept the blame. Blaming others when things go wrong is one of our minds’ favorite ways to get out of a jam. Pushing blame to someone else relieves us of responsibility, or so we think. Blaming someone for a situation is unhelpful. Personal accountability is taking ownership of mistakes, communicating clearly to those impacted and fixing the problem. High functioning teams function as one unit. Achieving desired outcomes is enabled in a culture of problem solving and support. Everyday leaders support team cohesion by not throwing team members under the bus.

How Accountability Can Change Our World

Mark Samuel, author of The Accountability Revolution, defines accountability as people counting on one another to keep performance and communication agreements. This can results in increased synergy and a climate safe for experimentation and change. Employees can feel the climate of support and trust. Ultimately this leads to higher employee morale and satisfaction. Managers can rely on performance management principles to improve accountability with five steps:

1. Involve employees in setting clear, challenging attainable goals and objectives. Give employees the authority to accomplish these goals.
2. Coach employees when they need help and support employees in all aspects of job performance.
3. Monitor progress towards goals and provide feedback that includes credible, useful performance measures.
4. Provide the necessary training and resources to employees to do the work.
5. Recognize employees for good performance both formally and informally.
To be held accountable is to be required to answer for accomplishing a goal or assignment. Too often the word accountability has negative consequences and is synonymous with punishment. If accountability only means punishment, then the workplace is riddled with fear and anxiety. There are ways to highlight the value of a positive approach to accountability with valuable results.

The positive results of practicing a constructive approach to accountability include:

- Improved Team Performance
- More employee participation and involvement
- Increased feelings of competency
- Increased employee commitment
- More creativity and innovation
- Higher employee morale and satisfaction with the work

Managers who involve employees in goal setting find that employees understand expectations better and are more confident that they can achieve expectations. In turn employees perform at a higher level. When employees do not fear failure and are recognized for their accomplishments, they are more likely to be creative, innovative and committed to their work. Managers must support their employees when goals become difficult if employees are to thrive.

Creating a Leadership Development Plan

Striving to be a strong leader can be a commitment you make during any stage of your career. You own your leadership development plan and your ability to maximize your personal influence and impact. Here are five steps to create a leadership plan. Remember that leadership is continually evolving journey.

1. Assess Where You Are Professionally. Strengths, weaknesses assessments help raise your self-awareness. You want to understand how your current skillset contributes or detracts from your capacity to lead. Assessments can be informal as you list your own strengths and weaknesses and solicit the opinions of your friends, family and coworkers. How you see yourself in leadership roles may be quite different from how others perceive you in leadership positions. Emotional intelligence is increased as you become more aware of how others experience you.

2. Set an attainable goal using the PACE model. In this model you pick a leadership goal and apprise others in your inner circle of the goal. Your inner circle conversations provide information on how you will go about achieving your goal. You collect specific ideas on how you can improve your leadership skills and go about that. As you are working on your goal you elicit ongoing feedback from your inner circle. Your inner circle is the group that cheers as you make progress and encourages as you face setbacks. The goal you pick should identify a goal you can go after which increases your personal effectiveness. There should be both a short- and long-term portion of your leadership goal. Leadership is often achieved via small steps along the way to your overarching leadership goals.

3. Engage in leadership training. Training provides practice and skills needed to boost your confidence in your ability to lead others. When in leadership you spend a great deal of time empowering employees and influencing others. Leadership training shows you how to get better at both of these necessary skills.

4. Interact with your network. Your network should include other leaders and those who are impacted by your leadership. Interacting with a network is nurturing relationships. Your network should not just be a group of individuals that you do transactions with. Your network should be a group of individuals you spend time learning from and exchanging ideas with. When considering your network it should be as vast as your desired level of impact. If you network consists of individuals that are identical to you in lived experiences, education, hobbies and life outlook you are not getting the most out of your networks. Effective networks are diverse in both primary or visible definitions of diversity and secondary or discovered dimensions of diversity.

5. Hone your soft skills. Soft skills are critical for leadership. Soft skills include the following:
   a. Communication
   b. Career planning
The purpose of this program is to instill the confidence and competence needed to embrace the reality that all of us have the potential for leadership right now in our current roles, whatever that may be. Bringing everyday leadership will only increase your ability to be effective in your role. Everyday leadership is something we do often without acknowledging our own efforts. We lead teams, family, friends, colleagues, youth groups, journal clubs, book clubs and recreational sports leagues.

With every interaction and every decision, we have the potential to positively impact others as well as cultivate our own personal growth and development. In business settings everyday leadership has been shown to not only increase individual job satisfaction but to increase team morale, team effectiveness and client satisfaction. Increased employee retention and decreased employee turnover are also outcomes of effective everyday leadership.

Too often we disqualify ourselves as leaders; we doubt our ability to lead. Leadership is not perfection, leadership is an authentic embrace of our full humanity as we seek to interact with the humans we encounter at work and at home. Everyday leadership is transparent as we allow employees to know more about us that simply our work title. Everyday leadership is authentic as we seek deep connections with our team and think of them as more than just individuals doing a job.

Leadership starts with an acceptance of the role of a leader no matter what your work title is. We have all seen leaders who have been appointed or put into a role who actually are not leaders at all. Additionally we have seen individuals who without regard for their title assume the role and habits of a leader. Individuals who assume the privilege and responsibility of leadership agree to set the culture of our workplaces, accomplish business goals and protect the team being led.

Leadership is a continuum and requires work, effort, intentionality and humility. Leadership gives the benefit of being a part of a group of individuals who accomplish more than initially appears possible. Leaders guide teams. Nothing can stop a team from accomplishing its collective goal.

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Nuts and Bolts of Practice Management

Monica Dixon Perry, CVPM

Veterinary Practice Management Essentials

https://www.vetpartners.org/practice-management-resources/

- Task Force Members:
  - Dr. John Alber
  - Dr. Karen Felsled
  - G. Lynn Davis
  - Debbie Boone
  - Betsy Choder
  - Dr. Mary Gardner
  - Jennifer Inbody
  - Monica Dixon Perry
  - Terra Shastry
  - Martin Traub-Werner

Veterinary Practice Management Essentials

STRATEGIC PLANNING

- Establish a clear vision:
  - Identify the mission, values, and goals of the practice
  - Differentiate your practice

- Establish and prioritize

FINANCE AND ACCOUNTING

- Ensure financial stability
- Maintain cash flow
- Monitor financial health
- Implement cost-containment strategies
- Monitor and report financial performance
THANK YOU!

Monica Dixon Perry, CVPM
monica@burzenski.com
www.veterinaryfinancialadvisors.com
Top Tips of Personnel Management

Monica Dixon Perry, CVPM

Effective Communication

- Implementation of new policies
- Team Meetings
- Discussing Profitability (or the lack there of)
- Articulating Expectations

Document, Document, Document

- Job Descriptions
- Training Programs
- Evaluations
- Observational Interviews
- Disciplinary Action
Team Involvement/Engagement

- Open Book Management
- Observational Interviews
- Additional Duties
- Post Conference Committees

Questions

THANK YOU!

Monica Dixon Perry, CVPM
monica@burzenski.com
www.veterinaryfinancialadvisors.com
Conducting Awesome Evaluations
Monica Dixon Perry, CVPM

Common Types of Evaluations
- Annual Evaluations
- 360-Degree (Peer) Evaluations
- Real-Time Evaluations

Pros and Cons
(Annual Evaluations)

Organizational Chart
Conducting Awesome Performance Evaluations

- Expectations in writing (Job Descriptions)
- Provide a copy of evaluation during new hire orientation
- Establish when they will be evaluated
- Inspect what you expect
- Address issues sooner than later

Conducting Awesome Performance Evaluations

- Schedule evaluation one month in advance
- Stick to the scheduled time/date
- Be prepared
- Focus on strengths, not areas of opportunities
- Solicit feedback from management only

Conducting Awesome Performance Evaluations

- Open communication
- Discuss administrative topics at the end of the review
- End on a positive and encouraging note
- When is the next evaluation?
- Schedule the next evaluation

Job Description
Conducting Awesome Performance Evaluations

Training Program

Conducting Awesome Performance Evaluations

Performance Evaluations

Dolphin Theory of Management

They may forget what you said, but they will never forget how you made them feel.

-Carl R. Buechner
THANK YOU!

Monica Dixon Perry, CVPM
monica@burzenski.com
www.veterinaryfinancialadvisors.com
Recruiting New Grads:
Pay, Benefits and More!

Monica Dixon Perry, CVPM

Feedback from Dr. Tierra Price

- 2020 Virginia-Maryland CVM Graduate
- BlackDVM Founder & CEO
- Emergency Veterinarian, Veterinary Emergency Group (VEG)

What is MOST important?

- Competitive wages
- Fits budgeting needs
- Flexible time/flexible schedule
- Efficient, effective, productive co-workers
- Appreciation for a job well done
- Benefits offered
- Efficient co-workers
- Organized team and operations
- Healthy culture and environment
- Relationships with co-workers
- Growth opportunities/Career development
- Being included in determining the direction of the practice
- Mentorship program
- Feedback/Evaluation
- Consultation of difficult cases
- Opportunity to learn new surgeries/procedures
- Practice’s commitment to participating in community events
- Assistance with student loan repayment

What Do Associates Say?

- Base Salary
- Production 88%
- Professional 12%
- Hourly Only 7%
Three Options for Wage

Salary

Production

ProSal

No Guaranteed Base
Percentage of Gross Production: 18-25%
What determines percentage?
Total Compensation Statement
Production is defined as fees generated and collected for services the doctor is formally involved in the delivery of service.

Three Options for Wage

Salary + Production = ProSal

ProSal Worksheet

Associate Prosal Compensation

Based on Percentage of Production

How Compensation is Applied

Guaranteed Base

$140,000

1. Production Percentage = 23.10%
2. Production Bonus = $140,000.00 × 0.2310 = $32,340
3. Base Salary = $140,000 - $32,340 = $107,660
4. Total Compensation = $107,660 + $32,340 = $140,000

Associate Associate

$10,000

1. Production Percentage = 22.15%
2. Production Bonus = $10,000.00 × 0.2215 = $2,215
3. Base Salary = $10,000 - $2,215 = $7,785
4. Total Compensation = $7,785 + $2,215 = $10,000...
Set Up Two Doctor Codes

Dr. Jones
&
Dr. Jones Rx

Questions

THANK YOU!

Monica Dixon Perry, CVPM
monica@burzenski.com
www.veterinaryfinancialadvisors.com
Develop a Rock Star Customer Reception Team

Monica Dixon Perry, CVPM

Develop a Rock Star Customer Service Representative Team

- Present a Clear Vision
- Leverage to Make Decisions
- Solicit Regular Feedback and Value Their Opinions
- Invest in On-Boarding Services
- Provide Growth Opportunities
- Provide Resources on how to Deal with Clients Face to Face and Over the Phone

Customer Service Language

- Learn How to Say “No”
- Empower CSRs to Be Brand Ambassadors

Pay Better

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Customer Service Language

Learn How to Say “No”

Customer Service Basics and Expectations
Develop a Rock Star Customer Service Representative Team

Empower CSRs to Be Brand Ambassadors

Questions

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Monica Dixon Perry, CVPM
monica@burzenski.com
www.veterinaryfinancialadvisors.com
Using Technology to Meet Client Expectations

Eric Garcia
Simply Done Tech Solutions, LLC
Tampa, Florida, USA

For me, technology is a career. But for almost everyone else, it’s a way of life. Whereas we might have once considered certain areas of technology, like smartphones, online shopping and FaceTime, to be generation-specific, the data shows this is no longer the reality.

We all now shop online, with 2017’s $2.3 trillion in global e-commerce sales expected to grow to $4.5 trillion by 2021. We binge-watch TV via Netflix, send text messages and request Uber rides. Hands reach for the ceiling without hesitation during my lectures when I ask, “How many of you use this type of technology each day?”

Still, despite this growing consensus and our love of tech conveniences, veterinary practices are hesitant to adopt the latest technology solutions. Some practice owners I’ve met tell me that “mostly millennials use tech” and that millennials are generally broke, so why focus on them?

Well, some of the sentiment is true. Many millennials are broke, having pursued higher education at a much greater cost than what average wages can pay off. We’ve all read about the rising student debt.

But take note of this: When it comes to pets and pet care, millennials are doling out big bucks. Statistically, millennials are the largest pet-owning demographic, spending an average of $1,285 a year on pet care, with the majority of that figure designated for veterinary care. Millennials play a larger role in the veterinary economy than many practice owners readily acknowledge.

We live in a world where technology is accelerating into our daily lives. We’re simply too far along in the process to reverse course, even while governments learn to appropriately regulate behemoths like Google and Facebook. Whether it’s Uber, Netflix, Airbnb or another tech solution, pet owners leverage conveniences and apps each day because they make life easier.

I would be remiss not to mention Amazon, perhaps the most notable example of the new technology revolution. Amazon simply provided everything that other retailers didn’t, whether it was a better experience, better service or greater convenience. Only when brick-and-mortar retailers started losing revenue did they react, but their responses were too little, too late for many of us.

The Golden Rule

That takes us to the essence of this article: Veterinary practices deprive pet owners of the same conveniences and experiences we demand in our daily lives.

We expect to fetch a quick ride through a ride-hailing app, never dialing a phone number, but we force pet owners to call us when they need something. We also expect
them to call us and then drive to our clinics to pick up essentials like food and heartworm and flea medications while we — practice owners, consultants and staff alike — do our personal shopping online.

Similarly, we leverage telemedicine by consulting with our physicians online, but we make pet owners take time off work for simple rechecks and evaluations. We receive texts from businesses of all types, but seldom do we provide the same functionality for pet owners.

Even though there’s room for improvement, we need to give our profession credit where it’s due. The veterinary industry is outpacing industries once known to excel in client communications. My analysis, experience and research tell me that we’re outpacing human dentistry, which used to set a standard for communication. Our industry is changing at a much more rapid pace than even a decade ago.

It’s an Online World

Work remains to be done, specifically in areas like telemedicine, online pharmacies and text communication. Many argue that online pharmacy sales pale in comparison to in-person purchases, but we’re seeing more online scripts being filled than ever before from places like Chewy and 1-800-PetMeds. Some practices drag their feet amid such change, while others leap toward it. I urge practices to partner with an online pharmacy and promote the option.

Additionally, I urge you to begin texting clients, giving you more intimate, convenient access to the 277 million American adults and teenagers who actively text. (See my article “Keeping in Touch: Texting Pet Owners” at http://bit.ly/2mMCBr6.)

We also need to use more mobile apps — many clients would like instant access to their pet’s medical records — and we need to leverage telemedicine, a veterinary advent that is just around the bend.

Let’s learn our lesson from Netflix predecessor Blockbuster, the taxi companies and smaller retailers that refused to move toward online solutions and e-commerce. Change is something that can’t be prevented or stopped. Instead, it must be embraced.

So, I’ll ask one final question to that effect: Would you rather try something once it’s too late, or would you rather be an innovator, embracing the new world we live in and offering the best of 21st-century technology solutions to your patients? To me, the answer is all too clear.
About the Author: Eric Garcia is an IT expert. Digital marketer. Industry thought leader. When it comes to helping veterinary practices streamline their technology and attract and retain clients, Eric Garcia has a proven track record of educating the industry and producing results. Eric is an IT and Digital Marketing consultant working exclusively with veterinary practices. In addition to a long list of satisfied clients, Garcia’s work has been recognized throughout the industry. He speaks regularly at conferences all throughout the world. Facebook: facebook.com/EricGarciaFL Instagram: @EricGarciaFL
Building the New Client Experience in a Post COVID World

Eric Garcia
Simply Done Tech Solutions, LLC
Tampa, Florida, USA

THE NEW DIGITAL MARKETPLACE

Smartphones have changed the way that we do just about everything. Whether or not you’re personally using an iPhone, Android, another device of choice or even if you’ve opted out of the tech-revolution entirely, you can’t step foot outside of your door without seeing a smartphone somewhere close by. The reality of the matter is that the explosion in the use of these devices has opened up a whole new world of connectivity, while redefining many of the ways that we used to commonly do business. With 2.6 billion smartphone subscriptions active around the world (measured as of 2015 and increasing rapidly still as recently reported by global telecommunications company Ericsson) we’ve watched smartphone usage increase exponentially in just under a decade, while taking stock of the myriad of effects that this smartphone usage has for people across all ages and demographics.

Some of these effects have occurred for better and some for worse, but regardless of your stance on the rapid rise of technology, it’s widely accepted that smartphones have forever altered the way that our world works with implications that reach far into the future.

Effective use of technology can enhance your veterinary practice and your personal life too! Maybe WhatsApp allows you to stay in touch with family members who are residing across the globe while the QuickBooks App allows you to track expenses easily from your mobile device and increase your bottom line with effective savings and tax-deductions.

It’s the way that you choose to use this technology that’s paramount to maximizing your effectiveness and the success of your veterinary practice’s adoption of apps and technology. Ineffective and overuse of tech can lead to distraction, overstimulation and glaring inefficiencies even in the workplace. For example, if you’re commonly distracted and check your texts repeatedly during work hours, keeping this device on during your typical 9-5 business hours may actually harm your productivity overall. Yes, even if it’s a brand new iPhone 6S!

However, if you use the device to make and receive important calls, check only on time-sensitive material and enhance your efficiency at specific tasks with certain apps, the smartphone may actually be increasing your efficiency exponentially! Deciding how to best use these tools is ultimately up to you, but Simply Done Tech Solutions believes that knowing when to use the right apps, versus when to turn your phone off altogether, can make all the difference.
WHEN TO GET #UNPLUGGED

That’s part of what makes these devices so useful, so ubiquitous and frankly, so addictive. You’ll get out of these devices whatever you put into them. At Simply Done Tech Solutions, we support deliberate and efficient adoption of new technology and the use of smartphones to do everything from refill veterinary prescriptions to sending text-message updates to pet owners and well beyond!

However, we also know that too much dependence on these devices can actually impede deep connections with others, cause stress with constant stimulation and even lead to frequent burnout. To combat some of these more problematic effects, we support a movement called, #UNPLUGGED, which you can learn more about by visiting SimplyDoneTechSolutions.com. Through this program, I personally unplug from technology, email, smartphones etc. for approximately two weeks out of each year. I even recommend this to my employees and business partners as well. Why?

Being subject to a constant influx of new data, pinging, notifications, calls, texts, etc. can render you more vulnerable to stress and decrease your capacity for effective decision making when you’re too distracted.

In fact, a recent study conducted at Stanford University and written about extensively via Forbes found the following to be true after in-depth analysis on the subject of technology and multitasking:

“Research conducted at Stanford University found that multitasking is less productive than doing a single thing at a time. The researchers also found that people who are regularly bombarded with several streams of electronic information cannot pay attention, recall information, or switch from one job to another as well as those who complete one task at a time.”

While of course, this is only one study and there is much more research on the subject to be done, the implications from this study are wide-ranging. Perhaps by trying so adamantly to become more efficient and squeeze even more into each and every day, we are doing less and creating an environment where we absorb less. No matter your age or demographic, you now have access to more data than ever before. Even a single, routine day of checking email, Facebook, Twitter and our favorite news sources can mean that we are seeing hundreds of articles, pictures and videos, simply while surfing the web, tending to tasks or being social online.

WHEN TO GET #PLUGGEDIN

This is, in fact, the double-edged sword of constant connectivity: the ability to hook into an immensely large data pool and cultural circle through the utilization of smartphones and technology, but at the same time, the distractions and myriad of psychological consequences that can come with these constant connections.
While Simply Done Tech Solutions does encourage you to learn more about our #UNPLUGGED program and tips for getting started, we of course support deliberate and effective usage of technology. Simply Done Tech Solutions knows firsthand the profound power that well-implemented technology can have on businesses across the world. Whether I’m visiting a local veterinary practice in Florida or giving a lecture across the world in China, it’s clear that technology has swept across the globe in a truly profound way, and as a result, is revolutionizing our very lives.

From crowdsourcing solutions to everyday problems, to micro-investment apps, rapid transportation solutions like Uber and Lyft and beyond what is even imaginable today, new apps are literally solving problems that have impacted societies for decades. That’s why learning when to get #PLUGGEDIN is a crucial part of evolving and growing your veterinary practice. Many millennials are pet owners and are quickly learning to expect new technology and services from businesses they care about. Younger generations are growing up with iPads in the classroom, smartphones in their pockets and retina screens in their computer labs! They are rapidly learning to expect personalized yet quick service from the places where they do business.

Yes, this might mean expecting to track their food online if they order a pizza, but it also means the increasing expectation of receiving online resources from their local veterinary practice, text-message updates when they drop-off their pet and/or Pet Portal prescription refills when it’s time to resupply an important medicine. Skipping these levels of tech implementation now can mean losing out on business, not just from younger demographics, but also from pet-owners across the board. The good news is that simply by adapting now and refining your relationship to technology over time, you stand to gain new business, while out-pacing other competitors that may not be so quick to adapt at all.

To help you make the most out of the wide world of technology, we’ve compiled some of the apps that can enhance your veterinary practice from the ground up. We’ve seen these apps help veterinary practices to gain traction online and prepare their business to thrive amidst a new and profound age of technology:

**YouTube**

We’ve all heard of YouTube before. While many people use the desktop version of the website to stream videos from the comfort of their homes, others use it to manage their own channels and reach out to a wide audience. When it comes to your veterinary practice, YouTube is a great way to feature pet stories, introduce your veterinary staff and create rich, compelling visual media that younger demographics have come to expect! If you can complement your website with video content, YouTube is simply the easiest way to do it. Additional functionality of the app allows you to subscribe to your favorite channels, create playlists, edit and upload videos, create comments and engage with other users!
The app is easy to use via iPhone, iPad or Android and is free to use. Users who stay loyal to the platform can accrue large audiences and attract more attention to their website directly from the channel. Even if you’re new to the platform, try making a video and keeping it private. Once you have enough time to edit and refine, you can make the video public and send it out to your audience of choice!

**PHN3D**

This is a truly compelling app that comes available on Android Apps from Google Play. This tool, primarily used by veterinarians, allows you to educate pet owners directly in the exam room by using vivid and rich 3D animations, radiographs and instructional videos. Since sometimes the nuance of pet care can be difficult to explain, especially when it comes to anatomical function, this app helps to fill in the blanks along the way.

It allows you to drive home some of your most important medical recommendations and communicate precise care instructions for each pet clearly and carefully. If you’re looking for new ways to communicate with pet owners and transition toward the digital age in one fell swoop, this is definitely one of the easiest ways to do so!

**iDiA**

This app is slightly more expensive, but comes with a huge array of educational information. The app works flawlessly on the iPad, allowing you to give overviews of pet conditions directly to your clients. Rather than fumble over the right words or explain in depth when there’s a time-sensitive issue, this app relies on captivating graphics and imagery to give comprehensive insight into the health and well-being of your pet.

The app comes with a huge array of program files and presentations, email functionality and ready to go client handouts for some of the most frequently encountered pet care topics. By spending a bit of time getting used to this app, you can enhance client interactions and ensure that each time you explain a topic in detail, each pet owner is satisfied with the answers provided.

**PICK YOUR FAVORITES!**

Rocking your veterinary practice with the best apps and implementation of technology takes time, but can enhance the way you do business and allow you to shed new insights directly to your clients, allowing them to become more engaged and put at ease when it comes to understanding a procedure or gaining insight into your veterinary practice.

Whether it’s through a YouTube channel that you maintain and edit with your veterinary team or compelling visual/medical apps like PHN3D, there are many ways to leverage the very best of technology to make sure that your veterinary practice stays on the cutting-edge of what the app-world has to offer. It’s important to remember that no app is going to work for you, unless you put it to work! Learn a new app inside and out,
create an approach toward marketing or implementation via the app, implement and refine!

If you take the time to pick the best app, use it wisely and engage with your clients about their experience, you’ll be setting the framework for successful tech-implementation that can be further enhanced over time.

Just as important as using the app, it’s also important to remember when to put them down. Being too plugged in can hurt your efficiency and cause you to become fatigued. Use technology deliberately and with attention to detail, and you stand the most to gain. Technology has changed the way that we do business around the world! Pay close attention to the new tools you have at your disposal and you’ll ensure that your veterinary practice is ready to engage with a wide audience that’s eager to learn more about the care and well-being of their pet! There are so many opportunities to integrate apps and technology into the things that your veterinary practice already does well. Try downloading one of the aforementioned apps today and enjoy!

About the Author: Eric Garcia is an IT expert. Digital marketer. Industry thought leader. When it comes to helping veterinary practices streamline their technology and attract and retain clients, Eric Garcia has a proven track record of educating the industry and producing results. Eric is an IT and Digital Marketing consultant working exclusively with veterinary practices. In addition to a long list of satisfied clients, Garcia’s work has been recognized throughout the industry. He speaks regularly at conferences all throughout the world. Facebook: facebook.com/EricGarciaFL Instagram: @EricGarciaFL
As Easy as 1, 2, 3, E-Commerce and Telemedicine

*Eric Garcia*
*Simply Done Tech Solutions, LLC*
*Tampa, Florida, USA*

**Telemedicine**

“In a few years, the idea of receiving medical treatment exclusively at a doctor’s office or hospital will seem quaint.” - *Harvard Business Review*

Great medical resources, from hospitals to state-of-the-art diagnostic equipment and cutting-edge research, have long been the cornerstone of modern societies that strive to create a healthier, better world. In the modern age, we pride ourselves on having convenient solutions to ailments that once seemed impossible to cure or prevent. Look no further than the common cold and solutions like *Zicam* or *Airborne*, to see how we no longer simply cure sickness, but we now prevent it too.

Alongside solutions like these, come new and convenient ways to receive medical consultation as well. The new and rapidly growing frontier of “telehealth” is a broad yet significant field, which refers to the use of *any* technology that’s used to deliver medical attention, information or education to a client. In a medical context, imagine this to be an emergency therapy session occurring via Skype, or even the ability to text pictures of an injury to a care provider in order to gauge its severity. This, in a nutshell, is the world of telehealth and telemedicine.

While this area provides a wide-range of new opportunities, it also needs to be treated with the utmost responsibility to accommodate the myriad of new scenarios it encompasses. To that effect, the American Veterinary Medical Association (AVMA) vocally supports this emerging type of care, but also places emphasis on ensuring that the convenience of telemedicine does not create a reduction in the quality of care provided. AVMA policy explicitly states the following:

“The AVMA encourages the development of smart-device applications, other platforms and technologies that appropriately help connect current or lapsed clients and patients with veterinarians.”

With the current ubiquity of smartphones and increasing usage of all types of technology (especially from growing Millennial populations), it’s intuitive for the AVMA to encourage the responsible use of this practice. This being said, there are some vital best practices that still need to be followed without exception. Primarily, it’s widely believed that telemedicine should only occur as an *extension* of the care that occurs within an existing “Veterinarian-Client-Patient-Relationship” also known as *VCPR.*
This is crucial for several reasons. Of course, the convenience of digital communication and telemedicine in general makes it a practical option for busy pet owners and veterinary practitioners alike. However, if a pre-existing relationship doesn’t exist, the core knowledge needed to make accurate, intelligent and real-time assessments of health simply won’t be there.

Take the following scenarios into consideration for example.

**Scenario 1:** Pet owner “Timmy” has been seeing his veterinarian for years. This veterinarian has comprehensive paperwork for Timmy’s Labrador Retriever on file, including age, blood results, vaccination history, diet and notes from a recent physical examination.

Timmy is away on a family vacation, when his dog begins to show signs of agitation, including excessive panting and a lack of appetite.

Based on his existing VCPR, Timmy engages in a video call with his veterinarian to explain the situation and even to show his Labrador exhibiting symptoms on camera. Based on the call and symptoms provided, Timmy’s veterinarian makes some quick dietary suggestions to ease discomfort in his pet and tells him to see a local emergency veterinarian if the symptoms do not subside in 24-hours.

**Scenario 2:** Pet owner “Judy” has recently moved to a new city and doesn’t have an active VCPR for her Persian cat. Over the course of one weekend, her cat seems to lose her appetite almost completely, and also shows behavioral changes like becoming fatigued and reclusive. Judy attempts to call and text several local veterinarians in the area to get their opinion over the phone.

However, none of these veterinarians have any paperwork to reference and cannot provide an accurate assessment.

In this scenario, although Judy attempts to use the convenience of telemedicine, she does so inappropriately because no VCPR exists. The example of ‘Scenario 2’ is actually dangerous because it could cause an inaccurate assessment of the symptoms. Here, telemedicine is simply not an appropriate solution.

Most global veterinary associations clearly assert that although telemedicine is convenient, it still must be comprehensive. They often state the following:

“The veterinarian is expected to provide oversight of treatment, compliance, and outcome, as well as document the patient’s continuing care and treatment in the medical record. The animal owner’s consent for the use of telemedicine must also be obtained and documented.”

While the growing field of telemedicine provides incredible opportunities for remote consultations and care that’s more accessible and convenient than ever before, it
cannot supplant the traditional Veterinarian-Client-Patient-Relationship. This being said, it can complement this relationship by providing more options to both the veterinarian and pet owner in optimizing the overall health of their pet.

Ultimately, if both pet owners and veterinary practices handle the opportunity for remote care and telemedicine responsibly, ongoing benefits to the world of veterinary medicine may be profound. When technology allows us to facilitate more accessible care for pets and their owners, it's a win-win. When veterinarians can provide consult more regularly and conveniently to their patients, it's a win-win-win.

As long as we remember to be mindful and ensure that we use telemedicine as an extension of traditional in-person care, and not a replacement, we can look forward to seeing telehealth continue to grow as a result.

E-Commerce

For the past decade, online pharmacies have grown in popularity, attaching themselves from one veterinary practice to another. It's no surprise that the turn towards online pharmacies has only expanded in the wake of COVID-19. With quarantine in place along with the ongoing need for social distancing, veterinarians across the country and around the world have had to come up with all kinds of creative and innovative options for clients such as contactless delivery, curbside pickup, and personalized telehealth appointments.

But even before COVID and the rush to go remote, the need for veterinary practices to move online had been a long time coming...

When I first was made aware of the growing competition in the online veterinary pharmacy space, the predominant concern was the constant presence 1-800-PetMeds. Now we're looking at Chewy, Fosters and Smith, and even Walmart rapidly moving into this landscape as well. That's not to say that we should treat these businesses like the enemy. There's actually a lot we can learn from their business models if we're willing to look at what makes them so successful.

Competition with online pharmacies isn't just about which company can offer the cheapest product or the best value - although, it’s important to note that those features are certainly major deciding factors for consumers, too. At the end of the day, it all comes down to one thing - convenience.

In this time of quarantine and physical distancing, the demand for online shopping has risen to the point where online retailers have grown overwhelmed by the sudden influx in requests. Major retailers like Amazon have had to hire on as many as 75,000 more workers in order to keep up. With this increase in demand, big or small, this is a great time for online pharmacies to get started.
I can think of many ways I benefit from the convenience of online shopping. Thanks to subscription services, I cannot remember when I last ran out of my preferred daily vitamins or every day household items. I rarely ever even have the chance to run out of anything I need.

My dog food is no different. The convenience of my veterinary practice’s online service means monthly deliveries are just a click away. Sure, sometimes that means I end up with too much pet food but changing my delivery preferences is simple. Knowing I have certain items squared away on a regular basis means one less thing for me to worry about. That may just be the biggest value proposition of them all...

The buying power of convenience can’t be overstated. We can harness this power for ourselves, along with the trust that comes with being a practice your clients know and believe in. But how do you get started?

Your online pharmacy integrates with your management software, providing your clients with a digital prescription that can only be used through your online pharmacy. It seamlessly automates requests and prescriptions for your clients on a regular basis. All of the moving parts: the delivery process, technology upkeep, warehouse, staffing costs, etc. are all built into this system and pricing. Sure, it may cost you more to sell some items from your online store than directly from your distributor, but you don’t have to worry about extra operating costs (or headaches).

It’s systems like these that make online pharmacies and services like Chewy and 1-800-PetMed so successful, and your clients appreciate simplifying their pet care needs. When we consider our own shopping habits, and what draws us to purchase a product or service, especially online, we can’t fault our clients for demanding the conveniences that other online stores regularly provide.

If at this stage, your practice is still looking for a sign to create an online pharmacy, this is it. Even as we begin the process of returning back to business as normal, online shopping and its conveniences aren’t going to go away. In fact, they’ll continue to accelerate.

If you already have this option for your practice, congratulations on meeting step number one! The next step is to promote it.
**About the Author:** Eric Garcia is an IT expert. Digital marketer. Industry thought leader. When it comes to helping veterinary practices streamline their technology and attract and retain clients, Eric Garcia has a proven track record of educating the industry and producing results. Eric is an IT and Digital Marketing consultant working exclusively with veterinary practices. In addition to a long list of satisfied clients, Garcia’s work has been recognized throughout the industry. He speaks regularly at conferences all throughout the world. **Facebook:** facebook.com/EricGarciaFL  **Instagram:** @EricGarciaFL
Getting Clients to Say Yes to Your Recommendations

Eric Garcia
Simply Done Tech Solutions, LLC
Tampa, Florida, USA

TELL YOUR STORY

People are often under the impression that Facebook is solely about peer-to-peer interactions. This, however, couldn’t be further from the truth. Facebook is a platform that’s become as universal as the water cooler itself. Successful veterinary practices around the world leverage Facebook as a place to tell their unique story. Your veterinary practice has a narrative; a year it was founded, a founder (or two, or more) and a style and perspective that makes it entirely unique.

Use Facebook to tell your story and not only capture, but captivate your audience!

Tell us about your success stories: the pets that you care for and the difference that you’ve made today. All of these things foster community, trust, interactions, and keep your trusted pet owners coming back for more.

These success stories are technically known as:

Case studies – a story particular to a specific pet, place and time.

These case studies are of crucial importance for a multitude of reasons, but primarily because they help your audience to see firsthand the type of stellar care that your veterinary practice provides!

In a particular case study, be sure to provide your audience with:

- Why the pet came in to receive veterinary care
- What you did to provide care for the pet
How the pet is doing today

A photo, or quick video of the pet!

By providing this level of in-depth information on a pet, you tell the story of your patient and ensure that you can deliver the same quality of care to any prospective pet owner who needs it. You’ll be able to forge an immediate bond with pet owners who appreciate your attention to detail, and the accountability needed to provide optimal care for a pet.

People want to hear of your successes, which will brighten their day and instill them with confidence about your veterinary practice. In exceptional circumstances, news coverage has even come about after particularly sincere and uplifting pet stories. This results in absolutely tremendous publicity, and simultaneously helps you to market your services to a wider audience. This wider audience can soon grow and enhance your veterinary practice online, and in the local community.

Case studies are also a great opportunity to educate your clients. By highlighting a particular toxicity (like xylitol, grapes, or lily toxicity in a cat) you can spread the important information in a success story that will resonate with pet owners. These posts can be timed for specific times of year (the “chocolate holidays”, the start of flea season, holiday dangers) to help your clients stay aware of how to best care for their pet, and to keep your practice at top of mind.

GET PERMISSION

Yes, you should receive permission from a pet owner to share their story, pictures or a video of their pet on Social Media or elsewhere. This is an important thing to note and emphasize, as some members of your staff may be appointed to collect signed Photo/Video Release Forms, to ensure that you’re permitted explicitly to share various types of media.

Most pet owners don’t hesitate at the opportunity to share the joy of their pet with the world and online, but receiving permission firsthand is definitely a must.

Sample topics for case studies can include:
By using Facebook, photos, and videos to create and communicate compelling stories, you can enhance your marketing efforts, stay on the cutting edge, and attract more pet owners to your veterinary practice.

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How To Handle Online Haters

Eric Garcia
Simply Done Tech Solutions, LLC
Tampa, Florida, USA

As with most industries over the past few decades, the veterinary field has gone virtual — and with benefits ranging from online pharmacies to simpler appointment scheduling and telehealth visits, to online advertising, and of course, greater access to client feedback thanks to social media, there’s a lot to be grateful for.

But this level of access also leads to much simpler (and more anonymous options) for leaving hate. Negative comments or constructive criticism is one thing: cyberbullying is another.

20% of the American Veterinary Medical Association (AVMA) members have reported encountering some form of cyberbullying from vicious reviews to threats of physical harm.

As a world and as a profession, we’ve forgotten the Golden Rule, and it’s time we face this problem before it causes any more harm to our field.

Here are some tips I shared at the virtual 2020 PSIvet Business Symposium, to help you determine what you should do if you or your practice start to encounter online harassment:

- **Leave private hate private.** Facebook groups and other niche forums can both bring about a great sense of community and (unfortunately) an even greater sense of entitlement. It’s amazing what people feel confident to say with their real photos and names attached to their profiles — and it only gets worse when there’s greater anonymity. Somehow typing a rude response feels like less of a risk than insulting someone in person, but whether you feel tempted to argue their point or defend your practice on any of these forums you’re better off leaving it alone. They did not write this comment to you — you are not going to change their mind. You’re better off leaving the debate to other people within the private group - and chances are they’ll move onto another topic by the time you’ve formulated your response anyway. The more attention you give the hate (through comments or replies) the more you can unintentionally add fuel to an already dying flame.
• **Take a moment before engaging with negative reviews.** When a client has lashed out online, you are better off giving them anywhere from 24-72 hours to cool down before you reach out personally. This means pulling out their pet’s medical records, picking up the phone, and seeing what you can do to help them in person first, before settling for responding to their comment online. If they are unavailable, then your first step is to publicly apologize for their experience and note that you haven’t been able to get a hold of them. This way they (and anyone else who sees the negative comment) will know that you and your practice are trying to make things right. Apologizing isn’t about admitting that you or your practice is in the wrong: it’s about expressing empathy and acknowledging the client’s perspective — you cannot fix a situation without first acknowledging their feelings. Although it’s tempting to share your side of the story in your apology, avoid discussing anything about their pet’s case online — especially if it’s on a public forum. There is still client confidentiality at risk, and you could get reported to your state board. Yikes.

**Sample Reply to a Negative Review**

“We’re sorry for your experience. We have not been able to reach you by phone. We would like to make this situation right with you. Please call our medical director at (xxx) xxx-xxxx.”

• **Report slander and false reviews.** There’s a difference between an angry (but honest) client review and a false one. The good news is that with Google and Yelp and other public reviews, there are actual guidelines in order to protect businesses like yours. If a review is fake, false or misleading, or otherwise in violation of the review’s host site, you can send requests for their removal and get them taken down.

Here are some simple steps you can take to get false reviews taken off Google and Yelp:

**Google**

• Log into Google My Business at [google.com/business](http://google.com/business).
• Select “Reviews” from the menu.
• Find the review in question. Click the 3-dot menu, then select “Flag as inappropriate.”

**Yelp**

• Claim your business page at [biz.yelp.com](http://biz.yelp.com).
• Locate the review in the “Reviews” section of your business account.
• Click the 3 dots and click “Report Review.”
Choose the reason for removal from the dropdown list.
  - Grounds include containing false information, threats, lewdness or hate speech, not describing a personal customer experience, and being posted by a competitor or ex-employee.

**Overwhelmed? You don’t have to go it alone.** It can be emotionally draining and generally exhausting to find yourself and your place of work under attack. The good news is that AVMA has its own cyberbullying hotline to help veterinary professionals of all types with immediate support and a free counseling session to help get your practice’s reputation back to what it used to be. Simply call (626) 531-1140, and you’ll have a safe space to discuss your concerns and your side of the story without ticking anyone off or violating confidentiality guidelines.

**Stop vet-on-vet harassment.** Unfortunately, not all cyberbullying comes from clients. Whether it’s coming from competitions veterinarians or unhappy staff, veterinary professionals should know better than engaging in unnecessary online harassment campaigns — especially when they consider that the veterinary field is already a profession that is prone to suicide. When you have a problem with another vet, you can do much better than calling each other out in negative ways on social media. We all have bad days: venting to a friend is a much better idea than spilling all of your problems on Twitter or Instagram. And if you’re upset about something unfair or unjust happening in your profession, write to your state board or national governing bodies, or write an article or create a series of podcasts — make something positive for change instead of giving into simply complaining for sympathy points or petty revenge.

**And finally, skip social media with your breakfast.** One of the number one rules of the internet is “don’t read the comments” but you can extend that to mindless scrolling on social media, too. Of course, as a business, you do need to respond to legitimate criticism and reviews but if you want to be happier, start your day without checking social media. You’ll be glad you did.

**About the Author:** Eric Garcia is an IT expert. Digital marketer. Industry thought leader. When it comes to helping veterinary practices streamline their technology and attract and retain clients, Eric Garcia has a proven track record of educating the industry and producing results. Eric is an IT and Digital Marketing consultant working exclusively with veterinary practices. In addition to a long list of satisfied clients, Garcia’s work has been recognized throughout the industry. He speaks regularly at conferences all throughout the world. **Facebook:** facebook.com/EricGarciaFL **Instagram:** @EricGarciaFL
Plenary Sessions
INTRODUCTION:
DEI can feel like vast and insurmountable topics. Veterinary care professionals may struggle to understand why DEI is important to our industry. Our focus on DEI can be explained by going back to our oath. The veterinary oath begins with this sentence. “As a member of the veterinary medical profession, I solemnly swear that I will use my scientific knowledge and skills for the benefit of society.” The first sentence of our oath points our profession to the general public first, not to animals. The oath goes on to state “I will perform my professional duties conscientiously, with dignity, and in keeping with the principles of veterinary medical ethics.” Our oath requires that we actively think about professional ethics. “I will strive continuously to improve my professional knowledge and competence and to maintain the highest professional and ethical standards for myself and the profession.” Finally, our oath encourages us to strive for continual improvement both personally and professionally.

Diversity, Equity and Inclusion (DEI) concepts in veterinary medicine are based on the understanding that the veterinary profession exists to serve its customer, the public. Not only is the public the veterinary services’ customer, but also the public represents the talent pool of future veterinary health professionals. Diversity, equity and inclusion work together to allow individuals from diverse backgrounds to be equipped with what they need in order to be their authentic selves as their unique contributions are welcomed and appreciated. As a regular part of their work and lives, veterinary professionals meet and interact with culturally diverse people and groups. While the diversity of people and groups interacted with can be assumed the need for equity and inclusion cannot be understated. Data from the Bureau of Labor and Statistics indicates that veterinary medicine is one of the least diverse professions in the U.S. The sustainability of the veterinary profession is tied to both the supply of veterinary care providers and the demand for veterinary care from animal owners. Taken together, diversity, equity and inclusion enable fulfillment of the veterinarian’s oath; to promote public health, practice conscientiously and engage in lifelong, continual improvement. It is helpful to review data regarding the Canadian public in an effort to understand the importance of DEI in the Canadian veterinary medical profession.

Data from Statistics Canada indicates a shift in ethnocultural diversity for Canada. At the time this manuscript was written, it was projected that by 2041, half of the Canadian population would be made up of immigrants and their Canadian-born children. In 2041, 1 in 4 Canadians will be born in Asia or Africa. The racialized population in Canada was 22.2% in 2016 and it is expected to grow to anywhere from 38.2%-43% of the Canadian population by 2041. This population expansion will be dominated by youth. Over 1 million Canadians identify as part of the LGBTQ2+ community and one third of this community’s members are younger than 25 years old.

A 2020 survey of 1445 veterinarians in western Canada indicated that 68.4% of practitioners identified as female and females outnumbered males in the youngest age of practitioners (26-35 years of age). Companion animal, mixed animal and food animal were the most frequent types of work settings identified. Males worked more full-time hours than female practitioners. Males
were also 1.7 times more likely to be practice owners. This survey also showed a decreasing trend in practice ownership. Our profession has more female representation, less rural/food animal practitioners and fewer individuals pursuing individual practice ownership.

**Definitions:** The following definitions are provided to give common language to the discussion of DEI. Common language allows individuals to have richer dialogue about topics that may at first glance appear to be uncomfortable. Listening with empathy, seeking to understand and valuing different perspectives are all tactics that will remove the fear of saying or doing the wrong thing and replacing it with the belief that a respectful dialogue leads to increased awareness:

1. **Antiracism:** actively opposing racism and supportive of a truly just and equitable society.
2. **Diversity,** according to Jeanne McNatt at Northeastern University, consists of the differences in age, culture, education, ethnicity, experience, gender, race, religion, sexual orientation and others that make people unique. Diversity can be thought of in dimensions. Primary dimensions of diversity are often visibly identifiable. Primary dimensions of diversity include gender assignment at birth, age and select physical qualities.
3. **Equity** is defined as “the state, quality or ideal of being just, impartial and fair.” The concept of equity is synonymous with fairness and justice. It is helpful to think of equity as not simply a desired state of affairs or a lofty value. **To achieve and sustain equity,** it needs to be thought of as a structural and systemic concept.
4. **Equity vs. Equality** Equity involves trying to understand and give people what they need to enjoy full, healthy lives. Equality, in contrast, aims to ensure that everyone gets the same things in order to enjoy full, healthy lives. Like equity, equality aims to promote fairness and justice, but it can only work if everyone starts from the same place and needs the same things.
5. **Inclusion** The action or state of including or of being included within a group or structure. More than simply diversity and numerical representation, inclusion involves authentic and empowered participation and a true sense of belonging.
6. **Inclusive Culture:** An organizational environment that allows people with multiple backgrounds, mindsets and ways of thinking to work effectively together and perform to their highest potential in order to achieve organizational objectives based on sound principles.
7. **Individual Racism:** A personal belief in the superiority of one’s race over another.
8. **Institutionalized Racism:** The imposition by an organization as a whole of philosophies, guidelines and practices that have the intent or effect of promoting systematic, discriminatory treatment of persons on the basis of their race.
9. **Internalized Racism:** Acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth.
10. **Microaggressions:** Brief and commonplace subtle insults (verbal, nonverbal and/or visual), often unintentional, that communicate hostile, derogatory or negative messages to target persons based solely upon their marginalized group membership.
11. **Psychological Safety:** A shared belief that a workplace or a workplace team is safe for interpersonal risk taking. This term is not meant to suggest a careless sense of permissiveness nor an unrelenting positive affect, but rather a sense of confidence stemming from mutual respect and trust that a workplace or workplace team will not reject, embarrass or punish someone for speaking up.
12. **Racial Microaggression**: Brief and commonplace subtle insults (verbal, nonverbal and/or visual), often unintentional, directed toward people of color automatically or unconsciously that communicate hostile, derogatory or negative racial slights and insults.

13. **Racial Privilege and Racial Oppression**: Like two sides of the same coin, racial privilege describes race-based advantages and preferential treatment based on skin color, while racial oppression refers to race-based disadvantages, discrimination and exploitation based on skin color.

14. **Sense of Belonging (at work)**: The extent to which individuals feel they are a part of, included in and connected with people at their organization. Creating a sense of belonging—an employee’s perception of acceptance within a given group—provides HR leaders a good opportunity to reinvigorate their inclusion approach and goals. Belonging is a key component of inclusion. When employees are truly included, they perceive that the organization cares for them as individuals, their authentic selves. HR can help make that happen. Three elements foster belongingness: diminished outsider practices, enhanced caring mechanisms and transparency.

15. **Sex-Based Discrimination**: Any act that involves treating someone unfavorably because of that person’s sex. This includes discrimination against an individual based on gender identity (including transgender status) or sexual orientation.

16. **Stereotype**: A set of cognitive generalizations about the qualities and characteristics of the members of a group or a social category. Like schemas, stereotypes simplify and expedite perceptions and judgments, but they are often exaggerated, negative rather than positive, and resistant to revision even when perceivers encounter individuals with qualities that are not congruent with the stereotype.

**APPLYING DEFINITIONS**: The above definitions cover a lot of ground. For the sake of our discussion let us focus in on our three key terms diversity, equity and inclusion.

**DIVERSITY** acknowledges and celebrates our uniques across gender, ethnocultural, religious, and cultural spectrums. Diversity is evidenced by the mix of persons, perspectives even geographical regions present among individuals. To use a colloquialism diversity can be thought of as who is present. Diversity can be grouped into primary and secondary dimensions. Primary dimensions of diversity are those which are visible such as ethnocultural, age, (certain) physical capabilities and certain religious beliefs. Secondary dimensions of diversity are not seen but have to be discovered. These can include socioeconomic status (SES), marital status, military service, certain physical capabilities and sexual orientation.

Ashely Stahl in the *3 Benefits of Diversity in the Workplace* cites the many benefits associated with diversity in the workplace. Diverse teams make better decisions than homogenous teams 87% of the time. Diverse teams offer broader perspectives and bring more information to the table. Teams outperform individuals when making decisions and that improves as diversity improves. Diversity creates a safe place for employees.

**EQUITY** asks if all individuals present have what they specifically need to actively participate. Equity is distinctly different from equality. Equality is the practice of treating everyone exactly the same. Treating everyone the same should be avoided as it
completely overlooks the individual. Needs and wants are presumed rather than solicited when all are treated equally. To provide a visual demonstration, when individuals are treated equitably, a shorter individual may be given a box to stand on in order to see over a fence. An individual using a wheelchair as a means of transportation would have no ability to use a box to see over a fence; an elevated ramp however would be appropriate.

**INCLUSION** is really the most important concept we will discuss during this presentation. AFOA Canada describes it as “the act or process of fostering an environment where those within diverse categories feel welcomed and valued”. The sum of dimensions of diversity equate to a whole person. When thinking about inclusion individuals should feel comfortable in bringing their whole selves to the work site. To use a colloquialism inclusion identifies who in the room has the power to lead and make decisions. The lack of inclusion is what often leads to the leaky bucket syndrome of retention efforts. If diverse individuals are brought into worksites but not included in strategic development and/or decision making they are likely to leave organizations. Feeling undervalued and underappreciated they are likely to seek organizations where they feel valued and appreciated. Taken together Bersin by Deloitte defines diversity and inclusion (D&I) as the variety of diverse people and ideas within a company, and the creation of an environment in which people feel involved, respected, valued, connected and able to bring their authentic selves (i.e., their ideas, backgrounds, values and perspectives) to the team and to the business. Canadian businesses have been investing more in D&I since 2009. The top three reasons Canadian organizations invest in D&I are to enhance employee engagement, enhance the ability to acquire new talent and to be branded as socially responsible. In the same Deloitte study the US the top three reasons organizations invest in D&I are to serve customers better, increase speed and agility.

**Final Thoughts:** Often our pivotal DEI learnings have occurred at home, having been taught to us by people that we love and trust. DEI are not only complex topics but topics that we hold longstanding opinions about. The work of improving DEI is difficult as it requires self-awareness and recognition of the history of nations, individuals and organizations. The public we have is the general public that represents clients, producers, customers and veterinary professionals. Intentionally, society is the first group impacted by veterinary medicine in our oath, not animals. Our profession focuses on human beings and they animals they live with and around. It should suffice to say that DEI is important because we are members of society serving society. DEI has been ushered into the forefront of society’s attention due to recent national and international social unrest incidents. Zellenials (Gen Zers and millennials) who are the most diverse cohort of society we have ever had, increasingly expect that worksites reflect societal diversity. We owe it to society to sustain the profession of veterinary medicine. The business case for DEI in veterinary medicine is that in order to benefit society we have to be consciously aware of who makes up our society. Aware of the society our profession seeks to benefit we must then commit to lifelong learning to move our profession forward. DEI allow us to fully embrace all members of society.

**RESOURCES:** It is important to devote time to self-study in understanding how personal beliefs, conscious and unconscious biases affect one’s demonstration of DEI. As we come to understand ourselves better, in all of our dimensions of diversity, we are more aware that others have similar dimensions of diversity. Being cognizant of
one’s dimensions of diversity allows individuals to model inclusive and equitable behaviors as they invite others to be their authentic selves. We show up as our authentic selves when we allow ourselves to display all of our dimensions of diversity at work and at home. There are numerous resources available to better understand DEI. The best resources are those that are frequently utilized as individuals and groups move along the spectrum of comprehension and demonstration of DEI concepts.


2. Canada in 2041: A larger, more diverse population with greater differences between regions.


7. 10 steps businesses can take to improve DEI in the workplace https://www.forbes.com/sites/ashleystahl/2020/07/21/10-steps-businesses-can-take-to-improve-diversity-and-inclusion-in-the-workforce/#30a226bf343e

8. AAVMC Diversity Assessment https://docs.google.com/spreadsheets/d/1cW4F25fB9wWxvM28HwDP5A-HgW_UX1-i0v/bFY-ggkOY/edit#gid=1833355709


15. Culturally Responsive Leadership https://culturallyresponsiveleadership.com/


17. How HR professionals can help staff talk race relations at work https://associationsnow.com/2020/08/how-hr-professionals-can-help-staff-talk-race-relations-at-work/?utm_medium=email&utm_source=rasa_io

Cat Friendly Practice: Because You're Worth It!
Kelly A. St. Denis, MSc, DVM, DABVP (feline practice)

The Cat Friendly Practice (CFP) Program recently celebrated its 10 year anniversary with the publication of 2 new guidelines, the 2022 AAFP/ISFM Cat Friendly Interactions Guidelines\(^1\) and the 2022 ISFM/AAFP Cat Friendly Veterinary Environment Guidelines.\(^2\) AAFP member clinics can utilize these guidelines and the CFP Program criteria checklist to dramatically improve the care provided to cats in the practice. AAFP members can also attain individual certification as a Cat Friendly Veterinarian, Cat Friendly Veterinary Professional or Cat Friendly Veterinary Advocate. Over 88% of caregivers feel that their cat's welfare is impaired before, during and after the veterinary visit.\(^3\) Half of caregivers indicated that they were also stressed. Full body restraint of feline patients is still a commonly used approach in veterinary clinics in cats of all demeanours.\(^4\) Handling methods have been connected with perceived patient stress by caregivers and may prompt caregivers to seek care for their cat elsewhere.\(^3\) We need to be Cat Friendly if we hope to get cats back into practices for both routine wellness and sick cat care, and if we hope to improve our feline interactions, thereby earning and keeping the trust of caregivers.

**What is Cat Friendly?**
Simply put, Cat Friendly respects the cat as an individual while still achieving the required clinical outcome. Cat Friendly team members understand, interpret, and appropriately respond to a cat's emotional states. The goal is to give cats a perceived sense of control throughout the veterinary experiences, thereby reducing the protective emotions of fear, anxiety, and frustration.\(^1\) The environment is developed to promote positive (engaging) and reduce negative (protective) emotions in the cat.\(^2\)

**Is it worth it?**
Many practice owners and managers worry that the changes needed will be extensive and expensive. They worry that there will be no return on investment should they decide to make changes. Data are now available that clearly show the financial benefits of being a CFP, with even the smallest change having potentially big impact. CFP certification does not require renovations and there are many creative, inexpensive solutions limited only by our imagination.

**The Business Case**
A retrospective study in Spain comparing Cat Friendly Clinics (CFC; the European equivalent to CFP) to non CFC practices showed that CFCs had a greater percentage of feline patients to total active patients.\(^5\) CFCs also had a higher percent of their revenue from feline patients, 12% higher transaction value with feline patients and 30% higher frequency of visits/year/feline patient. Feline caregivers spent 45% more per year at the CFCs studied, and 40% were more likely to buy pet food from their practice. Additional data presented at the 2022 AAFP Annual Veterinary Conference in Pittsburgh showed that CFPs not only have increased feline visits and revenue per year, CFPs also had a higher proportion of clinical diagnostics, finding more health conditions. These data were attributed to the improvement in Cat Friendly interactions allowing in more successful sample collections.\(^6\)

**A Reduced Risk of Injury**
The AVMA Professional Liability, Business and Personal Insurance group, as well as HUB international, in partnership with veterinary industry-specific insurance carriers, analyzed workers’ compensation data claims from 2015 to 2018, comparing CFP to non-CFPs.\(^7\)
CFPs had a lower overall cost per workers’ compensation claim, lower animal contact cost per workers’ compensation claims and reduced modification factor. The latter decreased the cost of the workers’ compensation insurance premium. CFCs have also been shown to have a lower cat-related injury rate.

**Cat Friendly is Better for Everyone**

From cat to caregiver to veterinary team, CFP practices report improved knowledge (94%), increased visits (86%), increased revenue (75%) and positive team dynamic impact when handling, treating and caring for cats (94%). Top benefits in the 2021 CFP Survey included less stress on feline patients, higher satisfaction among current cat clients, improved client retention, and more staff time and attention with each feline patient.


6. 2022 AAFP Annual Conference


8. 2021 Cat Friendly Practice Survey Results
NODULAR LUNG DISEASE IN THE DOG: IS IT BLASTOMYCOSIS OR METASTATIC CANCER?
Jinelle A. Webb, DVM, MSc, DVSc, Diplomate ACVIM (Small Animal Internal Medicine)
VetLink Mobile Imaging, Oakville, Ontario

Introduction

When a dog presents with respiratory signs, thoracic radiographs are one of the most common diagnostic tests performed. Some dogs will have thoracic radiographs performed without respiratory signs, such as prior to general anesthesia in an older dog, or as a metastatic disease check when a non-thoracic mass in noted. The presence of a diffuse, nodular lung pattern (sometimes casually referred to as a ‘snow storm’ pattern) is concerning for either blastomycosis or metastatic cancer. Other radiographic patterns can also suggest either of these diseases. Reaching a quick diagnosis is imperative, as the implications of diffuse metastatic pulmonary disease, or diffuse pulmonary blastomycosis, are significant. The ability of blastomycosis to mimic metastatic lung disease can lead a clinician to suspect a grave prognosis for their patient. Blastomycosis, although a potentially fatal disease, has the potential for a cure, whereas a cure is highly unlikely for metastatic lung disease.

Presentation

Blastomycosis:

Dogs with blastomycosis typically present with cough, exercise intolerance, tachypnea, cyanosis and/or respiratory distress. It would be extremely unlikely for a dog with pulmonary blastomycosis to have an absence of respiratory symptoms. They are more commonly younger (1-5 years old), and are often large breed, male intact dogs living in endemic areas.

Metastatic pulmonary cancer:

Dogs with metastatic pulmonary disease may or may not present with respiratory signs. When respiratory signs are present, these often include cough, hemoptysis, tachypnea, and exercise intolerance. Pulmonary metastatic disease is most commonly seen in pets with hemangiosarcoma, osteosarcoma, carcinoma, histiocytic sarcoma, soft tissue sarcoma, adenocarcinoma, melanoma, and lymphoma. The age of dogs presenting with pulmonary metastatic disease is much higher than those with pulmonary blastomycosis; most dogs are senior or geriatric.

Diagnostic Options

Most dogs presenting with pulmonary blastomycosis or pulmonary metastatic disease will have significant respiratory signs, which will warrant thoracic radiographs. There are several different radiographic patterns that can be present with both blastomycosis and metastatic lung disease, and some pets will have more than one pattern. A summary of the patterns is provided below.

Blastomycosis can have a variable radiographic appearance. The classic appearance is a large number of small, diffuse pulmonary nodules. However, many different radiographic patterns can be seen with blastomycosis, such as a solitary mass mimicking a primary lung tumour, a smaller number of variably sized nodules, or a bronchial or alveolar pattern. Pleural effusion is noted in a small number of cases.
Dogs with metastatic lung disease most commonly present with diffuse, smaller nodules, especially in cases of metastatic hemangiosarcoma. However, dogs with osteosarcoma often have a smaller number of very radio-opaque, medium to large nodules present. Very few cases of metastatic lung disease will present with an alveolar or bronchial pattern, however it is possible. If hemorrhage has occurred, pleural effusion can be present, however it is uncommon to have neoplastic cells present within the pleural effusion. If lameness or bone pain are noted, radiographs of the area with pain are indicated.

Radiographic Appearance

<table>
<thead>
<tr>
<th>Location</th>
<th>Appearance</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alveolar</td>
<td>Focal uniform soft tissue opacity, usually with distinct margins; air bronchograms</td>
<td>Not commonly seen with blastomycosis or metastatic lung disease, but is possible with either</td>
</tr>
<tr>
<td>Bronchial</td>
<td>Rings and lines through lung field, including periphery</td>
<td>Not commonly seen with blastomycosis or metastatic lung disease, but is possible with either</td>
</tr>
<tr>
<td>Interstitial—nodules or miliary pattern</td>
<td>Multiple diffuse small to variably sized nodules</td>
<td>Commonly seen with blastomycosis and metastatic lung disease, especially hemangiosarcoma</td>
</tr>
<tr>
<td>Larger nodules, solitary masses</td>
<td>Solitary mass or smaller number of larger nodules</td>
<td>Solitary masses have been reported with blastomycosis. Metastatic lung disease would rarely present with a solitary mass, but may present with a small number of larger masses.</td>
</tr>
</tbody>
</table>

Ultrasound examination can be of use in cases of suspected metastatic lung disease and blastomycosis. Thoracic ultrasound can evaluate larger, peripherally located pulmonary masses, and assess for occult pleural effusion, pericardial effusion, and mediastinal masses or lymphadenopathy. Thoracic ultrasound may reveal an abnormal area that can be sampled. Abdominal ultrasound is useful in cases with a suspicion of metastatic neoplasia, as many cases with metastatic lung disease have primary abdominal neoplasia.

CT scan can provide more anatomic information for cases with pulmonary nodules, can give information about occult intrathoracic masses, lymphadenopathy, and mild pleural effusion, and can be used to screen the abdomen for primary neoplasia. However, as CT scan requires heavy sedation or general anesthesia, it is less ideal for dogs with respiratory compromise. Much of the information obtained can be found on ultrasound examination, for which little to no sedation is needed.

Laboratory Testing

Cytology – In many cases of metastatic lung disease and pulmonary blastomycosis, material for cytology is obtained via bronchoscopy and bronchoalveolar lavage, or occasionally via transtracheal wash. If blastomycosis is present, there is a good chance of obtaining a diagnosis. However, if metastatic lung disease is present, then exfoliation into airways is low and it is uncommon to reach a diagnosis. If sputum is expelled during coughing, this can be examined cytologically.
If a large enough nodule is present in the periphery of the lung to allow ultrasound guided fine needle aspiration, this material can be assessed cytologically. In cases of blastomycosis, attaining a diagnosis is quite likely. For metastatic lung disease, neoplastic cells may or may not be present in the obtained material. However, there is a higher likelihood of obtaining a diagnosis via ultrasound guided lung aspirate as opposed to bronchoalveolar lavage, as cells typically are within lung parenchyma versus airways.

If pleural effusion is present, material can be obtained via thoracocentesis. Cytology of pleural effusion in cases of blastomycosis will result in a diagnosis as long as organisms are present within the pleural effusion. This is also the case for metastatic lung disease, however neoplastic cells are not commonly present in the pleural effusion.

**Histopathology** – it is uncommon to obtain material for histopathology in dogs with blastomycosis or metastatic lung disease. If a consolidated lung lobe or abscess is removed, and is the site of blastomycosis or metastatic lung disease, then histopathology is likely to be diagnostic.

**Culture** – material obtained via bronchoalveolar lavage, pulmonary fine needle aspiration, thoracocentesis of pleural effusion or expelled sputum can be submitted for fungal culture, however it is unlikely to result in a diagnosis for blastomycosis.

**Antigen and Antibody tests** – There is an immunoassay available for the detection of *Blastomyces dermatitidis* antigen in urine that is highly sensitive, especially for cases with pulmonary infection. This test is recommended both for initial diagnosis, and monitoring response to therapy. The difficulty in using this test is the timeline to receive results, which can be several weeks. In a more critical patient, this timeline may render the test not useful for the diagnosis of that patient. In addition, it is an expensive test. Serological testing (antibody levels) has not proven very useful.

**Summary**

Dogs with pulmonary blastomycosis or metastatic pulmonary disease can have very similar presentations, and it can be challenging to obtain a diagnosis, especially in more critical patients. Utilization of the pet’s signalment, history, physical examination and thoracic radiographs can help with determining the level of suspicion for each of these two disease processes. An older dog with a mild cough or no respiratory symptoms and a diffuse, nodular pulmonary pattern, especially if hemoptysis is present, is much more likely to have metastatic lung disease than blastomycosis, and warrants an abdominal ultrasound as the next diagnostic step. A young, male intact hunting dog from a region endemic to blastomycosis, that presents with a marked, productive cough and has a diffuse, nodular pulmonary pattern present, is much more likely to have blastomycosis and therefore warrants cytology or a urine antigen test. However, it is important to remember that both blastomycosis and metastatic lung disease can occur in any signalment of dog, and presentation and radiographic appearance do not always match what is classically expected of either disease. Dogs with pulmonary blastomycosis, although requiring expensive treatment of a long duration with close monitoring, can often have a cure and great long term prognosis. Dogs with metastatic lung disease, while having options for improvement in cancer burden and quality of life in some cases, usually have a poor prognosis.

References available from the author upon request.