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# 2019 OVMA CONFERENCE & TRADESHOW

WESTIN HARBOUR CASTLE, TORONTO, ONTARIO, JAN. 31 – FEB. 2, 2019

## CONFERENCE PROCEEDINGS

# FOREWORD

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# **SMALL ANIMAL PROGRAM**

## MANAGING DOGS AND CATS WHO HATE THE VET

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Every veterinary staff member knows the dread of seeing a WILL BITE sticker on a patient file. Immediately, they know that this appointment will be more difficult and time consuming than a typical appointment. Aggressive pets can drain the practice's resources as increased time is spent handling them and the risk to staff members is increased. When dealing with an aggressive animal even the simplest things such as giving a vaccination becomes difficult. Considering the time, trouble and potential risk to the staff, it is easy to understand why some veterinarians are reluctant to treat this subset of patients.

Providing medical care to aggressive pets doesn't have to be stressful. While there will always be animals who will have to be sedated for examinations, most patients can be handled safely in the veterinary hospital without sedation. The keys to providing low-stress, medical care for an aggressive patient are: 1) changes in management of the patient; 2) alternative restraint methods; 3) in clinic behavior modification; 4) staff and doctor education; and 5) at home behavior modification. The ability to handle an aggressive patient with the least amount of stress to the patient increases client retention and patient welfare. In addition, the patient's aggression will decrease over time making her easier to handle at subsequent appointments.

Why do patients act aggressively at the veterinary hospital? Most frequently, it is about self defense and self preservation. Even the large Rottweiler lunging at you is most likely doing so because she perceives that you will hurt her. Animals who are fearful have a limited number of options: fight, flight, freeze or fidget. While veterinarians and their staff certainly recognize fight or flight, they may not recognize freeze or fidget. Often, dogs and cats who are perceived as "fine" are anxious or fearful. By identifying these patients and changing their management in the veterinary hospital, future aggressive incidents can be avoided. Veterinary staff can further increase their knowledge of dog body language by attending continuing education, looking to veterinary behavior textbooks, joining professional organizations focused on behavior, or viewing educational DVDs on the subject. It is imperative that each staff member be able to recognize the signs of stress and react appropriately.

Whether restraining dogs or cats the same basic principles apply:

- Make a box around the animal using your body, the table, the wall or other items such as a towel.
- Block the vision of the animal.
- Move animals into different positions slowly.
- Support their weight at all times. Dogs don't fly!
- Ground your body by bending and grounding your elbows, knees or torso.
- Try to eliminate any air between you and the patient.
- Concentrate on the restraint, not other tasks.

### Tools

- Towels
- Elizabethan collars
- Rugs or non skid surface
- Squeezable food

- Great treats
- Slip leads
- Head collars
- Regular leashes
- Cat bag
- Food toys
- Manners Minder

## Dogs

When dealing with an aggressive or fearful dog, the staff should avoid direct eye contact, reaching for the dog, reaching for the owner (including handing something to her), a frontal approach to the dog or close contact. It should be noted that petting may not be viewed as a reward for fearful dog. Instead, it is likely to be regarded as punishment.

If the dog has shown aggression or extreme fear previously, special preparations should be made for future appointments. Aggressive dogs are best scheduled when the lobby is quiet such as in the morning or just after afternoon surgery. Owners should be instructed to bring their dog hungry so that food rewards will be more enticing. In general, two people who are known to the dog should attend the appointment. This will allow one owner to handle the dog while the other fills out paperwork, accepts medications or checks out. Lobby waiting should be avoided. If possible, the dog and owner should wait in the parking lot until the examination room is ready. Dogs who wait in the lobby are continually bombarded by stimuli causing them to become more aroused before they even step foot in the examination room. If there are two owners present, the dog can wait with one owner in the parking lot while the other provides the patient history and presenting complaint. Based on the presenting complaint, the examination room should be stocked with all necessary supplies before the dog enters. It is generally better to be over prepared with aggressive dogs so that the entry/exit into the room by doctors and staff is minimized. Each entry/exit has the potential to agitate the dog further.

Once the examination room is ready, the veterinary technician can escort the client into the room. At this point, the owner should muzzle the dog. Some dogs are better muzzled in the lobby, in the car or at home. If using a basket muzzle, concerns about the patient wearing the muzzle for extended periods of time should be minimized. Since the presenting complaint and history have already been taken, the technician should leave the room and return when the veterinarian is ready for the appointment.

A common mistake made in handling aggressive dogs is the attempt to “be friends.” Aggressive dogs don’t want to “be friends.” They want what you want: great medical care dispensed very quickly so that they can leave your hospital. To this end, interactions involving touching and feeding the dog right off the bat should be avoided. If the dog has bitten in your hospital before it is unlikely that an initial treat is going to buy her off anyway. Veterinarians should ignore the dog completely when they walk in so that they will not in any way be viewed as a threat. Most dogs will be calmer if they enter the room after the veterinarian and their staff is already present as opposed to the veterinarian entering the room last. Once a restraint technique has been chosen for the pet, all tests and examinations should be done quickly. The veterinary staff should as if they will only get one chance to complete all tests before the dog’s behavior becomes unmanageable.

There are many alternatives to traditional restraint which can make the dog less reactive and still keep the staff safe. One alternative is using a towel around the dog’s neck like a neck brace to immobilize the head during an exam. Another method involves using a head collar and a

basket muzzle together. Some dogs are better when “restrained” by a wall and a corner instead of being held by a person. Other dogs can be more easily managed when wrapped in a towel burrito style or when wearing an Elizabethan collar.

During restraint, the animal should receive constant feedback on her behavior. The type of restraint and your reaction to the dog’s behavior will affect her future behavior at the veterinary hospital. In general, if the dog is being quiet and not resisting, the restraint should be lessened. Not so much that the dog can get away from you, but enough that she can feel the difference. If she struggles, tighten your restraint a little. This type of conditioning can be effective in teaching the dog that quiet, calm behavior is rewarding, however if the dog is struggling vigorously, urinating or defecating, this type of conditioning will not be effective because the dog is at a high state of neurochemical arousal. It goes without saying that there is no place for hitting, yelling or harsh physical restraint when working with any animal. It is counterproductive and unethical. Sedation is always around the corner for the dogs who are simply impossible to handle any other way.

If feeding is not contraindicated based on the presenting complaint the dog should be fed throughout the examination. Feeding in this way acts as a distraction to the dog and conditions the dog to associate the veterinary hospital with positive things. Squeeze liver paste or cheese can be very effective in cases where food can be used. Food should be offered from the point just before the dog begins being restrained and should continue until just after the veterinarian has completed all tests. When the veterinarian has completed the examination and all tests, she can again leave the room and let the client remove the muzzle. When the appointment is over, the dog should be escorted out to the parking lot with one owner while the other owner receives the discharge instructions, medication and checks out.

Clients should receive instructions on where to purchase a basket muzzle and how to condition their dog to the muzzle as soon as any sign of aggression is noticed. Clients can be very sensitive to the idea that their dog may have to be muzzled while at the veterinarian’s office. It can be personally offensive to them and should be approached with care and sensitivity. Most owners can be convinced that it is less stressful for their pet and in their dog’s own interests to have a well fitting, comfortable muzzle that they are conditioned to wear. Muzzles can be found at [www.morrco.com](http://www.morrco.com), [www.thedogmuzzle.com](http://www.thedogmuzzle.com), and [www.hunterpet.com](http://www.hunterpet.com). Muzzles can even be found for French Bulldogs so no excuses for improper muzzling! If the dog is not aggressive to the owner, muzzle conditioning can be completed very quickly at the owner’s home. Other helpful control tools are head collars and the Calming Cap® (Thundershirt) which blocks the dog’s vision.

A desensitization and counterconditioning treatment plan should be laid out for all dogs who are aggressive at the veterinarian’s office. These types of plans are intended to condition the dog to behave in a calm way while being examined and restrained.

### **Cats**

Cats can be especially difficult to examine and treat because most don’t have any training and very little socialization. As a result, they not only become suspicious and fearful of the veterinarian, but also car rides and being placed in a carrier.

Examination rooms should be comfortable. The examination table should be covered with a towel before placing the carrier on the table. As soon as the cat is in the exam room, the carrier should be placed on the table and opened from the cat if the cat is not likely to come out on her own. Then, the cat should be covered with a towel which has been previously sprayed with

Feliway® (Ceva). Many cats can be examined in their carrier. If this is not possible, the cat can be lifted from the carrier wrapped in a towel once the top of the carrier has been removed. Cats should not be dumped from the carrier. Anyone who has tried to dump a cat from a carrier, holding it vertically as the cat clings for dear life inside knows that this is the wrong way to start a visit to the veterinarian's office.

The old phrase "go slow" is especially true with cats. This doesn't mean that the technician should be tentative because we all know that can result in a worried and anxious patient. Movements should be slow and deliberate taking time to give the cat feedback on her behavior. While food can be offered during an examination as it would be with a dog, this is generally unsuccessful in cats because their level of fear is too high. The towel is your friend when restraining cats. There are many methods which are described in detail elsewhere which outline the various ways to wrap a cat for restraint. By far, this is the easiest and safest method of handling a cat who is aggressive.

Owners of fearful and aggressive cats should be taught how to condition their pet to the carrier so that they do not continue to link the event of being put in the carrier with the trip to the veterinarian's office.

Working with fearful and aggressive animals can be rewarding when done correctly. The veterinary staff and the patients will be less stressed, client retention will go up and the veterinary staff will be able to provide excellent medical care to all patients regardless of disposition.

### **General guidelines for PVPs**

Familiarize yourself with the medications which can safely be given in each species that you treat including the side effects, duration of effect, metabolism and dosing. If patients have a known travel or veterinary hospital FAS consider a PVP. All medication mentioned here should be given medication 2 hours prior to the veterinary visit. If the owner gives the medication one hour prior to the veterinary visit, the patient will most likely be arriving at the veterinary hospital at the time that the medication is starting to take effect. This will cause an inadequate medical response because the patient most likely has mounted a stress response in the car either when they see the carrier (cats) or when they pull into the parking lot. This may contribute to the phenomenon often seen in veterinary medicine where our patients "fight" the sedation in the hospital only to be sedated at home for the rest of the day. Manage owner's expectations by following up regularly after recommended test doses and trials. Educate them on the number of trials and test doses it may take to find the right medication or mix of medications for their pet. The less stressed the patient is, the more likely the medications are to be effective. In other words, administration of medications alone is not enough to reduce FAS.

After test doses or potentially practice visits have been completed, you may find that additional medications need to be added to achieve the level of sedation required. In general, start with one medication at an effective dose and test dose it at home and on a hospital visit. If the effect is good, but not adequate instead of abandoning that medication, consider adding in another as you might do if you were attempting to alleviate pain in a patient.

There is a possibility when using a medication which alters mood that the patient will become disinhibited. If FAS are causing aggressive responses, diminishing that FAS may cause the aggressive responses to decline. If FAS is suppressing aggression, when you diminish the fear it is possible for the aggressive responses to increase. It is impossible to know the outcome

before you administer the medication. Make sure to inform clients of this possible outcome.

As in many veterinary disciplines, the medication dosages used in behavioral medicine are based on empirical use, extrapolation from human dosages and a research studies. Few pharmacokinetics studies are available in dogs and cats for the medications discussed here.

There is no advantage to discontinuing SSRIs and TCAs prior to veterinary visits which involve sedation. Abrupt discontinuation has the potential to cause rebound side effects. It is acceptable to discontinue the morning of the sedation because the patient is likely to be NPO, but the medications should be restarted as soon as the patient is awake and eating.

<b>General Guidelines for PVPs (supplements and medications)</b>
Consider: patient temperament, clinical signs of FAS, health status, owner ability to medicate, and if further sedation is likely to be necessary.
Assess clinical signs in the patient, correlate (if possible) those signs with the neurotransmitter that may be causing that effect, then make a medication choice.
Dose 2 hours prior to appointment
Responses to psychotropic medications vary widely depending on the individual.
Test doses must be completed at home when the pet is not coming to the hospital for an appointment to assess side effects, duration of effect, onset of action and clinical effect.
“Practice appointments” may be necessary to find the right medications for each individual patient.
In-hospital management will affect the patient’s stress level and response to medications.
Always assess and manage pain.
Any medication which alters mood can cause a worsening of clinical signs or a disinhibition of learned behavior.
Dose supplements within the dosing range for each ingredient, not necessarily what is on the label of the brand name supplement.
Dose medications and supplements at the low end of the range and slowly move up within the dosing range to get desired effect.
Avoid combining SSRIs, TCAs and MAOs with other medications which increase serotonin such as tramadol. In general, avoid combining medications or supplements which increase the same transmitter or cause similar side effects.

## Supplements

Supplements are popular with many clients because they generally have less potential side effects than traditional medications. In recent years, the market for supplements targeted to alleviate FAS has exploded. In this paper, ingredients and supplements are listed in alphabetical order. As with medications, supplements should be used with low stress handling. Many supplements can be used concurrently with medication. Make evidence based decisions based on individual patient needs.

### Alpha-casozepine

Alpha-casozepine is a bovine protein supernatant derived from cows milk. It is lactose free and has it's affect at the GABA<sub>A</sub> receptor. It is dosed at 15-30 mg/kg SID for dogs and cats. This ingredient can be dosed acutely, but generally takes 30 days to maximum effect. The veterinary product with alpha-casozepine is Zylkene™ (Vetquinol).

### L-theanine

L-theanine is derived from green tea extract and acts as a structural analog of glutamate. It also has GABAergic and dopaminergic qualities. It is dosed at 5-10 mg/kg BID for dogs and cats. This ingredient can be dosed acutely, but generally takes 30 days to maximum effect. The veterinary products with L-theanine in the Suntheanine form are Anxitane™ (Virback), Solliquin™ (Nutramax) and Composure Pro™ (VetriScience).

### Magnolia officinalis and Phellodendron amurense

The bark of these trees contains active compounds which have their effect at the GABA<sub>A</sub> receptor. When given together, then act synergistically to reduce FAS. In humans, this combination has been shown to reduce cortisol over time implying that the chronic stress response has been reduced. There is one study which reports a decrease in FAS in dogs who were thunderstorm phobic. The dosing is approximately 250 mg/20.7 kg.

## Medications

### Benzodiazepines

Commonly used benzodiazepines include alprazolam, diazepam, and clonazepam. There are other benzodiazepines which should be considered as well which are not outlined here.

Pro	Con	Tips
Minimal to no adverse physiologic effects	Highly variable responses in each individual and within the medication class in the same individual.	If the patient is fearful and shows no aggression, can use as a single agent. If aggression is noted, should be combined with other agents.
Relieves FAS	Paradoxical excitement and disinhibition potential.	Increase GABA which causes inhibitory effects in CNS
Slight sedation	No analgesia	Safe to combine with many other

		sedation/anesthesia protocols
Increased appetite	Cannot use diazepam in cats	If the first benzodiazepine isn't effective, but there are no side effects, try a different benzodiazepine.
	Don't provide potent sedation	Possible side effects include ataxia, hyperphagia, sedation, paradoxical excitation and behavioral disinhibition.

## RELIEVING STRESS IN HOSPITALIZED PATIENTS

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All of us have been at our workstations writing up reports while a dog in the background barked relentlessly. Maybe you have felt annoyed or put on your headphones to drown out the sound. Maybe you have heard the cat yowling and growling from the wet table as a technician tries to take blood for a glucose curve. Those types of experiences are daily occurrences in most veterinary hospitals. Now we understand that those patients are stressed and that the stress they are experiencing can have deleterious effects on their health, healing and well being.

There are lots of ways that we can change the environment and our behavior in order to alleviate patient stress. Species specific pheromone diffusers, calming classical music playing and calming scents like lavender can help to alleviate stress in cats and dogs. Low stress handling can be a very effective way to mediate stress. Tools such as non invasive blood pressure (e.g., Sun Tech) can allow procedures to be completed with as little stress as possible.

Alleviating stress medically in hospitalized patients who may be critical can be stressful for the veterinarian. At what point should the veterinarian prescribe? What medication is right for which clinical sign? How should the veterinarian respond to nurses who have concerns about patient stress?

Before prescribing for any patient, the veterinarian should be familiar with the potential interactions, side effects and contraindications of available medications. The Plumbs and VIN formularies are good options for finding this information.

Options for relief of fear, anxiety and stress in hospitalized patients include benzodiazepines (e.g., diazepam, alprazolam), trazodone, gabapentin and acepromazine. When oral anxiolytic medications are administered to a patient who has already mounted a physiologic stress response the latency to effect will be longer and the clinical effect will be reduced. Avoid administering very high doses to accommodate for the stress response. The patient's body has to metabolize that additional medication even though you may not be seeing the clinical effects of the medication. Often, patients will show signs of sedation 3-4 hours later. The best way to avoid this is to prescribe low doses of anxiolytics orally at the first signs of fear, anxiety or stress. Ideally, anxiety relief orders would be written by the attending doctor when the patient is admitted so that nurses know what medication to administer.

Benzodiazepines are generally effective at reducing anxiety and fear and there is a low likelihood of interactions with the medications commonly used in veterinary medicine. In addition, they can be used in geriatric patients safely. Paradoxical excitement is a fairly common side effect in dogs given this class of medications. There are certain patients such as post op orthopedic patients for whom this side effect would be dangerous. Consider a benzodiazepine in those animals who are fearful or anxious, but for whom the potential side effect of paradoxical excitement would not be dangerous. In addition, benzodiazepines are reliable appetite stimulants.

Gabapentin is well tolerated in dogs and cats. It relieves anxiety as well as certain types of pain. It is safe in geriatric animals and has a low incidence of interactions. It should be used with caution in animals whose kidney function is compromised due to decreased clearance.

Trazodone (Desyrl®) is a serotonin antagonist, reuptake inhibitor. It causes a calming or sedative effect. It should be used with caution in patients who have compromised liver function.

Acepromazine is a sedative which can be used at low doses in healthy patients who are hospitalized in order to help them sleep.

Sileo is a transmucosal form of dexmedetomidine. It has a calming effect within 20-30 minutes. The effect lasts for approximately 2 hours. If dosed per the label directions, the patient receives 125 mcg/m<sup>2</sup> of dexmedetomidine. The bioavailability of the dexmedetomidine across the mucosa is 28% making the effect of the 125 mcg/m<sup>2</sup> consistent with a calming effect and not a sedative effect.

## EARLY INTERVENTIONS. RECOGNITION AND TREATMENT OF PSYCHIATRIC DISORDERS IN PUPPIES AND KITTENS.

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Recognition of the early indicators of emotional disorders in puppies and kittens allows the veterinary healthcare team to intervene before the pet's behavior degrades the human animal bond causing the client to consider relinquishment or euthanasia. In addition, what happens at puppy and kitten appointments shapes the puppy's future behavior at the veterinarian and the client's likelihood of coming back to your clinic.

### Early signs of emotional disorders

#### Normal behavior Puppies

Understanding of body language is essential to being able to assess the puppy's level of fear or anxiety. The ideal puppy is friendly and more willing to approach than run away from new things. He is curious. When offered food, if otherwise healthy, he should at minimum sniff and ideally eat. His body language is loose, tail is not tucked under the body or tightly curled unless appropriate for the breed. His pupils are of normal size for the ambient light. He recovers quickly from stress.

#### Kittens

Like puppies, kittens should have loose, relaxed body language and be curious. When playing, they should bounce, swat, stalk and bite without deeply puncturing the skin. Aside from body language exhibited in interactions with the owner and other animals, kittens should display normal elimination behavior. While there is variation in how a particular kitten may eliminate including leaving feces or urine uncovered, most kittens urinate and defecate the same pattern.

#### Developmental Periods Puppies

##### Socialization

Socialization is a process in which the puppy learns to relate to other animals and people as well as the environment. The type of socialization (positive or negative) will shape the puppies behavior for the rest of its life. Shyness in puppies can be hereditary and some pups may never be able to be properly socialized. The in-utero environment, maternal nutrition and stress affect the reactivity and personality of the puppy. There are certain times when socialization has the greatest chance of shaping a puppy's future behavior. They are called sensitive periods.

##### Sensitive period

- 3-12 (16-20) weeks of age
- 3-8 weeks of age, puppies learn best how to socialize with other puppies.
- 5-12 weeks of age, puppies learn best how to socialize with people.
- 10-12 weeks and 16-20 weeks, puppies learn best how to explore new environments.

- If not exposed to new stimuli, people and other puppies during these periods, the puppies are more likely to be aggressive, defensive and fearful when exposed to those situations in the future.

### **Fear stages**

- 8-12 weeks of age, 4-6 months of age
- Fear postures begin to emerge at about 8 weeks of age.
- At about 12 weeks, sociability decreases. At this time, the willingness of an unsocialized puppy to interact with new people and novel objects decreases.

### **Puppy Classes**

Puppies should be enrolled in puppy socialization classes after the first vaccination and deworming. In these classes, puppies will learn how to accept other puppies, strangers and different situations. A study done by Duxbury, et al, showed that dogs that attended puppy classes have increased responsiveness to commands and increase retention in the home later in life.

Veterinarians may be concerned about illness when sending puppies to socialization classes prior to 16 weeks when they will receive their final set of vaccinations. A recent study done by Stepita, et al, all showed that puppies were not likely to contract Parvovirus when they attended puppy classes. Dog trainers who teach socialization classes in which puppies under 16 weeks are enrolled, should require one vaccination and 1 deworming at least 7 days prior to class. In addition, the medical histories should be collected every 2 weeks during class to ensure that puppies are being kept up to date with vaccinations and dewormings. Facilities should be indoors and cleaned with the 1:30 bleach solution. Ill puppies should not be admitted to class.

### **Kittens**

The sensitive period of socialization for cats is 2-7 weeks of age. Genetics are very important in determining a cat's final behavior. Some cats are "resistant" to socialization due to a genetic predisposition to fearful behavior.

Cats which are hand raised typically are more aggressive, energetic and have uninhibited bites. They frequently learn to play inappropriately resulting in aggressive play as they get older. They have a more difficult time forming attachments with other cats. When a kitten is rescued at a young age, it is essential that the kitten gets time with the queen and the litter so that she can learn appropriate feline behavior. When this is not allowed to happen, kittens are more likely to be reactive to new stimuli, participate in random, out of context activity, act inappropriately with other cats, play inappropriately with people and be slow to learn certain new tasks. They also are less likely to explore new environments. Orphan kittens should be cross fostered on another queen, if possible and handled, gently, three times each day for 5 minutes at a time in order to socialize them.

Environmental enrichment is an extremely important, yet overlooked aspect of managing behavior problems in cats. Cats who live in an underenriched environment are more likely to exhibit behavior problems. Owners should be aware of the importance of enrichment at the first kitten appointment.

### **Predispositions**

The following factors can predispose a puppy or kitten to the development emotional disorders later in life.

### Puppies

- Purchase from a puppy store
- Removal from the litter prior to six weeks
- Certain breeds are predisposed to development of separation related disorders such as Weimaraners
- Lack of exposure and socialization
- Hereditary influence
- Traumatic event

### Kittens

- Oriental breeds are predisposed to wool sucking and pica
- Lack of exposure and socialization
- Hereditary influences

### Assessment Puppies and Kittens

Unfortunately at this time there isn't a validated test which has been shown to accurately assess a puppy's temperament. There is evidence that fearful puppies will be fearful adults, even typical interventions. In kittens we have even less information. Because of this, when evaluating puppies and kittens consider the interaction as one snapshot in time. Combine your assessment with the owner's report of the pet's behavior at other locations and at home. The goal should be to find outliers and animals with sensitivities, slow recoveries, heightened, fear, lack of interaction with the environment and repeatable patterns. Be cautious when making predictions about the pet's behavior. Instead, try to talk to the client about predispositions and interventions.

### How can I go about Initiating earlier interventions with my patients who have behavioral issues?

#### Observe and Record Behavior

Use the tips above to assess puppies and kittens. Make sure to record your observations in the medical record so that you can show the client and remind yourself of the pet's behavior at future appointments. This allows you to track the progression of the pet as well. To ensure an accurate history try to ask clear and specific questions. For puppies ask about growling, biting, snapping, unwillingness to interact with people, animals or things, and urination or defecation in the house. Ask kitten owners if there has been any growling, biting, swatting, unwillingness to interact with people, animals or things, urination or defecation outside of the litterbox or hissing.

#### Know what interventions are available

For kittens, there are few hands on opportunities for intervention. For puppies, there are socialization classes, obedience classes, puppy classes, referral to a qualified dog trainer, work with a technician or nurse at your practice, pharmaceuticals, environmental changes and diet.

Pharmaceuticals shouldn't be considered a last resort. Early intervention with a supplement or medication which changes brain chemistry can contribute to a positive outcome in many cases. Supplements such as Composure Pro, Solliquin and Zylkene can be used in puppies and kittens safely. The CALM diet is also an option for puppies and kittens who are anxious or fearful. Outliers or those that are not responding to appropriate treatments should be considered for medical management.

## Resources

1. Florida Veterinary Behavior Service
  - a. [www.flvetbehavior.com](http://www.flvetbehavior.com)
  - b. Puppy and kitten articles, body language, information on how to find a good trainer and links to great websites.
2. American Veterinary Society of Animal Behavior
  - a. [www.avsonline.org](http://www.avsonline.org)
3. From Fearful to Fear Free, Marty Becker DVM, Lisa Radosta DVM, DACVB. Wailani Sung, DVM, DACVB, Mikkel Becker KPA CTC, CPDT-KA, CTC, CBCC-KA, CDBC
4. Puppy Start Right by Dr. Ken Martin and Debbie Martin
5. The Trainable Cat by John Bradshaw and Sarah Ellis
6. Catalyst Council
  - a. [www.catalystcouncil.org](http://www.catalystcouncil.org)
7. Fear Free Happy Homes
  - a. [www.fearfreehappyhomes.com](http://www.fearfreehappyhomes.com)

## EARLY INTERVENTIONS: CASES

Lisa Radosta DVM, DACVB  
 Florida Veterinary Behavior Service  
 www.flvetbehavior.com  
 Possessive Aggression

Possessive aggression occurs when a dog exhibits aggression over valuable items. A valuable item is defined by the dog and can be anything including, but not limited to a person, food, a food bowl, a space or a toy. In the Case One, we will examine the diagnosis and treatment of possessive aggression in a young dog.

### Case 1

- Bailey
  - 10 kg, 4 month old, female, Golden Retriever
  - Presenting Complaints
    - Aggression toward family members when food or items are nearby or taken from her.
    - Frequency-6 times in 8 weeks.
  - General History
    - Acquired at 8 weeks from a show breeder
    - Lives in a family with 4 children and 2 adults
    - Leash walked multiple times a day
    - Fed lams puppy food
    - One adult Labrador Retriever in the house
  - Medical history
    - Treated for 2 UTIs since adoption at the primary care veterinarian's office
    - Recent urinalysis-wnl with resolution of clinical signs
    - Otherwise healthy on physical examination at the primary care veterinarian's office.
  - Behavioral History
    - Began at 8 weeks, when adopted.
    - Plays with the children without aggression as long as there is no food nearby
    - Has bitten the owners over a hot dog bun, dirty dishes in the dishwasher and other pieces of food.
  - Do you need more information? What questions would you ask the owners?
  - Behavioral examination
    - Friendly and interactive. No signs of aggression or fear in the examination room.
    - Physical examination-wnl
  - Diagnosis
    - Possessive Aggression
  - Other differentials to consider:
    - Conflict related aggression
    - Fear related aggression
  - Recommendations
    - Safety
      - Owners were informed of liability and risk of ownership.
      - Keep the dog out of the kitchen when there is food out or they are cleaning up after meals.

- Move the dog's crate to another room in the house outside of the kitchen.
- Make a trade for stolen items until she is trained with basic control behaviors.
- Do not reach for her when she is aggressive.
- Move her with a food lure until she is trained with basic control behaviors.
- Don't push or pull her.
- Behavior Modification
  - Teach leave it.
  - Do not use positive punishment
  - Condition her to approaches at the food bowl
  - Enroll in positive reinforcement obedience classes
- Follow up
  - 1 month
    - No aggression over food
    - Owners have moved the crate to another room instead of the kitchen
    - Taught her to leave it
    - She moved away from an apple and a hot dog bun on two separate occasions when the owner asked her to "leave it."
  - 6 months
    - Rarely showed aggression toward the owners
    - She was still being fed outside of the kitchen
    - Would move away from items and drop items when told to leave it.

### Fear induced aggression

Fear induced aggression can surface at any age. There are many specific circumstances which can result in an animal showing fear related aggression. Generally, most circumstances fall into one of 4 categories: traumatic incident, hereditary factors, lack of socialization and learning. In Case Two, we will examine the case of a fearfully aggressive puppy who had a hereditary predisposition toward fearful behavior, wasn't well socialized and endured a traumatic incident.

### Case 2

- Dezi
  - 1.45 kg, 10 week old, male, Havanese
  - Presenting complaints
    - Growling and biting when he is being held
  - General History
    - Acquired from a breeder at 8 weeks
    - Lives in a family with two retired adults, no children
    - Did not get to interact with the sire or the dam.
    - Fed lams puppy food
  - Medical History
    - Unremarkable, healthy puppy
  - Behavioral History
    - Behavior began at 8 weeks, when adopted.
    - First incident was when the puppy was playing in the back yard with the owner and he kept running under the bushes. The owner continually told him "no" and pulled him out. When he ran under the bushes the 3<sup>rd</sup> time, the

- owner grabbed him by the scruff of his neck, pulled him out and yelled “NO” in his face. Then, she carried him inside. As she was carrying him, he began to bite and growl. The owner held his muzzle and said “NO” to which he responded with more growling. She put him down at that point.
- When being held by familiar people, he will suddenly start to growl and bite. He whips his head around and tries to bite the owner’s hands.
  - The owner has tried holding him tightly until he calms down which takes 1-2 minutes typically.
  - Frequency-5-6 times a week.
  - No training or socialization
- Do you need more information? What questions do you want to ask the owner?
  - Behavioral examination
    - Quiet in the exam room.
    - Lying under the owner’s chair.
    - Did not explore the room.
    - He was very fearful when interacting with the clinician.
    - He would approach for treats, but retreat when he got the treat.
    - The owner was able to handle him and restrain for an exam.
    - He was willing to touch the hand of an unfamiliar person.
    - Physical examination was unremarkable.
  - Diagnosis
    - Fear induced aggression
  - Other differential diagnoses
    - Learned aggression
    - Conflict related aggression
  - Recommendations
    - The owner was educated as to how to read dog body language and the role of dominance in aggression in dogs.
    - Safety
      - Do not physically punish the dog.
      - Don’t push or pull him.
      - Do not let new people pet him.
    - Behavior modification
      - SIT program
      - Enroll him in a positive reinforcement dog training class.
      - Start socialization.
      - Desensitize and Countercondition to
        - Handling
        - Restraint
        - Lifting
      - Use only positive reinforcement training
      - Don’t pick Dezi up unless absolutely necessary.
  - Follow-up
    - One month
      - Started socialization and puppy playtime classes
        - Puppy is very fearful in classes
        - Still growling a lot
    - Two months
      - Starting to be aggressive at the veterinarian’s office when temperature taken

- Handling is going well. Owner is still using food for handling. Can handle him and brush him as long as she has treats.
- Still actively socializing him.
- 5 months
  - Still attending puppy playtime
  - Starting obedience classes
  - Significantly less growling when handled
  - More confident when meeting people
- 7 months
  - Very aggressive at the groomer's office. Groomer refused to groom him again.
  - Owner tried to groom dog at home and he began biting her. She held his snout and held him down while yelling at him.
  - Dog stopped growling, but is very anxious since that time. He is showing a lot of stress signals.
- 8 months
  - recheck, 7.27 kg
  - Start on sertraline, 25 mg tablets, ½ tablet by mouth once daily
  - Plan for grooming and plan for veterinarian.
- 12 months
  - Aggression continues to decrease.
  - Owner is using hand touching and aggression toward strangers is decreasing
  - Owner is able to handle dog more easily. Still using food.
  - Last examination at the veterinarian's office went well.
- 3 years
  - Friendly with people
  - Active in agility and obedience
  - Examined at the veterinarian's office without incident
  - When sertraline was discontinued, dog's behavior returned, but at a much reduced intensity. Owner found that he was less able to concentrate and more fearful.
  - Sertraline was started again at a lower dosage and the dog has been maintained on this dosage for 3 years.

### Case 3

- Summer
  - 16 week old kitten
  - Presenting complaints
    - Growling and biting when he is being held
    - Intense play and biting including "drive bys."
    - Hissing at other cats in the household.
  - General History
    - Found as a 2 week old kitten and bottle fed.
    - Fed kitten food, lams
  - Medical History
    - Unremarkable, healthy kitten
  - Behavioral History
    - Behavior began at 6 weeks.

- When owner plays with cat, the cat becomes highly aroused and bites the owner. Rarely breaks skin. Pupils dilated and tail puffed up.
  - When the kitten becomes aroused, sometimes she runs by the owner's legs and bites lightly.
- Do you need more information? What questions do you want to ask the owner?
- Behavioral examination
  - Constantly moving.
  - Never stopped playing, investigating and moving around the room.
  - When offered a finger to bunt, she moved her head away.
  - When tried to pet, bit lightly.
  - Played constantly with dilated pupils. Pupils constricted when she was not playing.
- Diagnosis
  - Play related aggression
  - Impulsive aggression
- Recommendations
  - Safety
    - Do not let cat interact with any children or elderly.
    - Keep away from other cats in the household.
    - Separate from other cats until she can interact nicely with them.
    - Stop interactions if she is inappropriate.
  - Behavior modification
    - Teach go to the mat.
    - Teach a control behavior.
    - Desensitize and Countercondition to
      - Other cats
    - Enrich the environment.
    - Leave on back porch with the dogs whenever possible.
- Follow-up
  - One month
    - Cat is not enjoying living on the porch. Doesn't want to be out without the owner. Enjoys the dogs. Is learning well to go to a mat. More responsive to one cat at a time. More responsive to owner in the evening.
  - Two months
    - Cat can interact with some of the cats in the house for short time periods.
    - Owner can pet the cat and play with her over short periods of time without bites.
    - Drive bys have decreased significantly.

## AGING GRACEFULLY. COGNITIVE DYSFUNCTION UPDATE

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Companion animals are living longer than ever before in part due to high quality veterinary care and nutrition. Roughly 50% of our patients are at least 6 years of age and 14.7% are older than 11 years. Unfortunately, unique behavior problems can accompany increasing age. The incidence of cognitive dysfunction varies depending on the study cited. Azkona, Garcia-Belenguer and Chacon (2009) found that 22.5% of dogs over 9 showed signs of cognitive dysfunction. Neilson, Hart, Cliff and Ruehl (2001) found that 28% of dogs between 11 and 12 years of age reported at least one sign of cognitive dysfunction and 10% had 2 or more signs. In addition, in 15-16 year old dogs, 68% of dogs had at least one sign and 36% had two or more signs of cognitive dysfunction.

Unfortunately, clients rarely report geriatric behavior changes to their veterinarian unless prompted to do so. In one study, while 75% of owners noticed at least one of the signs of cognitive dysfunction in their pet, only 12% had spoken with their veterinarian about it. Another study found that only 7% of owners spontaneously reported geriatric behavior changes to their veterinarian. Yet another study reported that 14% of dogs aged 8 and older are affected with CDS, but only 1.9% are diagnosed. Many may be euthanized prior to diagnosis and treatment. Often, owners attribute their pet's behavioral changes to old age and assume that nothing can be done to alter them.

The communication barrier can be overcome by using a short questionnaire in the waiting room which can be reviewed by the veterinarian before the appointment begins. Five simple questions focusing on housetraining, loss of obedience, increased anxiety, fears and phobias and changes in the level of aggression should illuminate behavior problems sufficiently prior to the appointment. This way, the veterinarian will be able to assess if it is a topic that should be discussed with the client in more detail. In addition, it will bring behavior changes to the forefront of the client's mind so that he or she will be more likely to share concerns about their pet's behavior with the veterinarian. Purina has published an assessment form which can be downloaded at [www.flvetbehavior.com](http://www.flvetbehavior.com) on the Veterinarian's page. Practices can also request a tear pad with assessment forms.

Once the veterinarian and client have discussed the pet's behavior changes, consideration of contributing medical diseases is essential. The signs of cognitive dysfunction are general and can be caused by medical diseases such as osteoarthritis, metabolic diseases, seizure disorders, neoplasia and hearing or sight impairment. The primary signs of cognitive dysfunction in dogs are disorientation, interaction changes, sleep/activity changes, breakdown in housetraining and changes in anxiety, activity and aggression (DISHA) although owners also report apathy, indifference, and less playing. In cats, owners report housesoiling, excessive vocalization, apathy, less play and aggressiveness as the most common signs of CDS.

It is important to isolate specific medical and behavioral diagnoses for each dog so that each problem can be treated appropriately. For example, while cognitive dysfunction may be present, the dog may also have developed separation anxiety. Treatment should focus on treating both problems in order to get the best outcome.

Canine and feline cognitive dysfunction can be diagnosed if there is an occurrence of one or more geriatric onset behavior problems which are not accounted for by medical conditions. It is generally a disease of dogs over 10 years old and cats over 12 years old, but in one study, dogs as young as 8 years old reportedly showed signs of cognitive dysfunction. It is a progressive disease with older dogs and cats showing more signs than younger animals.

The pathology of CDS is similar to that of Alzheimer's disease in people including diffuse  $\beta$  amyloid plaques within the cerebrum and hippocampus and their blood vessels. It has been documented in

humans, dogs and cats that with age, there is cerebral atrophy, ventricular dilatation, lipofuscin accumulation, decrease in the number of neurons and an increase in the number of glial cells. Meningeal fibrosis and accumulations of ubiquitin-protein conjugates in geriatric dogs without the tangles of human Alzheimer's disease has been documented. In some species, there is a fall in serotonin levels, a decrease in the activity of the cholinergic neurotransmitter system, an increase in the activity of the adrenergic system and higher than normal levels of monoamine oxidase. There is also evidence that free radical production increases and clearance decreases.

Treatment for cognitive dysfunction is similar to other behavioral diagnoses and includes behavior modification, management changes, safety recommendations and medication when needed. Anipryl® (selegiline, Pfizer), a selective MAO-B inhibitor (when administered as directed), is FDA approved for use in canine cognitive dysfunction. It can restore the sleep/wake cycle and help to slow the progression of cognitive dysfunction. It inhibits the reuptake of dopamine, norepinephrine and serotonin, increases free radical elimination and alters dopamine function. Because of its broad scope of action, it interacts with multiple drugs which are commonly used in veterinary medicine. Medications which should not be combined with selegiline include medications which affect the reuptake of serotonin and dopamine on any level and other MAO inhibitors. Veterinarians should read the drug monograph for selegiline and any other medications prescribed concurrently before instituting polytherapy.

Selective serotonin reuptake inhibitors (SSRIs) can be used to treat the anxiety and aggression which may accompany cognitive dysfunction. In general, SSRIs have fewer side effects than selegiline, but still should be used with caution in animals with hepatic or renal impairment.

Nutritional therapy may also be indicated for dogs with signs of cognitive dysfunction. Canine b/d™ (Hills) diet has an antioxidant package which has been shown to improve age-related behavioral changes and learning ability in older dogs by limiting cellular damage in the brain. This diet can be helpful in prolonging normal cognitive function in older dogs. Practitioners may consider prescribing this diet instead of the "senior" diet that they typically prescribe for dogs 10 years or older. Owners could see a difference in as little as 30 days.

Neurocare™ (Purina) is approved for use as an adjunctive therapy for idiopathic epilepsy, however it has also been shown to improve the clinical signs of CDS in dogs in a double blinded placebo controlled study over three months.

Nutraceuticals can be helpful in the treatment of cognitive dysfunction. They can be stopped and started without weaning schedules and they generally have low incidence of side effects. They can be used as an adjunct to treatment with medications and behavior modification or they can be used alone. SAM-e has been shown in a double-blinded, placebo controlled study to be effective in alleviating the signs of cognitive dysfunction. SAM-e can be used in cats as well. Senilife® (Ceva) contains phosphatidylserine, pyridoxine, ginkgo biloba, resveratrol and Vitamin E. In general, the claims for this product include: increased blood flow, neuronal protection, brain metabolism and glucose consumption. In addition, it modulates the dopaminergic and cholinergic system. It has been shown to be effective in the treatment of CDS and can work in dogs and cats within 7 days. Neutricks™ (Quincy Animal Health), contains apoaequorin, a natural calcium-binding protein. The claims include that the product replaces the loss of calcium binding proteins which are lost in the aging process. In a study of elderly Beagles, apoaequorin was found to improve cognitive function in learning tests.

L-arginine is a semi-essential amino acid which in the brain is oxidized to nitric oxide gas. The gas can diffuse rapidly from cell to cell and appears to be one of the chemical messengers involved in learning and memory. Essential fatty acids have been shown to reduce brain inflammation and have positive effects on cognition, mood and aggression. Medium chain triglycerides (MCT) are converted in the brain to ketones which can be used as an alternative source of energy for the brain. In dogs, certain MCTs have been shown to improve cognition.

The general behavior modification treatment plan for dogs with cognitive dysfunction includes environmental enrichment, increased mental stimulation, structured interactions with the owners, institution of a predictable routine and retraining certain behaviors. In addition, any other specific behavioral diagnoses should be treated. In order to make interactions more predictable, owners should use the dog's verbal cues when communicating with him and alert him (with a word, sound or light signal) when they are going to touch him. Older dogs may be startled easily due to visual, hearing or orthopedic impairment causing them to act out aggressively or become fearful of everyday events. If the older dog is starting to act out aggressively, precautions should be taken to separate him from children or others who may instigate aggression while treatment is initiated.

Loss of housetraining is often of great concern to owners. Typically, the owners of dogs with cognitive dysfunction report that their dog goes outside for a walk and does not eliminate. After the walk, the dog immediately eliminates inside the home. In order to treat this problem, owners can re-housetrain the dog or train him to eliminate in a litter box or on paper. Confinement may be necessary when the older dog cannot be watched so that accidents do not occur. Ideally, older dogs would be confined in a small, tiled room with a bed and water. When this is not possible a crate can be used. For many older dogs, confinement at this stage of life can be very stressful.

If the older dog needs to be confined due to housoiling or aggression, but does not have recent experience with or was never confined to a crate, it may be a challenging and stressful to adequately train him to accept the crate. Confining an older dog who is anxious or impaired in a crate for the first time and leaving the house for long periods of time may precipitate the development of separation anxiety and confinement anxiety. In addition, he may cause injury to himself if he makes efforts to escape. The owner should expose the dog to the crate slowly using positive reinforcement. Once the dog can go into the crate on cue, the owner can begin to close the door. When the dog is successful at that step, she can begin to leave the dog alone for short periods of time. The owner should monitor the dog closely for any signs of anxiety (e.g., hypersalivation, panting, urination, defecation) while in the crate. If the dog is anxious, the owner has progressed too fast and should back up in the training.

An older dog may become the victim of aggression from a younger dog in the household, even if they got along previously. While this may be due to the younger dog's inclination to rise in rank, it is more likely because the older dog is not offering appropriate social signals due to impairment. Often, the younger dog is confused by the lack of appropriate responses by the older dog. In situations like these, it is usually not helpful to "reinforce" or "elevate" one dog over the other because the problem does not lie with rank within the pack. Instead, inappropriate signaling and posturing between the two dogs or increased anxiety on the part of one or both dogs causes increased aggression. Treatment focuses on keeping the older dog safe, controlling the younger dog with basic obedience exercises, structure exercises for both dogs, reintroduction to trigger situations after control has been established and anti-anxiety medications as needed.

Cats can show signs of cognitive dysfunction as well although it may not be as obvious as in dogs because cats may seclude themselves from the family which can be attributed to an independent nature instead of cognitive dysfunction. Cats most commonly are presented for housoiling and/or nighttime vocalization. As with dogs, cats who are presented for these problems should be evaluated for medical conditions such as renal failure and hyperthyroidism.

Many of the supplements and medications listed above can be used in cats. In addition, benzodiazepines can be used orally on a nightly basis to help alleviate anxiety. Owners should also be aware of stimuli which can cause nighttime vocalizations such as stray cats or animals outside. Blocking access to windows can, by itself help resolve this behavior. Owners should avoid reinforcing this behavior by feeding the cat or letting her outside. Instead, they can confine the cat to a comfortable room at bedtime so that she can't come to the owner's room at night and vocalize. Automatic feeders

can be used overnight to keep the cat occupied as well. Finally, owners should invest in toys and other environmental enrichment tools in order to keep the cat occupied during the day and overnight.

Geriatric cats who are no longer eliminating in the litterbox should be treated as would any age cat with this problem. They should be evaluated medically as above. Then, the environment should be examined for deficits and management changes should be recommended. The treatment of feline inappropriate elimination is described in detail elsewhere and won't be repeated here. The height, size and location of the litterbox is especially important for geriatric cats because there may be limitations in mobility and increases in urgency.

As dogs and cats live longer, the likelihood of age related behavior problems increases. With treatment of underlying medical problems and behavioral problems, the quality of life of our geriatric canine friends can be improved tremendously.

## How Identifying and Treating Juvenile Malocclusions Can Prevent a Lifetime of Pain

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### Overview

Puppies and kittens can have oral pathology that can lead to serious problems later in life. Many of these conditions can be treated successfully, thus preventing a lifetime of pain, discomfort and misery. During this session, we will address many common (and a few uncommon) oral conditions. Once learned and appreciated, the practitioner is armed with the beneficial knowledge to not only identify pathology at an early age, but also treat a condition. Fortunately, doctors frequently examine puppies and kittens at least 2-3 times during the first 2-6 months, thus allowing for more than one opportunity to closely examine the oral cavity.

### Objectives

- Appreciate normal oral anatomy of the dog and cat
- Understand eruption dates and when to intervene with treatment when teeth are not erupted
- Understand non-occlusal pathology (macroglossia, cleft palates)
- Understand the 4 Classes of Malocclusion
- Know the treatment plan for each Class of malocclusions
- Understand how crowding can affect eruption pathways
- Addressing fractured primary teeth and adult teeth

### Key Clinical Diagnostic Points

Normal oral anatomy of the puppy and kitten will be reviewed.

At 8 weeks of age, most primary teeth should be erupted and all visible by 12 weeks of age. Remember that there are no primary 1<sup>st</sup> premolars in the dog as well as any primary molars. If there is a soft tissue impaction visible over a non-erupted tooth, oral surgical intervention is needed (operculectomy). Dental radiography must precede this oral surgery to make sure that there adult teeth present.

There are a few problems that are recognized in the first weeks of life. The most lethal is macroglossia, which is an abnormally small tongue. The prognosis is hopeless and compassionate euthanasia is warranted. Cleft palates may be primary (rostral to the incisive foramen and including the lips) or secondary (involves the hard palate caudal to the incisive foramen). Primary palatal clefts can be presented with a unilateral or bilateral presentation. Secondary clefts are almost always on the midline and are usually involving the hard and soft palate. Ideally, it is best to wait to repair the

secondary cleft until the patient is healthy enough for anesthesia (12 weeks of age). Soft palate clefts can also occur and these can be unilateral, bilateral or at the midline.

The 4 classes of malocclusions

Class I: Normal occlusion with one or more teeth out of alignment or rotated. These include rostral and caudal crossbites and base narrow canines. A *rostral crossbite* occurs when one or more of the mandibular incisors are anterior to the maxillary incisors while the rest of the teeth occlude normally. Base-narrow or lingually displaced mandibular canines can traumatize the hard palate and/or the maxillary canine teeth. Left untreated, this condition is painful and can lead to oronasal fistula. (This condition can also occur with a Class II malocclusion)

Treatment of Class I: Rostral crossbites can be treated via orthodontic movement or extraction, depending on the severity of the condition. Base narrow mandibular canines can be treated with the following: Removable orthodontic appliance (kong therapy) may be used in selected cases; incline plane fabrication; crown extension with composites; crown reduction and vital pulp therapy; gingivectomy/plasty; extraction.

Class II: Known as mandibular brachynathism, overshoot or retrusive mandible. The lower premolars and molars are positioned caudal to their normal relationship. This condition can lead to significant oral pain due to trauma to the hard palate.

Treatment of Class II: Extraction or crown reduction and dentin bonding of the mandibular incisors if traumatizing the palate; crown reduction and vital pulpotomy therapy of the mandibular canines; extraction.

Class III: Known as mandibular prognathism, undershot or protrusive mandible. The lower premolars and molars are positioned rostral (mesial) to their normal relationship. Significant attrition (tooth on tooth contact) can occur with this form of malocclusion. A level bite is a form of this malocclusion.

Treatment of Class III: Odonotoplasty and dentin bonding (or extraction) of the right and left maxillary 3<sup>rd</sup> incisor of causing trauma to the mandibular canine teeth. Removal of any tooth causing attrition; crown reduction and dentin bonding (or extraction) of the maxillary incisors if traumatizing the mandibular gingival mucosa or incisors.

Class IV: Known as wry bite, there is a difference in length in the two maxilla and mandible due to asymmetry.

Crowding and rotated teeth can potentially prevent full eruption of an adult tooth. This condition occurs with many brachycephalic breeds. Crowded mandibular lateral incisors can prevent a normal eruption pathway for an important tooth such as a mandibular canine tooth, leading to a partially erupted tooth. These partially erupted teeth can have pathological suprabony pockets. Adult teeth need adequate bone to survive. If crowding or rotation occurs, a tooth then has limited alveolar bone space to

'live'. Compromised bone space with secondary inflammation can lead to mobility. Removal of rotated or crowded teeth can prevent this from occurring.

Fractured primary teeth that have pulp exposure should be extracted immediately. If an adult tooth has been fractured in a young pet and the fracture is within a 48 hr period, a vital pulpotomy may be performed. If the tooth has been fractured longer than 48 hrs, this tooth should be extracted.

### **Summary**

Aggressive action by the DVM can help prevent a lifetime of oral pain and discomfort for their patient. Early identification of pathology is necessary to achieve this goal of a pain-free mouth.

### **References/Suggested Reading**

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**WHILE LEAVING THIS ALONE IS NOT AN OPTION: TREATING THE DISCOLORED TOOTH****BARDEN GREENFIELD, DVM, DAVDC***Memphis Veterinary Specialists (MVS) Memphis, TN**Arkansas Veterinary Emergency and Specialty Hospital (ARVES) Little Rock, AR*

A study by Fraser Hale in the Journal of Veterinary Dentistry (Hale FA. Localized intrinsic staining of teeth due to pulpitis and pulp necrosis in dogs. *J Vet Dent 18[1]:14-20 2001 Mar*) showed that over 92% of intrinsically stained teeth were non-vital (partial or total pulp necrosis). Amazingly, only 42.9% of those cases had radiographic evidence of endodontic disease.

Teeth can become non-vital via a variety of ways. The first is obviously via direct pulp exposure as in a complicated crown fracture which if not treated within 48 hours is considered non-vital. Other ways teeth can become non-vital is via trauma as in a subluxation, luxation or even avulsion of a tooth. This interruption of the blood supply can cause *premature tooth maturation or tooth death*. Finally, blood borne infection called *anachoresis* can affect a tooth at the apex and kill a tooth.

**Why does a tooth discolor and what does this mean?**

When the blood supply to a tooth is interrupted, either temporarily or permanently, hemoglobin in the pulp cavity is degraded into byproducts (hematoidin, hetoporphyrin, hemosiderin ) and leeches to the dentin wall. This gives a pink/purple hue. If this becomes irreversible pulpitis, the hemocomponents continue to breakdown and the tooth can appear to be blue/grey in appearance.

**Reversible vs. Irreversible Pulpitis**

*Reversible pulpitis* is pulpal inflammation that over time returns to a viable pulp cavity. It can be caused by inflammation or trauma. The tooth discoloration may lessen with time and return to a normal ivory color. Radiography may not be beneficial with reversible pulpitis until pulp canal or apical lesions occur. Pain, however is very common in humans with acute periapical periodontitis and acute periapical abscess so please consider this the same for dogs and cats.

Ideally, it would be great to know the exact time of trauma or inflammation but usually it is not known. However, if the tooth tip or the coronal ¼ of the crown becomes acutely discolored, aggressive therapy should be performed initially to see if the tooth would indeed revert to a normal, healthy pulp.

*Irreversible pulpitis* can be a sterile event or it can be bacterial related. It can also be acute, subacute or chronic in nature. If bacterial infection combines with the hemoglobin breakdown products, iron sulfide is formed which causes a dark grey or blue color to the tooth. Once that color appearance is noted, it is almost pathognomonic for irreversible pulpitis.

According the Hale study, *“Inflammation within the pulp during the period of irreversible pulpitis stimulates nerves within the pulp leading to the perception of pain. Intact teeth undergoing necrosis are often more acutely painful compared with open-chambered, fractured teeth based on increased intra-cranial pressure. Over time, the pain diminishes with the onset of pulp necrosis. However, over time tissue-breakdown products, which include several mediators*

*of inflammation, may extend beyond the apical delta into the LDL space leading to periapical periodontitis which is quite painful (dull pain).* (Hale FA. Localized intrinsic staining of teeth due to pulpitis and pulp necrosis in dogs. *J Vet Dent* 18[1]:14-21 2001 Mar)

### **Radiographic evidence of a non-vital tooth**

Dental radiography plays an important role in identification of irreversible pulpitis, but it is not solely. With regard to the above-mentioned study by Hale, radiographic signs of endodontic disease were not present in 42.4% of these teeth. Therefore, the eyes (seeing intrinsic staining) and radiography do make for a very powerful diagnosis of over 92%

Radiographic signs: Wide pulp cavity with relation to the contralateral tooth; apical rarefaction (apical lucency); Narrowing of the pulp cavity due to pulp calcification (localized or generalized); and root resorption.

### **Treatment for reversible and irreversible pulpitis**

*Reversible pulpitis:* Therapy includes antibiotics (Ampicillin/Clavulinate or Clindamycin) for 7 days plus NSAID therapy for 7 days (providing renal and hepatic function is normal). If the discoloration does not return to normal after 2-3 months, it can be assumed that this tooth now experiencing irreversible pulpitis which requires immediate therapy.

*Irreversible pulpitis:* Root canal therapy or extraction is the two treatment options for a non-vital tooth.

Root canal therapy is highly recommended for strategic teeth in the mouth (maxillary and mandibular canines; maxillary 4<sup>th</sup> premolars and mandibular 1<sup>st</sup> molars) as well as lateral incisors in large breed pets. Root canal therapy removes the necrotic pulp, and hermetically seals the tooth root canal system with materials that allow the tooth to remain in the mouth for the life of the pet. Success rate of root canal therapy is over 90%. In many instances, a full coverage crown is recommended.

Surgical extraction is the other treatment option for an irreversible pulpitis tooth. While the success rate is 100% (provided dental radiography is performed after the procedure to confirm complete extraction), it does have limitations and consequences. Firstly, extractions can cause more pain than endodontic therapy and involve a longer recovery period. If the pet plays and retrieves or likes to chew, removing a strategic tooth may compromise that aspect of the pet's life. Removing a mandibular canine tooth has its own unique set of risks involved such as mandibular fracture or trauma to adjacent incisors and premolars. The tongue may loll or fall to one side once the canine is removed and this becomes an undesirable side effect of the removal.

## ORAL TUMORS IN THE DOG AND CAT - IT IS PRESENT MORE THAN YOU THINK!

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### Introduction

Oral tumors compromise 5.3% of all neoplasia in the dogs and 6.7% in cats.<sup>1</sup> Therefore, it is necessary for the clinician to be diligent in oral examinations and diagnostics. This lecture will review the most common oral tumors in dogs and cats, and treatment plans for each. The most common oral malignancies in dogs in order of occurrence: Malignant melanoma, squamous cell carcinoma, fibrosarcoma. In the cat, squamous cell carcinoma

### Malignant versus benign tumors

Malignant tumors tend to destroy bone and soft tissue, while leaving teeth in their normal arcade positions. This gives the impression of teeth being suspended in soft tissue with marginal bone. Benign tumors can move teeth due to the soft tissue expansion, thereby redirecting teeth.<sup>1,2</sup>

### Malignant melanoma (MM)

This is the most common oral tumor in the dog. Sex predilection: Males with a male-to-female ratio of 1.4-6.0:1. Typically occurs in older dogs (mean 11 years). Cocker spaniels, Labrador retrievers, Golden retrievers and German Shepherds and dogs with heavily pigmented oral mucosa may be predisposed. Non-pigmented (amelanotic) tumors do occur as well (33%) Melanomas are rare in cats. Otherwise, dark pigmented raised masses are noted.

These tumors have focal infiltration, with early metastasis to regional lymph nodes. Metastasis to the lungs and liver are less frequent. Bone destruction is common.

Location – Any part of the oral cavity including the dorsal tongue surface and lips. Encompassing mandibular and maxillary together, 32% were located rostrally and 20% were located caudally,

Diagnostic tests- Intraoral radiographs are needed to assess bone involvement (57%). FNA of mandibular lymph node, incisional biopsy. Once MM has been diagnosed, conventional oncology workup is recommended (CT, CBC, Serum chemistries, UA, 3 view thoracic radiographs and abdominal US).

Treatment options – Curative intent surgery with wide margins (1-2 cm margins), even as a sole treatment often extends PFI (Progression free interval) and ST (Survival times). Metastasis at time of diagnosis carries a poor prognosis and a lower ST. Rostral mandibular and maxillary masses provide the surgeon a more favorable clean tumor-free margin. Also, tumor size directly affects the ability for a surgeon to achieve clean surgical margins. Other therapy: Maximum tolerable dosage (MTD) chemotherapy (Carboplatin), xenogenic canine melanoma vaccine, radiation therapy, metronomic chemotherapy (combination of doxycycline, NSAID, cyclophosphamide), and interferon.<sup>1,2,3,4</sup>

Survival times – The survival time is short, ranging from <4 months to 5.8 months and 8 months in other studies. However, a longer survival time was noted with dogs with histologically well-differentiated melanocytic neoplasms (Mean survival time of 23 months and median survival time of 34 months after surgery)

**Squamous cell carcinoma (SCC) – Non-papillary / Non-tonsillar**

This is the most common oral malignancy in the cat and 2<sup>nd</sup> most common one in the dog (17-25%). This occurs in older dogs (mean 8-10 yrs) with larger dogs overrepresented. The gingiva is the most common site for this neoplasia. The gingiva usually appears ulcerated with secondary bone involvement (77%). Metastasis to regional lymph nodes is rare (<10%) and low to moderate metastasis to the lungs in dogs is noted (3-36%). Some facial changes (exophthalmos) can be noted.<sup>3</sup> These masses are slow growing, locally destructive mostly on the buccal mucosa. (See comparison of this mass with papillary SCC)

Location- In the dog, the gingival mucosa is the most common site. In the cat, premolar / molar area of maxilla, premolar region of the mandible, and sublingual lesion.<sup>3</sup> Metastasis is late to regional lymph nodes and distant organs. SCC is locally aggressive with bone involvement. Tonsillar and lingual SCC are less common but have a higher and earlier metastatic rate.

Diagnostic tests- Incisional biopsy and regional lymph node aspirates are recommended. Once the non-papillary SCC has been diagnosed, conventional oncology workup is recommended.

Treatment options- Wide surgical excision (1-2 cm margins). Rostral mandibular SCC is more favorable with cats but case selection prior to aggressive excisional surgery must be considered. Rostral mandibular provide a more favorable long-term prognosis. SCC is responsive to radiation therapy with a medium survival time (MST) of 16 months. Radiation is radiosensitive but not radiocurative. Cisplatin and piroxicam have been reported to be effective.<sup>3</sup>

**Papillary SCC (PSCC)**

Previously thought to only occur with young dogs, this form of SCC can occur with middle to older aged dogs as well. The mean age is ~4 years (0.5-9.0 years) in a 9 dog study. CT of these lesions showed bone lysis with or without osteoproliferation. These masses are more infiltrated, rapid growth, and atypical cellularity. PSS do not metastasize.<sup>5</sup>

Location - Most were large breed dogs and the most common location was the rostral maxilla (7/9), however, tumors were noted in the rostral mandible and mid/caudal maxilla.<sup>2,3,5</sup>

Diagnostic tests – Same as SCC

Treatment options – Surgical wide margins (1 cm) provide excellent clinical results.

**Fibrosarcoma (FSA)**

This neoplasia is the 3<sup>rd</sup> most common neoplasia in dogs (7.5-25%) and 2<sup>nd</sup> most common malignancy in cats (13%). The median age of 7.3-8.6 years in dogs, and <25% of dogs are <5 years of age. In cats, the average age is 10.3 years. There is a sex predilection of male to female of 1.4-2.8:1. Larger breed animals >50# (Golden Retrievers) have a higher predisposition for FSA. Metastatic potential is low and can occur late in the disease process with lymph nodes (19-22%) and lungs (6-27%) in dogs. The low metastatic potential is the same in cats.

Clinical appearance show a firm, flat, multilobulated and deeply attached to the underlying tissue with rare ulceration noted. Bone lysis occurs in 72% of canine cases.

These tumors are histologically low-grade and biologically high-grade which potentially provides confusion to the DVM when interpreting an aggressive oral tumor. These tumors may be misdiagnosed as benign fibromas or low-grade sarcomas. High grade anaplastic oral FSAs have a more metastatic potential than do low-grade tumors.

Location – The site predilection in dog is maxillary arcade between the canines and carnassial teeth (56-87%), hard palate (7-17%) and buccal or labial mucosa (4-22%). There is no site predilection in the cat.

Diagnostic tests – After initial incisional biopsy, routine staging with FNA of mandibular lymph nodes, 3 view orthogonal thoracic images, serum chemistries/CBC/UA and CT.

Treatment options – Wide surgical excision (2 cm) is warranted. Local recurrence occurs more frequently than metastasis. Radiation therapy post wide excisional surgery, radiation therapy alone, and radiation therapy with local hyperthermia can prolong the survival times.

Survival times- Median survival time (MST) is approximately 11-12 months for both mandibular and maxillary FSA resection with local recurrence rate of 46%. Radiation therapy MST is 6-26 months.<sup>2,3</sup>

### **Osteosarcoma (OSA)**

Oral osteosarcomas are the 4<sup>th</sup> most common oral tumor in dogs (6-18%). Feline oral OSAs are much less frequent (2.4%). Medium to large breed dogs that are middle aged to older are mostly represented. Females appear to be more represented.

Location – Most OSAs occur in the maxilla (43%) followed by mandibular (32%) and the calvarium (23%).

Diagnostic tests – After incisional biopsy, regional lymph node aspirates, along with conventional oncological workup.

The metastatic rate of oral OSA is lower than the appendicular counterpart. Occurrence in the mandible and maxilla are noted, with a more unfavorable outcome with OSA in the TM joint and caudal maxilla / mandible.

Treatment options – Wide radical excision (1-2 cm) of the tumor should be performed if possible. Dogs treated with surgical excision had a Median Survival Time (MST) of 329 days. Surgery resulting in complete excision improved prognosis, whereas calvarial tumor location and increased monocyte count were associated with a poorer prognosis. Radiation therapy and chemotherapy have not shown a decrease in hazard of death progression.<sup>3,6</sup>

### **Odontogenic tumors**

These tumors are derived from ectodermal, ectomesenchymal, or mesenchymal components of the tooth forming apparatus. These include Canine acanthomatous ameloblastoma (CAA), peripheral odontogenic fibroma (POF), and focal fibrous hyperplasia (FFH). Of the three, POF and FFH are relegated to the gingiva only.<sup>7</sup>

**Canine acanthomatous ameloblastoma (CAA)** – In a recent study of odontogenic tumors, CAA occurred 45% (68/152). This is an aggressive benign odontogenic tumor that is non-inductive in nature; therefore, the cells of ameloblastic origin do not induce the surrounding mesenchymal cells. Therefore, no dental hard tissues formed and is a soft tissue neoplasia. These raised, lobulated masses also cause local bone infiltration and tooth displacement. Metastasis to regional lymph nodes or distant organs has not been reported. CT is recommended prior to oral surgery to establish bone involvement. 1-2 cm margins are recommended. Intralesional bleomycin has been documented to resolve this oral mass with no recurrence. Local side effects to bleomycin injections have been documented. Predilection to the rostral mandible is common.<sup>7,8,9</sup>

**Peripheral odontogenic fibroma (POF)** – These are slow growing masses. These benign masses are not locally invasive, and occur in 31% of odontogenic tumors. Clinically, they appear as rough-surfaced masses on the gingiva. Radiographically and histologically, there may be dystrophic calcification within the mass, but no alveolar bone involvement. As with other odontogenic tumors, tooth movement due to expansion of the mass is possible. Regional

distribution is mostly the rostral maxilla (47%) and caudal mandible (21%), but these masses may occur anywhere along the gingival margin. There is controversy whether these tumors are actually remnants of the periodontal ligament, and whether removal of the tooth and adjacent periodontal ligament is warranted. Some recommend removal in the reactive zone and the surrounding pseudocapsule. Others recommend a more aggressive approach to remove the tooth and the PDL, which means removal of alveolar bone that supports the tooth, to achieve complete removal.<sup>3,7</sup>

**Focal Fibrous Hyperplasia (FFH)** – This encompasses 16% of odontogenic tumors in the dog. Clinically, these appear raised, smooth and sometimes very firm. Regional distribution of these masses are mostly relegated to the rostral maxilla (57%) as well as rostral (22%) and distal (17%) mandible. Surgical removal is similar with POF.<sup>3,7</sup>

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**Flaps and Flops...How The Right Flap Can Make Extractions Easier**

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**Introduction**

Whether you are extracting a maxillary or mandibular two rooted tooth, or extracting a maxillary canine or 4<sup>th</sup> premolar tooth, flap decision-making is essential. This is also essential in repairing an oronasal fistula. Your first flap should be your last one, when performing oral surgery. This lecture will address a variety of flap designs. Finally, understanding why flaps fail will prevent them from occurring in the future

**Design Tenets**

- 1) Flap must be large enough to allow proper visualization and manipulation of the surgical area
- 2) The base of the flap must be as wide or broader than the marginal release
- 3) Edges of the flap must be over intact bone when suturing
- 4) Zero (NO) tension when closing surgical site
- 5) Simple interrupted, cruciate, or embedded sutures may be used. Distance between sutures is 2-3 mm. (1)

**Suture material choices**

Small dogs and cats: 5-0 Poliglecaprone 25 or Chromic Gut with P3 needle

Medium to larger dogs: 4-0 Poliglecaprone 25 or Chromic Gut with FS-2 needle

**Equipment**

In order to perform adequate oral surgery, it is necessary to have proper equipment. This author recommends having sharp equipment in order to perform adequate procedures.

A few items recommended by this author will be shown in the lecture.

- #15 or #15C blade
- Periosteal elevator (author recommends Zoll Dental EX-52)
- Small oral surgical scissors (Iris, Goldman-Fox, etc...)
- Small Olsen-Hegar or Mayo-Hegar needle holders (4 ½" are idea)
- Miller bone curette
- Medium grit football and/or round diamond bur
- 4-0 and 5-0 absorbable suture material (4-0 poliglecaprone-25 or chromic gut)
- Tissue retractors and/or Minnesota retractor
- Direct or indirect digital dental imaging system

**Line angle definition** and principles of vertical releasing flaps: “A line angle is an imaginary vertical line forming the intersection of two adjacent vertical dental surfaces.”(1) Principles of line angle technique are defined to prevent trauma to adjacent teeth OR by having a vertical releasing flap overlying the surgical site alveolus, thus violating a basic tenet of oral surgery. A visualization of line angles will be shown in this lecture.

### **Envelope flap**

This flap should be used for maxillary (2<sup>nd</sup>-3<sup>rd</sup>) or mandibular premolar (2<sup>nd</sup>-4<sup>th</sup>) extractions. It may be used for maxillary 4<sup>th</sup> premolars and mandibular 1<sup>st</sup> molars as well. This flap is a full-thickness or split thickness flap used to access root surfaces without a vertical component. It is therefore replaced to its original position (1). When possible, this flap should be considered as there is less risk of vertical flap complication. A demonstration of this flap will be shown.

### **Single vertical releasing pedicle flap (triangle or 3 cornered flap)**

This dentist performs this flap when extraction of maxillary canine, maxillary 4<sup>th</sup> premolar, and mandibular canine teeth. This slightly divergent single vertical flap allows for excellent exposure of the extraction site, while preserving the distal aspect of the flap from any vertical component. When making a vertical release, a mesial divergent release is made. As blood flow arrives from distal to mesial in the maxilla and mandible, it is important to preserve blood supply to vital gingival tissue.

### **Bilateral diverging vertical releasing flap (four-cornered)**

This flap may be used for the above indications of the triangle flap. This flap does allow the practitioner complete visualization of the surgical site. Care must be made to avoid anatomical landmarks (parotid papilla, middle mental foramina). The author prefers this flap when correcting oronasal fistulas.

### **Periosteum release**

This is the most essential part of the oral surgical procedure. Release of the periosteum allows complete tension-free closure. Failure to release the periosteum will frustrate the clinician and surgical failure will inevitably occur.

**Removal of necrotic epithelium** or down growth of tissue (especially for deep pockets and oronasal fistulas). Failure to remove this tissue will result in suture dehiscence.

### **Home care and recheck examination**

Adequate home care will help oral surgical sites heal normally. Failure of the clinician to provide home care guidelines may lead to deleterious healing and contribute to patient morbidity. This will be reviewed in this lecture.

### **Reference**

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## Gingivostomatitis in Cats: What's New and What's Proven

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### CLINICAL PRESENTATION

This is a syndrome associated with pain on eating and/or opening the mouth. There is noticeable pawing of the mouth, dysphagia, weight loss, bad general condition, grooming deficiency, ptalism, and sometimes bleeding from the mouth. There is generally inflammation associated around the teeth, as well as in some cases caudal mucositis (caudal to the dental arch and lateral to the glossopalatine folds). This appears to be a multifactorial process of which the body cannot adequately respond to local inflammation of bacteria or viral pathogens. Calicivirus has been implicated with this disease process. While Bartonella has been discussed as a possible etiology to GS, it has been shown NOT to be involved with this disease process.

Clinically, the presentation of gingivostomatitis can be localized or generalized. Periodontitis and tooth resorptions (TR) may present with areas of inflammation so dental radiography is needed to differentiate among the three. Inflammation in the palatoglossal folds is a hallmark sign of generalized feline chronic gingivostomatitis.<sup>2</sup>

According to Lommer, "While it is believed that feline stomatitis results from an inappropriate immune response to oral antigenic stimulation, the initiating cause is usually not identified, may differ from case to case, and is likely multifactorial".<sup>3</sup> Although underlying immunological abnormalities have not been identified, an increase in mRNA for IL-2, IL-4, IL-6, IL-10, IL-12 and IFN- $\gamma$  have been identified with chronic stomatitis. Most noticeable in caudal stomatitis are IgG plasma cells and cytotoxic T-cells which could support the possibility of viral etiology in the development of this disease process. Plaque bacteria can stimulate the immune system that appear to contribute to ongoing inflammation. Successful treatment of chronic stomatitis requires minimizing this plaque.<sup>3</sup>

### INITIAL THERAPY

Sedation exam with periodontal cleaning, probing, and radiographs is essential. This may have to be done every 3-6 months. Initiation of home care is paramount if the client is going to try a more conservative therapy. Assessment of horizontal bone loss and aggressive gingivitis is needed. Home care should be initiated and clients should be told that this has to be done every day. Therapy is to include the following: daily tooth brushings, chlorhexidine oral rinses bid, VOHC-approved water additives, and a plaque-retarding polymer. However, home care is usually insufficient due to the pain of the inflammation, the unwillingness of the cat to accept therapy, or lack of owner to perform daily.

With most cases, full-mouth or caudal mouth (caudal to the mandibular and maxillary canines) is warranted. It is imperative that dental radiography be utilized with this treatment, as tooth remnants left will not allow proper healing of the gingival and caudal oropharyngeal lesions from healing. Therefore, crown amputation of teeth is not a viable treatment option. The effectiveness of dental extractions has been shown to be: 55% cure, 35% markedly improved, 10% no improvement. Therefore, approx. 90% of cats responds favorably to extractions.<sup>1</sup> If the canines do not appear to be involved and the lesions are caudal to them, it is recommended to first consider caudal mouth extractions and spare the canines. However, if the canines and/or incisors have any inflammation present it is recommended removing them and the incisors (full mouth extraction).

Extraction technique is very important as proper flap technique, complete extraction of teeth, alveoplasty

of marginal bone with an assortment of diamond burs (round and football), and tension-free closure utilizing 5-0 chromic gut, Poliglecaprone 25, or Polyglactin 910 with a P3 needle. Utilization of assorted winged elevators and small luxators help facilitate complete tooth extraction. Post extraction radiographs are essential to properly confirm complete removal.

### **REFRACTORY CASES (TREATMENT BEYOND EXTRACTION THERAPY)**

No one treatment has shown superiority to another with regard to refractory caudal mucositis. In a recent paper by Hennes at the 2011 Veterinary Dental Forum, he described a study (Harley et al 1999) in that there was a comparison of the effect of methylprednisone, spiramycin-metronidazole, sodium aurothiomalate and chlorhexidine over a 3 month treatment showed none of the agents were able to resolve the underlying pathology present in local gingivostomatitis cases at either a clinical or molecular level.

### **ANTIBIOTICS**

These should be used sparingly in gingivostomatitis cases as a primary treatment regimen. However, during refractory cases (after extractions) there may be a need for a course (3 weeks) of antibiotics. The most commonly used drugs are clindamycin, amoxicillin-clavulanic acid, doxycycline, and spiramycin-metronidazole. This helps to decrease the oral bacterial load over a significant period of time and there is improvement clinically. While many have chosen Azithromycin in Bartonella-positive cats with GS, a recent study (Dowers et al. 2010) failed to show a correlation between GS and Bartonella.

### **NSAID'S AND OPIOIDS**

NSAID's and opioids can certainly be used for pain therapy. With the addition of Robenacoxib, this approved NSAID therapy does have a place in pain management. NSAID's are better used for inflammatory control of Calicivirus positive cats. Buprenorphine therapy (0.02 mg/kg) sublingually q 8-12 hrs provides analgesia as well.

### **GLUCOCORTICOIDS / IMMUNE MODULATING DRUGS**

These can be used but try to avoid high doses in Calicivirus-positive or herpes-positive cases. Taper a 3 week regimen. If cyclosporine therapy is to be initiated, avoidance of steroids is advised.

Immune modulating drugs besides glucocorticoids, aurothiomalate (gold salts), cyclosporine, Omega interferon, and chlorambucil have been used. Feline Recombinant Interferon (Verbagen Omega) has shown promise in some refractory cases.

Cyclosporine inhibits T-cell activation by blocking the transcription certain pro-inflammatory cytokines which include IL-2 and IL-4. Need to evaluate every 2 weeks to identify cyclosporine blood levels. After 6 weeks of therapy, 52.7% improvement SDAI (Stomatitis Disease Activity Index). Establishment of tough levels of Cyclosporine was noted. Whole blood cyclosporine levels >300 ng/ml (72% improvement) while cyclosporine levels <300 ng/ml showed only a 28% improvement. (cat had to have undergone either premolar/molar or full mouth extractions) It is important for tough levels to be done on an empty stomach. It is best to avoid previous corticosteroid usage.<sup>3</sup> Potential side effects include toxoplasmosis for outside cats.<sup>2</sup>

Dosage is 2.5 mg/kg of Cyclosporine (Neoral) compounded with 1 ml cod liver oil with tuna base (60 mls). Standard is 1.0 mls po bid x 6 weeks.<sup>3</sup>

### **STEM CELL THERAPY**

Current study by UC Davis Dentistry and Oral Surgery Service and the Regenerative Medicine Laboratory: As stem cells are known to have anti-inflammatory and regenerative properties, this may have value going forward. The adipose tissue was harvested from an affected cat with GS and then injected back into the cat (IV) after culture expansion and characterization.

There is a potential side effect of blood clots and transfusion-like reaction so 48-72 hrs of hospitalization/monitoring needed post injection. Two sets of treatment, four weeks apart were performed on cats with non-responsive GS and the cat was rechecked monthly afterwards with ~50% success rate. The clinical

trial is still ongoing and the group is investigating the use of an autologous, allogenic and intralesional administration of stem cells.

### **CO2 LASER THERAPY**

This has been written in the J Vet Dent (Lewis et al., 2007). This case required multiple laser therapy and also rescue corticosteroid and IV fluid therapy.

Therapeutic lasers have received attention with regard to healing post surgical tissue, but no information is presently available regarding refractory GS and therapeutic laser tx.

Hennet P. Feline chronic gingivostomatitis: Extraction and what else? Advanced Lecture Series. Veterinary Dental Forum 2010. Boston, MA.

<sup>2</sup>Lommer MJ. Oral Inflammation in Small Animals. In Holmstrom SE. Veterinary Clinics of North America Clinical Veterinary Dentistry. May 2013. Elsevier; Philadelphia. Vol 43(3); 555-571.

<sup>3</sup>Lommer MJ. Efficacy of Cyclosporine for Chronic, Refractory Stomatitis in Cats: A Randomized, Placebo-Controlled, Double-Blinded Clinical Study. In J Vet Dent; Spring 2013; Vol 30(1); 8-17.

## THE PRURITIC CAT: REACTION PATTERNS, DIFFERENTIAL DIAGNOSES, and DIAGNOSTIC APPROACH

Douglas J. DeBoer, DVM, Diplomate ACVD

Presentation of a cat where the primary owner complaint is pruritus is common in general veterinary practice. Feline pruritic skin diseases tend to occur as “reaction patterns” (eosinophilic granuloma, miliary dermatitis, ventral symmetrical alopecia, etc.), but each of these reaction patterns tend to have the same list of underlying causes associated with it. Thus, for any feline pruritic condition (regardless of lesions) it is reasonable to use a uniform, systematic approach in an attempt to determine the underlying cause.

One consideration may be to convince yourself and the owner that the cat is indeed pruritic and removing hair intentionally, rather than the hair “falling out by itself,” by performing a trichogram. Broken hair tips suggest removal by self-trauma of licking and may convince the owner, especially if the cat has been hiding in order to remove hair.

### Is it a Parasite?

Start with careful examination and thorough flea combing. The finding of fleas or flea stool prompts an immediate diagnosis of flea allergy dermatitis until proven otherwise. In heavy flea areas, it may be necessary to provide continual monthly flea preventive as a “diagnostic therapy” test. Remove the hair and scale from the flea comb and pull it apart with the fingers, allowing scale and debris to drop onto the table. Collect this material from the table and examine in mineral oil for mites such as *Cheyletiella*. In cats, a proper parasite examination should consist of superficial scrapings, deep scrapings, and hair pluckings, each examined microscopically in mineral oil. The recent finding of **three** different species of *Demodex* mites in cats makes this especially important. When in doubt, provide monthly applications of fipronil or selamectin, which will control all parasites except *Demodex*.

### Is it Infection?

The next step is skin cytology. Surface material can be collected by direct impression (as with moist lesions) or by collection of material with a small spatula or with cellophane tape. Fortunately for cats, bacterial skin infections are uncommon, except as surface infections when the surface is moist and exudative; most infections are with cocci. Many dermatologists have seen rather bizarre, pruritic dermatoses in cats with a moist surface, cocci on cytology, and a surprisingly dramatic response to antibiotics. Therefore, the finding of surface cocci should prompt a 2-4 week course of oral antibiotics with assessment of response.

Yeast overgrowth with *Malassezia* is more common, and the finding of yeast should prompt a trial of antifungal treatment. Because dermatophytosis “can look like anything” dermatophyte culture is typically considered a basic diagnostic test that should be performed in all feline dermatoses, and is most conveniently performed using the toothbrush technique.

Whatever the cytologic findings, following treatment a recheck should be scheduled in about 2 weeks. Nearly all skin infections are secondary, and the recheck examination provides the opportunity to see the “real” disease without being masked by infection.

### What About Blood Tests? Should I Biopsy It?

Though routine hematologic evaluations are typically normal in most feline dermatoses, a blood count and serum chemistries should be considered in any older cat with recent onset of disease; or any pruritic dermatosis that has an unusual appearance. Occasionally, pruritus can

be a manifestation of internal disease or of a paraneoplastic syndrome. In addition, future steroid therapy may be contemplated and it is wise to evaluate any middle- to older-age cat systemically prior to such treatment.

The decision to biopsy is often a difficult one, mostly due to cost-benefit considerations. The most common feline pruritic dermatoses (parasites, allergies, infections) are *not* readily diagnosable by biopsy. Rather, the purpose of biopsy is to rule out other, uncommon to rare feline dermatoses such as pemphigus or epitheliotropic lymphoma. Therefore, biopsy should not be considered unless the clinical appearance is unusual, the disease is especially severe, or the disease has been recalcitrant to treatment. In any event, do make sure all secondary infections are cleared up prior to biopsy, as these may influence the histologic results.

### Is it Allergy?

Before deciding that allergy is the cause of a cat's pruritus, all of the above more common causes of itch should be ruled out.

- **Is it Food-Related?** Initial "allergy evaluation" typically begins with a dietary restriction-provocation trial in cats. There is no clear advantage of a home-cooked vs. a commercial hypoallergenic diet; the most important factor is that compliance with the dietary restriction is complete. Thus, choose a diet that the owner is happy to feed, and the cat is happy to eat. There is currently *no evidence* to support the use of serologic tests to support a diagnosis of food allergy! These tests are fraught with false positive and false negative results, and are therefore useless for initial diagnosis. The initial trial should be for 4 weeks, with an additional 4-8 weeks if improvement is occurring. Most importantly, if response is apparent, challenge with the original food to prove the food is responsible and not some other coincident factor.
- **Is it Environmental Allergy?** Diagnosing a cat with environmental allergies is difficult at best. There are no good, uniform criteria for "feline atopic dermatitis" as there are for the canine disease. Diagnosis rests primarily on exclusion: the finding of a pruritic cat where all other diagnostic and therapeutic measures have been exhausted, and there is simply no other choice left. Allergy testing, in particular, is problematic. Intradermal tests are difficult to perform and interpret in cats. Most important for the practitioner to understand is the use of serologic IgE tests in cats. Studies generally demonstrate that normal cats, or pruritic but nonallergic cats (for example, cats with fleas) are positive on these tests just as often as "truly allergic" cats! Thus, serologic tests must never be used to make a diagnosis of allergy. Rather, their only benefit is to attempt to determine what the relevant sensitivities may be in order to provide allergen immunotherapy as a treatment.

### Facial Dermatitis: A Pruritic Reaction Pattern

Feline facial dermatoses represent a reasonably common clinical complaint, but a very wide variety of underlying diseases forming a rather unique subset of conditions.

- **Parasites.** Facial dermatitis related to a parasite is most commonly related to a mite infestation, rather than to something more common like fleas or cheyletiellosis. Feline scabies (*Notoedres*) is rare, but causes an extremely pruritic dermatitis of the face and neck. One of the newly-described *Demodex* mites, such as 'gatoi' or the 'unnamed' mite may cause contagious, pruritic facial dermatitis. The good news is that all of these mites are easily found, and mostly easily treated!
- **Infections.** Not a common primary cause of dermatitis restricted to the face, with the strong exception of dermatophytosis. Nevertheless, cytology should be routinely performed to check for any bacterial or yeast overgrowth, which is typically secondary.

Wood's lamp examination and culture are mandatory in ANY facial dermatitis, no matter the appearance. The other important infectious cause of facial dermatitis is feline herpesvirus 1 (FHV1). Though rare, this aberrant infection causes severe ulcerative, necrotic, erosion to plaquelike lesions on the face which may be mistaken for other diseases such as eosinophilic granuloma. This is a good example of why it is important for any cat with persistent, rather severe, ulcerative facial dermatitis to be biopsied; generally the virus can be found by direct histologic examination or PCR.

- **Allergic Disease.** In theory, food allergy, environmental allergy, or insect allergy can all manifest as facial dermatitis. Of these, food allergy should be a primary suspect in feline head/neck pruritus and dietary restriction/provocation trials are mandatory.
- **Pemphigus Foliaceus (PF).** The most common autoimmune skin disease of cats, PF typically has a strong component of facial distribution, particularly on the bridge of the nose, around the eyes, and on the ear pinnae. Most cats also have involvement of their footpads and/or nailbeds ('caseous paronychia') but this is not always the case. The clinical appearance of PF is rather dramatic and unusual, however, it is important that biopsy confirmation is always obtained for this disease: there have been reports of PF-like disease in cats caused by primary infections, particularly with dermatophytes.
- **Feline Acne.** There is much 'myth and legend' over the causation of feline acne. Most cases are probably idiopathic, perhaps with a cause similar to that in humans – defective cornification in the follicle or sebaceous duct leading to "plugging" with keratinaceous debris and secondary infection. Rarely, a defined cause such as primary bacterial infection or dermatophytosis may be responsible. Causes such as the type of food bowl, food allergy, failure of chin grooming, etc. are much more speculative and there is no convincing evidence for this pathogenesis. Treatment relies initially on clearing any secondary bacterial infection with antibiotics, then providing daily facial hygiene with keratolytic topical products (2-3% benzoyl peroxide, or salicylic acid), which may need to be done periodically on a longterm maintenance basis. For recalcitrant cases, the author has had success with topical tretinoin gel or cream (0.025%, applied twice daily until resolution and then once every 1-2 days to maintain remission if needed).
- **"Dirty Face"** in Persian cats is an uncommon but frustrating idiopathic facial dermatitis that is considered by some to be a more severe form of feline acne. In this disease, comedones and crusts extend beyond the chin area to the facial folds and preauricular areas. Diagnostic evaluations should be performed as above to rule out definable causes, but most cases are idiopathic and may be a genetic alteration in this breed. Therapy is symptomatic, using topicals as for feline acne.
- **Indolent Ulcer.** Indolent ulcers appear as well-demarcated ulcer with raised borders present on the margin of the upper or lower lip. Initial diagnosis is generally straightforward, based on clinical appearance and cytology: remove surface debris with a gauze pad to expose the most underlying tissue and take impression smears. The finding of eosinophils on cytology is sufficient to make an initial tentative diagnosis. Note if cocci are also found, especially intracellularly, and if they are found, treat first with antibiotics as this disease can sometimes reflect an aberrant response to bacterial infection. In an otherwise healthy, young animal, it is acceptable to treat the cat with ONE course of injectable corticosteroids (20 mg methylprednisolone acetate, every 2 weeks for a total of 3 injections). If the disease is especially severe, recalcitrant, or recurrent, a search for an underlying cause, should be made (including biopsy with assessment for FHV1 presence) and repeated corticosteroid use should be avoided. Recalcitrant cases of idiopathic origin can often be successfully treated with ciclosporin (10-12 mg/kg/day for 4 weeks, then taper).

- **Feline Neck Ulcers (Idiopathic Ulcerative Dermatitis).** Feline idiopathic ulcerative dermatitis is an uncommon skin disease in cats of any age/breed/sex, characterized by large non-healing ulcerations on the dorsal midline of the caudal neck/shoulders. The lesion may start as a focal area of alopecia, but then progresses to erythema and ulceration. Cats will often self-traumatize dramatically leading to a “vicious cycle” of trauma and worsening ulceration. It is uncertain if pruritus or pain are involved, though there is speculation that some neuropathic “hypersensitive nerve ending” syndrome may contribute. Injection site reactions have been blamed in the past, but clearly this is not always the case, and most cases are truly idiopathic. Differential diagnoses include physical or chemical injury, injection-site reactions, foreign bodies, fungal, bacterial and viral infections, hypersensitivity disorders, and even a neoplasm. The standard “flowchart approach” should be taken, first ruling out parasites, then assessing for infection via cytology and culture (the latter best done with fresh tissue). Biopsy may be useful, especially searching for any evidence of unusual infections (viral inclusions) or foreign body reaction. Treatment has been remarkably difficult for many cats. Most recently, there have been case reports of cats responding to either oclacitinib (1 mg/kg BID) or topiramate (2.5-5 mg/kg BID).

## DIAGNOSIS AND MANAGEMENT OF FELINE OTITIS

Douglas J. DeBoer, DVM, Diplomate ACVD

Otitis externa (OE) is very common in daily practice, though much more so in dogs - accounting for around 15% of canine patient visits, yet only around 5% of cat visits in the USA. This lecture will focus on what we know about treating everyday otitis in cats and suggestions for how to increase success. We will also discuss a few rare causes of OE, as well as the emerging importance of otitis media (OM) in cats.

### Better Diagnosis with Cytology and Culture

There is no reliable correlation between the gross appearance of otic exudate and the causative organism (except, perhaps, in the case of ear mites). Ear cytology is really the only method that can help to identify organisms. Knowing the organism is an important determination in initial treatment. Cytology is a quick way to monitor progress at rechecks, and generates medically-justified revenue. *The main question in ear cytology is: are the organisms cocci, rods, yeast, or a mixture?*

Ear cytology is easy and quick, and an ideal procedure to teach your technicians. Insert a swab carefully and gently into the vertical ear canal, roll (do not smear) the contents onto a microscope slide, and heat-fix gently (especially if the exudate appears greasy). Any routine “Dif-Quik” hematology stain can be used. Examine using a good, well-maintained microscope and use 1000X oil-immersion. There are several possible cytologic results:

- Cocci. Here, there’s little problem, because this most likely represents growth of normal ear flora, and is unlikely to be antibiotic-resistant – though this is possible. But – check for inflammatory cells!!
- Yeast. Again, little problem, and easy to treat.
- Ceruminous debris, with few or no organisms. Inflamed, itchy ears without growth of microorganisms most likely indicates an allergic underlying etiology.
- Inflammatory cells. Here, caution is warranted, as the infection has gone beyond just “surface overgrowth” and is heading towards a soft-tissue infection.
- Rods. Caution here – more difficult to predict susceptibility and empirically choose an antibiotic. Important to recheck, and warn owner.

When should you consider a culture and susceptibility? Not for first-time, routine otitis. However, if the otitis is resistant to initial treatment or recurrent, especially if rods are present, it is clearly indicated. Remember, too, that this test reports susceptibility of organisms to tissue or plasma concentrations of antibiotics, and therefore is mostly relevant to choose a systemic antibiotic for more severe cases. The concentration of antibiotics in otic preparations is typically hundreds of times that attainable in plasma, so an “intermediate” result for an antibiotic means that the drug will probably work if used topically.

### Know Your Products: What Do I Reach For?

*Which antibiotic?* More than 80% of “everyday” otitis in cats involves *Malassezia*, *Staphylococcus*, *Pasteurella*, or coliforms. Staph and *Pasteurella* organisms almost always have broad susceptibility, and any of the common aminoglycoside antibiotics (such as gentamicin) should be effective. The same is true for most coliforms (~80%). Fluoroquinolone antibiotics are best reserved for *Pseudomonas* or other gram-negative bacteria WITH the appropriate susceptibility result. Polymyxin B is another possibility – it has increased activity

against gram-negative rods and could be considered as an alternative here. Bear in mind that this antibiotic is made less effective by exudate in the ear canal, so the ear must be clean to use it! Remember that fusidic acid is effective against Gram-positive organisms only, thus in ears, it should be used only if cytology indicates cocci. The newest antibiotic to appear in ear preparations is florfenicol. This antibiotic is similar to chloramphenicol; it is best used for otitis where cocci are present, as its activity against rod bacteria is highly variable and it is completely ineffective against *Pseudomonas*.

*Which antifungal?* If the organism involved is yeast, virtually any antifungal will work. “Resistance” of *Malassezia* to antifungals has been reported extremely rarely, if it exists at all. One misconception is that if yeast otitis is recurrent, it is a failure of antifungal treatment. Rather, this clinical situation reflects a continuing underlying inflammatory ear condition, usually allergy, and calls for corticosteroids rather than a different antifungal!

*Which steroid?* Almost all topical ear therapeutics contain a corticosteroid, usually a low to moderate potency drug and all are reasonably equivalent. Newer use of the “soft” steroids such as hydrocortisone aceponate or mometasone have the possibility of providing good anti-inflammatory action with less chance of adverse effects. Use of a steroid in the ear is critically important. It reduces physical narrowing of the canal, reduces cerumen secretion, and therefore makes it much more difficult for organisms to grow.

*What if the tympanum is ruptured?* First of all, most of the time it’s difficult to tell if the TM is ruptured, or not. Ototoxicity varies by species, and information is sparse in cats, though cats seem to be more prone to ototoxicity than dogs. We can say unequivocally that chlorhexidine is ototoxic if used at >0.2%, and I avoid it completely in cats. Common sense ways if the TM is obviously damaged, avoid the aminoglycosides if possible, but don’t worry too much about it.

*Should I use topicals in cats?* Some authors recommend avoiding topical treatment of cat ears, period – in part due to difficulty of application, and in part relating to the increased possibility for ototoxicity. Evidence-based studies to guide us in this regard are lacking.

### **To Clean or Not to Clean – and When?**

The simple answer here is: CLEAN, but be very, very gentle in cats. There is no question that debris, cerumen, and pus in the ear canal creates food, moisture, and “hiding places” for growth of microorganisms, impedes flow and efficacy of topical preparations, and inactivates some antimicrobials. Thus, initial cleaning of an infected ear (in the clinic) is important. Depending on how severe and sore the ear is, this may be done with or without sedation. This author’s bias is that the more gentle you can be, the better. Using mild, pH neutral detergent or solvent solutions is best in an inflamed, sore ear. Demonstrate to the owner how to hold the pinna up, instill the cleaner into the ear canal without touching the tip to the pinna, massage the canal to loosen and solubilize the debris, and then wipe or blot it out with a gauze pad around a finger. Repeat until the material being blotted is no longer colored or laden with debris. BE GENTLE!

There are two reasons that “cleaning solutions” are used: (1) to physically soften, dissolve, and remove debris; and (2) to leave an antimicrobial ingredient in the ear, to help with limiting growth of organisms. Reason (1) is most important in initial treatment. Choose a cleaner that is effective at loosening debris, yet gentle. I avoid use of acid-containing or low-pH cleaners in a sore ear. Once the ear has healed, and the goal is more the antimicrobial action, you can think more about the antiseptic ingredient. The latter is especially important in long-term “preventive maintenance” if it will be required.

Once the ear is initially cleaned in the clinic, have the owner apply topical medication per label instructions. My bias is to recommend no cleaning for the first 3-4 days, until the ear is more comfortable. At that time, the owner may begin GENTLE cleaning (as you have demonstrated) every 2-3 days if and as debris accumulates. Often, this is really not necessary, not to mention difficult for cats.

### The 5 Causes of Recurrent Otitis

When otitis is recurrent, there are really only 5 reasons why this situation exists. Note that there might be just one reason, or any combination of:

- An antimicrobial-resistant organism (most often, *Pseudomonas*)
- Ear pathology causing persistent stenosis or occlusion; this includes stenosis from edema, hyperplasia, or scarring; and occlusion from the same or from a mass lesion. In a young cat, the obvious example is a nasopharyngeal polyp; in older cats, a neoplasm.
- A persisting underlying cause that has not been addressed (the most common is allergy, though again, this is extremely common in dogs, less so in cats)
- Otitis media is present and has not been treated (an emerging problem – see below!)
- The owner is unable to comply with your recommended treatment

The key to successful treatment of recurrent otitis is to carefully consider each of these causes, one by one, determine which are present, and take steps to resolve each one.

### Maintenance Therapy

Maintenance therapy of ear disease is one of the most valuable, though most overlooked, aspects of dealing with recurrent OE. Typically, maintenance therapy will consist of:

- Regular cleaning at home (every 1-2 weeks), with an ear cleaner that contains an antimicrobial ingredient. This both removes debris, and makes the ear inhospitable to further growth of organisms.
- Often, regular application of a corticosteroid-only ear drop. Start daily, and then taper every 2-7 days as proactive *preventative* therapy. It is best NOT to use an antibiotic-steroid combination product long-term, as intermittent use of an antibiotic is a great way to lead to bacterial resistance!! Products containing 1% hydrocortisone are often too weak in potency to help much. Fluocinolone/DMSO drops (Synotic®, Zoetis) is the only moderate-potency, steroid-only ear drop that is made commercially. As an alternative, the author routinely uses a solution of 1 mg/ml dexamethasone in propylene glycol. To formulate this, mix one part of propylene glycol with 1 part of dexamethasone injection (the 2 mg/ml product). This is an inexpensive and effective longer-term treatment for inflammatory ear disease. It is also possible to use Cortavance® topical spray off-label for this purpose; most authors recommend 0.25cc drawn up in a 1cc syringe and instilled in each affected ear.

### The Plot Sickens: Primary Bacterial Otitis Media in Cats

Whether from more frequent use of advanced imaging or from more awareness, feline bacterial otitis media (OM) is receiving much more attention in the literature and in discussion amongst dermatologists. Though we often consider otitis media to be an extension of OE (it can be in cats, and it almost always is in dogs), recent focus has shifted to chronic sinusitis and respiratory disease creating “ascending” OM in cats.

- Several studies in the radiology literature have reported a high prevalence of “incidental” finding of bulla effusion in cats imaged for non-otologic reasons

- Studies at necropsy indicate a high prevalence of “incidental” OM in cats (grossly and/or histologically)...cats without any complaint of ear disease
- There are increasing reports of cats presenting with neurologic disease (head tilt, nystagmus) with bulla effusion on CT...sometimes with extension of the inflammatory process to meningoencephalitis
- Common organisms cultured in these cases are *Pasturella* and *Mycoplasma* – organisms highly associated with chronic sinusitis in cats.
- Shelter medicine veterinarians have recently reported outbreaks of *Streptococcus zooepidemicus* in cats, with respiratory disease, otitis media, and deaths.
- CONCLUSION: bacterial respiratory infections in cats quite commonly lead to otitis media; this may remain completely quiescent (most cats), or may erupt into fulminant disease at a later time.

What about treatment? In one recent small study of 16 cats with OM (11 of which had neurologic signs), eleven cats were managed only medically, though with a variety of protocols. Followup information was available for 9 cats – and 8 of these resolved without relapse. We can conclude that in cats presenting with clinical signs of OM, or otherwise diagnosed as OM, medical treatment may be a viable option if a mass lesion is not the cause.

## DIAGNOSTIC APPROACH TO THE PRURITIC DOG

Douglas J. DeBoer, DVM, Diplomate ACVD

Though there are many causes of canine pruritus, this is a clinical presentation that lends itself well to a systematic, stepwise approach. The author suggests a 3-step approach, in which the first step is to identify obvious causes such as parasites; step 2 is to eliminate secondary complications such as bacterial and yeast skin infections; and step 3 is to carefully observe the remaining clinical signs. This in combination with the patient's history often helps guide further diagnostic evaluation to find the true cause of the itch.

### Step 1. Identify the Obvious Things

The first step is to consider common and usually obvious things like parasites. A careful search for fleas, mites, lice, etc. should be made with a combination of the following steps:

- Flea combing and visual examination
- Check for history of application of flea preventive products and other antiparasitics
- Superficial and deep skin scrapings; trichogram (hair pluckings)
- Empirical treatment for scabies (very hard to find mite) – usually one of the avermectin or isoxazoline drugs

### Step 2. Extinguish the Fires

Most patients with pruritic skin disease of any severity have their primary disease complicated by one or more “layers” of secondary complications. This often makes it impossible to see what the true nature of the primary disease is. Therefore, it is critically important as a next step to **identify and treat any and all possible secondary complications**. This should be addressed on the first visit. The most common pruritic secondary complications are infections. These can be identified by physical examination of the skin, in association with skin cytology where necessary.

- Superficial staphylococcal pyoderma can be recognized by the typical appearance of papules, pustules, and epidermal collarettes. There is typically pruritus, ranging from mild to severe. All of these lesions, and in addition any draining or ulcerative lesions, should be assessed by cytology, looking for typical cocci organisms. If present, treat with appropriate antibiotic (or topical chlorhexidine) for 4 weeks. If the pet has been treated with multiple courses of antibiotics in the past, a culture and susceptibility test is recommended to detect multi-drug resistant staphylococci.
- *Malassezia* yeast dermatitis is usually very pruritic, and there is a greasy, moist, or waxy appearance to the skin, with odor. The pruritus is often nonresponsive to corticosteroids. With chronic disease, there is lichenification and hyperpigmentation to form an appearance of “elephant skin.” The most common areas are the feet, legs, neck, axillary, inguinal, and perineum. If these clinical signs are present, look for yeast by cytology. Remember that the condition is actually a hypersensitivity reaction to the yeast, so the number of yeast may be very small. If there is any doubt, TREAT! For initial treatment, the author prefers oral ketoconazole (or any other oral azole), 5-10 mg/kg/d for 2-4 weeks.
- Remember to treat any otitis externa if present. This is a common secondary problem in many dogs and contributes greatly to the patient's discomfort.
- If staphylococcal or yeast infections are being treated, avoid use of systemic and topical corticosteroids. There are 4 reasons this is important: (1) they may lengthen or alter the course of staphylococcal pyoderma; (2) they don't usually help yeast dermatitis anyway; (3) the anti-inflammatory effect may reduce the severity of lesions, resulting in the

infection looking better, which may cause the owner to stop treatment prematurely; and (4) **a major diagnostic goal early on is to determine if pruritus persists after elimination of secondary infections.** It is acceptable to use antihistamine medications here – they won't interfere with resolution of infection, may cause slight sedation to help the pet sleep, and give some 'psychological' comfort to the owner.

### Step 3. What Remains?

Following this, *the patient's response to infection control* is a valuable clue to the underlying disease and will aid greatly in planning logical diagnostic evaluation for each patient. Here, the clinician will have treated with infection and parasite control ALONE, for 3 to 4 weeks, and observe the clinical response. We can then propose 4 groups of possible underlying causes, depending on response.

- *If the response to infection control is a complete clearing of lesions, yet with substantial remaining pruritus,* (implying the underlying disease is in the "pruritic but not lesional" group) allergic causes such as adverse food reactions or environmental allergy should be strongly considered. Here, application of "Favrot's Criteria" to make a clinical diagnosis of canine atopic dermatitis may apply. If the dog has any five of the following eight criteria present AFTER control of infections and parasites, there is a high likelihood that atopic dermatitis is present: Age of onset < 3 years; Dog lives mostly indoors; Corticosteroid-responsive pruritus; Chronic or recurrent yeast infections; Affected front feet; Affected ear pinnae; Non-affected ear margins; Non-affected dorsal lumbosacral area.
- *If the response to infection control is a partial clearing of the lesions,* but the skin is not completely normal and pruritus remains, this implies that the underlying disease is in the "pruritic and lesional" group. Possible diagnoses to consider include parasitism, adverse food reactions (some of which include lesions), primary seborrhea, and dermatophytosis. Diagnostic steps in this case might include repeated skin scrapings and trichogram, empirical treatment for scabies mites, a hypoallergenic diet trial, and fungal culture. If the underlying cause is not forthcoming, a later step might be skin biopsy. Biopsy can often shed light on the cause of scaling, seborrheic diseases. Along with these differential diagnoses, the possibility of inadequate initial treatment should be considered.
- *If there is little or no clinical response to infection control,* factors such as antibiotic resistance or poor client compliance should be considered. It is also possible that the diagnosis of pyoderma was not correct—non-pyoderma pustular diseases like pemphigus foliaceus may be present. Bacterial culture and susceptibility testing, fungal culture, and skin biopsy might be considered with this response pattern.
- *If the response to infection control is a complete clearing of both lesions and pruritus,* you must think about what went wrong with the skin that made it more susceptible to developing infection. Especially in an older dog, consider underlying systemic disease. In a younger dog, many authors believe that very early allergic disease can first manifest as increased susceptibility to skin infections, without little or no pruritus remaining after infection control. In this event, diagnostic evaluation consists of evaluation for systemic disease with blood and urine analyses, and possible evaluation for allergic disease.

By applying these diagnostic principles to canine pruritus, your chances of reaching the proper diagnosis, and therefore administering the proper treatment, will be greatly enhanced!

## DIAGNOSING AND MANAGING RECURRENT PYODERMA IN DOGS

Douglas J. DeBoer, DVM, Diplomate ACVD

Staphylococcal skin infections can be stubbornly recurrent in some dogs. The client (and the veterinarian) must understand that staphylococcal bacteria are normal flora; infection cannot occur unless something has gone wrong with the skin or its defense systems. Thus, particularly in recurrent infections, the first step is to attempt to define the underlying cause with appropriate diagnostic investigation. In younger dogs with recurrent infections, common causes of recurrence include external parasites and allergic disease. Older animals can also develop recurrent infections from hypothyroidism or any other underlying systemic disease. Despite thorough testing, some patients with recurrent infections defy diagnosis—their infections respond completely to antibiotic treatment, yet continue to recur soon after such treatment is discontinued. For such patients with “idiopathic recurrent pyoderma,” there are several measures that may help to prevent or limit recurrence.

### Important Factors in Pathogenesis

Advanced techniques have allowed a more careful examination of the host factors and bacterial factors that may be important in the pathogenesis of recurrent pyoderma. The first step in infection is *adherence* of the bacterium to the cells or tissue of interest. Recent studies have shown that it is much easier for staphylococci to adhere to canine skin cells than to feline skin cells. Perhaps this helps explain why infection occurs more often in dogs. As another example, the epidermis, as part of its normal defense system, secretes bactericidal peptides called *defensins*. It is now well established in humans that individuals with atopic dermatitis may have decreased production of these substances compared with nonallergic people; this may explain the predisposition to recurrent infection in atopic human patients. These factors are currently being studied in dogs. With regard to bacterial factors, several studies have attempted to find some characteristic of the organism itself (for example, the particular species or strain of *Staphylococcus*) that makes it particularly virulent or prone to cause recurrent infection. So far, these factors have not been uncovered, leading one to speculate that the most important factors may be those associated with the host. There has been a recent increase in reports of multidrug-resistant staphylococcal strains. In particular, the methicillin-resistant staphylococci (MRS) are of concern, and, of course, presence of a highly resistant bacterial strain may complicate treatment.

In some cases of recurrent pyoderma, there are complicating factors. We must consider several forms of pyoderma in which additional factors contribute to the pathogenesis and make treatment difficult. Examples include German Shepherd Dog pyoderma/cellulitis—a special form of deep pyoderma in which there is evidence of a genetically determined cellular immunodeficiency—and interdigital pyoderma, in which, in addition to staphylococcal infection, the deep infection that occurs between the toes is, in part, a foreign-body reaction to hair shafts, perhaps entrapped in scar tissue. Recent evidence suggests that at least some cases of interdigital pyoderma truly begin as cystic structures that become secondarily infected.

### Assessing Patients with Recurrent Pyoderma

From a clinician’s perspective, the main underlying causes of a recurring pyoderma can be divided into 4 groups, depending on the response to antimicrobial treatment. Routine procedures such as skin scrapings for mites, dermatophyte culture, careful history, and physical examination should be conducted first to eliminate common and obvious causes of recurrence. Following this, the patient’s response to antimicrobial treatment is a valuable clue to underlying factors and will aid greatly in planning logical diagnostic evaluation to uncover the predisposing

factors for each patient. The clinician must treat with topical antimicrobials (or, less commonly, antibiotics) ALONE, for 3 to 4 weeks, and observe the clinical response. We can examine the 4 groups of underlying causes more carefully, depending on response.

1. *If the response is a complete clearing of lesions, yet with substantial remaining pruritus*, allergic causes should be strongly considered as underlying causes.
2. *If the response is a partial clearing of the lesions*, but the skin is not totally normal and pruritus remains, underlying factors to consider include inadequate treatment, parasitism, food allergy, primary seborrhea, and dermatophytosis. Diagnostic steps in this case might include repeated skin scrapings, empirical treatment for scabies mites, a hypoallergenic diet trial, fungal culture, and skin biopsy.
3. *If there is little or no clinical response* to antibiotic treatment, factors such as antibiotic resistance or poor client compliance should be considered. It is also possible that the diagnosis is wrong—non-pyoderma pustular diseases like pemphigus foliaceus may be present. Bacterial culture and susceptibility testing, fungal culture, and skin biopsy would be indicated with this response pattern.
4. *If the response is a complete clearing of both lesions and pruritus*, the main underlying factors to consider include systemic disease, very early allergic disease, and idiopathic recurrent superficial pyoderma. In this event, diagnostic evaluation consists of evaluation for systemic disease with blood and urine analyses and possible evaluation for allergic disease. Failing to find a specific cause, the diagnosis of “idiopathic recurrent pyoderma” can be made; several treatment options are available for attempting long-term control and prevention of recurrence.

### **Methicillin Resistant Staphylococci: An Added Complication**

There has been a recent increase in reports of multidrug-resistant staphylococcal strains and MRS in canine pyoderma. In some areas of the United States, more than 50% of skin cultures performed at dermatology specialty practices are MRS. These strains include the methicillin-resistant *Staphylococcus pseudintermedius* species (canine infections, referred to as “MRSP”) or methicillin-resistant *Staphylococcus aureus* species (human infections, referred to as “MRSA” and, fortunately, much less common). Veterinarians should endeavor to use correct terminology when discussing these infections with clients; incorrectly referring to a canine MRSP infection as “MRSA” may be alarming to the client. If laboratory testing indicates the presence of MRS, the isolate will be *clinically resistant to all penicillins and cephalosporins*.

What is the significance of these organisms? First, if you treat a dog with staphylococcal pyoderma with a beta-lactam antibiotic (cephalosporin or penicillin) and there is limited or no response, *culture and susceptibility testing is now mandatory*. Fortunately, most veterinary strains of MRS are still susceptible to routine antibiotics such as trimethoprim-sulfamethoxazole, clindamycin, or a fluoroquinolone such as enrofloxacin or marbofloxacin. However, it is important to note that it is impossible to predict with any certainty which antibiotics are indicated without performing a susceptibility test. Empirical “antibiotic hopping” is hazardous, as with each cycle of treatment, multiple drug resistance becomes more likely.

Second, if you do identify an MRS organism, especially if it appears to be very resistant, *you should order a staph speciation test*. If you have a patient with MRSP (i.e., the canine strain) in your hospital, you need not have the dog under full isolation procedures, but you should isolate the patient to the extent you can and eliminate traffic from this patient to other dogs in the clinic, especially the surgery and critical care areas. If the organism turns out to be a methicillin-resistant, human-origin *S. aureus* (MRSA), the owner should be notified of this fact so they can discuss the situation with their own health care provider, and gloves should be worn when

examining the patient. This patient is a potential human health hazard and should be considered so until all lesions have completely resolved. The concern here is that without proper precautions, the MRSA could colonize the owner, you, your staff, or others. It is important to understand that merely becoming colonized with MRSA is not inherently dangerous. After all, 3% to 5% of people are already colonized at any given moment, and colonization is dynamic and transient. Where the situation becomes potentially dangerous is if the colonized person becomes injured or immunosuppressed.

Third, the emergence of MRS in the veterinary world suggests that we must redouble our efforts to use antibiotics wisely and judiciously and reconsider all efforts to use alternative, nonantibiotic treatments, if possible, in the face of recurrent infections.

### **Reducing Antimicrobial Use and Preventing Infection**

Antimicrobial topicals are the first line of defense with recurrent skin infections. Shampoos containing 2% to 4% chlorhexidine appear especially helpful for preventing new lesion development when used once to twice weekly and allowed to remain on the pet for 5 to 10 minutes before rinsing. Any patient with a history of recurrent pyoderma, even if it is bathed infrequently, should have a chlorhexidine-based routine cleansing shampoo. It seems that a combination shampoo with miconazole can be even more beneficial. The miconazole helps to eliminate any concurrent yeast infection, but also has antimicrobial properties that are synergistic with chlorhexidine. Other ingredients, such as benzoyl peroxide, are also effective but tend to be drying and irritating with prolonged use. Products that are formulated to remain on the skin may have a longer duration of action on the skin than a shampoo and, in many cases, are easier for the owner to apply frequently. For localized areas, treatment with a cream, ointment, or wipe may suffice. For broader regions of the skin, spray-on products, mousse formulations, or “leave-on” conditioner products are recommended. To help prevent relapse of recurrent pyoderma, begin with every-other-day application. If effective, the applications may be tapered down to every 3 or 4 days in many patients. The overall principle here is to limit, to the extent possible, prolonged or repeated courses of antibiotic treatments to minimize the potential for development of antibiotic resistance.

Whether used daily as primary treatment or every few days as preventive maintenance, the following ingredients are the most useful in topical products for staphylococcal pyoderma:

- Mupirocin 2% ointment—applied daily to areas of local infection as a primary treatment; not for prevention.
- Chlorhexidine—spray, mousse, or gel formulation, for treatment or prevention; combination miconazole/chlorhexidine products seem especially good.

Immunomodulatory therapy can be remarkably effective for some patients with idiopathic recurrent superficial pyoderma. Its use for recurrent deep pyoderma, or for recurrent pyoderma associated with allergic disease, is less well studied. In particular, staphylococcal bacterin products are very useful. These “staph vaccines” are either available commercially (SPL, Delmont Laboratories) or are prepared by a local laboratory as autogenous bacterins. They generally must be used long-term to prevent recurrence; however, their use avoids the necessity of prolonged antibiotic treatment in some pets. SPL has a variety of immunomodulatory actions; unfortunately, these have mostly been studied in mouse models or in vitro and rarely in dogs. Recent gene-expression microarray studies in dogs suggest that SPL may exert its effect via up-regulation of interferon-gamma production. SPL is administered at 0.5 cc subcutaneously, twice weekly, for a trial period of 10 weeks. During the first 6 weeks of injections, antibiotics are administered concurrently. After 6 weeks, the antibiotics are stopped and the injections continued. Success is manifested as failure to relapse, much milder relapse,

or infrequent relapse compared with before use of the SPL. If SPL is effective, it can usually be reduced to once-weekly injection, and sometimes once every 2 weeks.

Continuous antibiotic treatment via “pulse therapy” has always been a last-resort treatment for recurrent pyoderma, but with the current resistance situation, it should be avoided at all costs. The emergence of MRS has virtually guaranteed that such treatment will eventually result in colonization by a resistant strain, a phenomenon that is growing worldwide.

## NEW DRUGS ON THE BLOCK

Douglas J. DeBoer, DVM, Diplomate ACVD

What's new in the world of dermatology therapeutics? A number of new treatments have appeared on the market in the past few years, and for others that are not "new," there is new information that may allow you to use them more effectively.

### Oclacitinib

Perhaps the most talked-about drug in recent dermato-history, most clinicians are now familiar with its use. Recent practical experience with oclacitinib (Apoquel) by dermatologists has provided insights as to how best to use the drug. First, two rare adverse effects have been seen, even at the label dose: demodicosis, and slightly lowered WBC count. Apoquel may not work very well on the pruritus associated with skin infection, either staphylococcal or yeast. Therefore, it is important to treat these infections before using Apoquel (or at the same time) because you will not be able to adequately judge response if infection is present. Also, think of Apoquel as an antipruritic drug, useful against allergic itch – and not a drug for "any dog with skin disease." It has no effect on noninflammatory alopecia. It is not a substitute for steroids in autoimmune diseases such as pemphigus or autoimmune hemolytic anemia. Some clinicians have observed that it is not always useful in conditions where there is inflammatory swelling, such as severe otitis externa. Recent information on its off-label use in cats suggests that it can be effective in feline pruritic disease, however owing to its very short half-life in this species, it must be administered at 1.0 mg/kg BID to be reasonably effective. This dose appears safe for a couple of months, at least, but the long-term adverse effects of oclacitinib in cats are completely unknown.

### Lokivetmab

Biological therapies are cutting-edge, unique, exciting, and potentially immensely useful treatments in medicine, with many potential targets, and constantly advancing technology. The first to achieve widespread use is the monoclonal antibody (mAb) lokivetmab (Cytopoint). Lokivetmab is a caninized mAb that binds to canine IL-31, thus achieving pruritus control in canine atopic dermatitis that can last 4 weeks or more after a single injection. It appears to be effective in perhaps 70% of dogs with AD. This author's bias is that this very targeted treatment works best after the "cytokine storm" of severe AD has been quieted by other, less targeted treatments such as corticosteroids. Because Cytopoint is a canine protein, it should NEVER be used in any other species – not would it likely be ineffective, but it would be highly likely to cause an allergic reaction if injected more than once.

### Miscellaneous Antipruritic/Antiallergic Treatments.

Limited success has been achieved in pruritic dogs with palmitoylethanolamide (PEA), gabapentin, or maropitant. Gabapentin and maropitant should be considered "niche" drugs that are not generally useful in itchy dogs, but which may be effective in a limited number of dogs and can be tried as a "last resort." Ultramicronized PEA (Redonyl) seems to have limited antipruritic action on its own, but the most convincing evidence is that it can be medication-sparing, IE it can reduce the amount of other antiallergic medication that is needed for a dog or cat. Because it is safe, relatively inexpensive, and easy to administer (chewable treats), it is gaining popularity as an accessory treatment "supplement" in allergic disease.

A biological treatment on the horizon is use of recombinant allergen molecules in allergen-specific immunotherapy, such as Allerderm-HDM, a recombinant Der f 2 protein in a pullulan

(carbohydrate polymer) base. Though expensive, this treatment requires fewer injections than conventional injection immunotherapy, and may work faster.

### Miscellaneous Drugs of Note

- **Ciclosporin or Chlorambucil** have received some mention in treating recalcitrant indolent ulcer (“rodent ulcer” or “eosinophilic ulcer”) in cats. It seems like a fairly high starting dose of ciclosporin is necessary for efficacy in this feline condition – starting at 10-12 mg/kg once daily for 4 weeks, then gradually tapering. Chlorambucil has been used at 0.1-0.2 mg/kg (usually, equal to 1 mg/cat) every other day for a 2 month trial period. A CBC should be done every 2-4 weeks in cats receiving chlorambucil, as it can be myelosuppressive.
- **Topiramate** has occasionally been used to treat feline idiopathic ulcerative dermatitis (“neck ulcers”). It appears safe when used at 2.5-5 mg/kg PO BID, though overall response rates and long-term toxicity are not known.
- **Deslorelin implants** have gained some favor for treating canine “Alopecia X.”
- **Famciclovir**, used for feline herpesvirus infection, is now recommended at a higher dose than previously - the new dose is 90 mg/kg BID.
- **Mycophenolate mofetil**. This drug, an immunosuppressive agent for autoimmune disease, is being used more commonly in dogs. Typical doses are reported as 15-20 mg/kg BID, but at such doses gastrointestinal adverse effects are common. Lower doses (5 mg/kg BID) may be successful in some dogs, and limit toxicity.

## ANALGESIA FOR THE ILL OR INJURED PATIENT

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Analgesia is an essential component of treatment for many patients presenting for an illness and for all patients presenting with injuries. An injury always causes pain although the degree of pain that is associated with the injury will vary depending on the location and severity of the trauma. The clinician should always make a conscious decision not to give analgesia rather than the other way around.

Analgesia is an essential component of treatment for all surgical patients. Surgery is a form of deliberate trauma. Trauma always causes pain although the degree of pain experienced by the animal will vary depending on the location and severity of the trauma. The clinician should always make a conscious decision not to give analgesia rather than the other way around.

Pain has many detrimental physiologic effects. Pain can negatively impact cardiopulmonary function, metabolism, endocrine status and immune function. Premature ventricular complexes, ventricular tachycardia, tissue hypoxia, atelectasis, hypoventilation (leading to significant acidosis), anorexia, muscle weakness, and delayed tissue healing are all potential sequelae of pain. In addition pain **KILLS**. NO patient is so critical that pain relief cannot be provided. In more seriously ill or injured patients the dose of the drug or drugs being used may need to be decreased; however, analgesics should never be withheld.

### Preemptive Analgesia

Wind-up, or a resetting of the pain threshold that makes the patient more sensitive to pain, occurs when pain occurs before adequate analgesia is provided. Analgesia should always be given as soon as the animal is noted to be painful to avoid further “wind-up”. In situations where a painful procedure is going to be performed (i.e., any type of surgery) analgesics should always be given preemptively to prevent wind-up from occurring.

### Route of Administration of Analgesics

Ideally analgesics should be given intravenously initially to ensure adequate blood levels are achieved as rapidly as possible. The subcutaneous route should be avoided in dehydrated or poorly perfusing animals since uptake of the drug is too unpredictable. Intramuscular injections can be painful and ideally should be used only in the short term or when no other route is available. Opioids along with local anesthetics and  $\alpha$ -2 agonists also can be given via the epidural route (injection or catheter).

### Epidural Analgesia

Epidural analgesia can significantly decrease the dose of systemic medications required. Drugs can be given by injection or via a catheter. Epidurals usually are administered under anesthesia; however, with practice both injections and catheters can be placed under sedation and local anesthesia. Depending on the location of the tip of an epidural catheter effective analgesia can be provided for the pelvic limbs, abdomen or thorax. Usually morphine or hydromorphone are administered; however, many other opioids as well as  $\alpha$ -2 agonists have been used via the epidural route. If anesthesia is desired then local anesthetics are infused in addition to the opioid. The beneficial effects may last as long as 6 to 24 hours. Although multiple side effects including bradycardia, hypotension, respiratory depression, paralysis, hypothermia, urinary retention and infection have been reported, all but bradycardia are rare in the author’s experience. Some of these effects are minimized and paralysis is avoided if local anesthetics

are not infused. Local anesthetics should be avoided or should be used with extreme caution with high epidurals (epidural catheter in thoracic region).

### Opioids

Opioids are the class of drug most commonly given to animals in pain. Pure agonists include meperidine, morphine, methadone, hydromorphone, and fentanyl. Meperidine is a mild analgesic that is very effective but it is also very short acting. Morphine can be used effectively for short-term analgesia (sometimes less than 20 minutes) and is an excellent choice for constant rate infusions for controlling significant pain. It may last as long as 4 hours in animals that are not very painful. It is also very effective when given epidurally. Morphine can cause vasodilation and emesis. Hydromorphone is an intermediate acting opioid that clinically is effective at controlling most pain. It seems to be clinically less effective than oxymorphone with more side effects (emesis, dysphoria). Common side effects include panting (actually hypoventilation) and noise sensitivity. It may last as long as 6 hours in animals that are not very painful but more commonly it needs to be given every 2 to 4 hours and it can also be given as a constant rate infusion. Methadone is an intermediate acting opioid similar to hydromorphone with fewer side effects. Fentanyl is an inexpensive, short acting opioid that must be delivered frequently (every 10 to 20 minutes) or via a constant rate infusion. It is an extremely effective analgesic that is 100 times more potent than morphine.

Long acting fentanyl is available in a transdermal form and a topical formulation. Transdermal fentanyl patches are useful adjuncts in controlling pain. They are rarely effective at completely controlling pain unless the pain is mild; however, the use of patches avoids the episodes of moderate to severe pain that can break through between intermittent injections or oral pain medication administration. Patches can take 12-24 hours to reach peak effect and may last as long as 5 days. Patches should be avoided in very critical patients since the amount of drug the animal can be exposed to can cause severe depression and sedation. If half patches are being used the patch is not cut in half but rather the adhesive is removed from only half of the patch. When placing a patch care should be taken to ensure the animal (or other animals in the household or small children if the patient is being discharged with a patch) cannot eat it. Topical fentanyl is a recently approved formulation (Recuvyra, Elanco) designed to provide 4 days of analgesia.

Butorphanol is an agonist/antagonist. It is a short acting analgesic that clinically is most effective for treating soft tissue pain. It has minimal sedative and respiratory depressant effects. Because of these characteristics it is very useful in very critical patients or in patients who have not been fully cardiovascularly resuscitated. It can also be titrated effectively intraoperatively with minimal cardiorespiratory effects. It has a short duration of action (sometimes as little as 20 minutes) and clinically is not as effective as other opioids so it is less useful for treating significant soft tissue pain and musculoskeletal pain. It may last as long as 4 hours in animals that are not very painful. Given as a constant rate infusion it is very effective at controlling soft tissue pain in cats while avoiding most of the long term side effects that often occur with other opioids in this species.

Buprenorphine is a partial agonist that is effective for mild soft tissue pain. Clinically it also seems to be more effective in cats than in dogs. Because it is a partial agonist it has fewer side effects than the pure agonists. It may take 1 to 3 hours to reach peak effect which makes it an inappropriate drug to use in the acutely painful animal. The duration of effect may be as long as 6 to 12 hours. Because of its high affinity to mu receptors it blocks other pure agonists from binding to the receptors. For this reason caution should be exercised in giving this as a first line

drug in animals that are already experiencing pain since, if it is not effective, the pain may be very difficult to get under control until the drug wears off.

### **Tramadol**

Tramadol is a weak opioid that has questionable efficacy in dogs. A recent study in dogs with osteoarthritis showed no significant difference from baseline in lameness assessment. Two metabolites are produced. The more effective metabolite (o-desmethyltramadol) is produced in limited quantities by dogs. It appears to be produced in higher quantities in cats. The metabolite has 1/10<sup>th</sup> the potency of morphine at the mu receptors. It is also a kappa agonist and inhibits serotonin reuptake. Every 8 hour dosing is recommended in dogs and cats although based on its relatively short half life in dogs dosing every 6 hours may be necessary. It is unlikely to be effective as a sole agent in either species.

Side effects of all opioids can include bradycardia and respiratory depression. These are both uncommon except in critical patients unless high doses were administered. If opioids are required to provide analgesia but respiratory depression becomes evident then positive pressure ventilation may be required. When bradycardia is noted a blood pressure should be checked and an anticholinergic drug (atropine, glycopyrrolate) should be administered only if the animal is concurrently hypotensive or if the bradycardia is associated with a heart block. Routinely treating patients with an anticholinergic should be avoided since tachycardia increases myocardial oxygen demand and may worsen arrhythmias. Opioids are metabolized by the liver and effects may be prolonged in patients with liver disease.

### **Nonsteroidal Antiinflammatory Drugs**

Nonsteroidal antiinflammatory drugs are extremely helpful in managing pain in small animals especially when used in conjunction with opioids. They generally should not be used in patients with underlying renal, hepatic or gastrointestinal disease or those with poor tissue perfusion. They should be avoided in critically ill or injured patients due to their negative gastrointestinal and renal effects. In addition some have negative effects on coagulation. Even the COX-2 specific drugs are not recommended in patients with hypovolemia, compromised gastrointestinal perfusion (related to circulatory disturbances or underlying disease processes), and renal disease.

### **Ketamine**

Constant rate infusions of low dose ketamine given to effect also have been used in painful patients and may be a helpful adjunct. Ketamine can be used in conjunction with morphine, hydromorphone or fentanyl and lidocaine and given as a constant rate infusion. When using this combination the patient should be assessed regularly and the dose titrated to the minimum possible to avoid sedation and anorexia – both are common complications.

### **Gabapentin**

Gabapentin has primarily been used for neuropathic and chronic pain. It has a structure similar to GABA but it does not bind to GABA receptors or affect degradation or uptake of GABA; the mechanism of action is basically unknown. Both research as well as clinical studies in dogs and cats have shown no improvement in pain scores between gabapentin and other drugs such as opioids or non steroidal antiinflammatory drugs. It has been shown to have anxiolytic effects in cats. Absorption may be impacted by the use of H<sub>2</sub> receptor antagonist antacids, but not proton pump inhibitors. Human products containing xylitol should never be used in dogs.

### **Local Anesthetics**

Local anesthetic drugs can be injected into wound edges, onto tissue beds (via splash block or wound diffusion catheter), regionally, intraarticularly, intrapleurally, intercostally, and intraabdominally. They are extremely effective at controlling pain. Lidocaine or bupivacaine or both mixed together in 50:50 volumes can be used. The advantage of using the two drugs combined is that the animal gets the benefit of the rapid onset of action of the lidocaine and the prolonged duration of effect of the bupivacaine. Pain related to the acidic nature can be modified by warming the drug(s) to body temperature or by adding 10% of the volume as sodium bicarbonate. Dilution of lidocaine to a 1% solution can be useful in minimizing total drug dose while provided adequate volume needed for the block.

Local anesthetic agents also can be used intravenously at low dose constant rate infusions to provide additional analgesia. Patients receiving intravenous doses or higher doses of local anesthetic agents should be monitored closely for hypotension and arrhythmias.

### **Analgesia for the Hospitalized Patient**

Opioids should be scheduled but should be given on an “as needed” basis rather than adhering to a schedule per se. It is important to avoid breakthrough in pain control that occurs frequently when intermittent injections are administered. If the patient is very painful or if frequent injections are needed serious consideration should be given to using a constant rate infusion. The patient should be closely monitored and at the first sign of pain further medication should be given. Nurses should be trained to recognize the signs of pain and standing orders should be provided to nurses to allow them to provide analgesia without checking with a doctor except with the more critical patients.

### **Analgesia for the Surgical Patient**

Medication should be given preoperatively to prevent windup and should be continued intraoperatively and postoperatively. If the patient is responding to surgical stimuli and is deemed to be perceiving pain, then analgesics should be provided. Increasing the dose of an inhalant or infusing additional propofol does nothing for the pain except mask it, which can lead to more windup. In addition these drugs may worsen hypoventilation, cardiac output, and hypotension.

### **Analgesia for the Critical Patient**

No patient is too critical to receive analgesia; however, 25 to 50% of the normal dose should be provided initially and the dose titrated upwards based on the patient’s response. Opioids should be given intravenously. In very critical patients milder opioids should be used initially until it is known how the patient will respond to the drug.

References available on request.

## IMPENDING DOOM – RECOGNIZING THE PATIENT IN TROUBLE

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Knowing when patients are getting into trouble is part science and part art. It requires constant contact with the patient, repeat physical examinations and very close monitoring. It also requires a thorough understanding of physiology and pathophysiology. The goal of monitoring and assessing patients is to ensure minor changes in patient status are noted – and corrected – prior to these minor changes becoming major events whose progression can no longer be affected. Because the doctor is rarely capable of spending the amount of time with patients this requires due to other responsibilities, he or she must learn to rely on nurses. Teamwork is essential. Nurses must be well trained in physical examination skills as well as monitoring skills and must feel comfortable relaying information to the doctor. The doctor, in turn must know what to do with that information and be able to respond quickly and efficiently in order to ensure patient morbidity and mortality are minimized. For instance, alterations in certain parameters such as elevations in respiratory rate, heart rate and blood pressure can be associated with organ dysfunction, pain or anxiety. When treating the ill or injured patient the assumption should be that the more serious problem exists and this should be ruled out first. Mistakenly assuming tachycardia was due to anxiety when, in fact, it was an indicator of hypotension, can have life-threatening consequences for the animal.

This seminar will provide an overview of abnormal physical examination and laboratory findings, the presence of which suggests underlying pathology. In the critically ill or injured patient these abnormalities can lead to serious problems if left unnoticed or untreated. Often a trend of change is far more important than absolute numbers. However, the presence of these abnormalities should, at the very least, prompt the clinician and nurse to pay closer attention to the patient and institute more frequent monitoring measures, even if no direct therapeutic interventions are made. Some patients have underlying diseases or injuries where some of the following abnormalities are “normal”. This obviously needs to be taken into account when making clinical decisions; however, good judgment should be exercised when tolerating abnormalities as being acceptable. It is essential to always treat the patient – not the numbers. Early goal directed therapy, especially related to ensuring hemodynamic stability, has been shown to minimize morbidity and mortality in humans and, whenever possible, the goal in veterinary medicine should be the same.

### Level of Consciousness and Central Neurologic Status

Any deterioration in the level of consciousness is an indicator that the patient’s prognosis is likely deteriorating. Stupor (arousable with extremely strong stimulus) or coma (nonarousable no matter how strong the stimulus) indicates a lesion in the cerebrum or reticular activating system. Pupil size, symmetry (equal size in both eyes) and response to light should be assessed in these patients. Reactive pupils are consistent with a lesion in the diencephalon and prognosis is better than if the pupils do not respond. Pupils that are mid range or dilated and non responsive indicate a brain stem lesion. Prognosis in these patients is grave. Other signs suggestive of brain stem lesions include an abnormal respiratory pattern. Normal mentation that progresses to altered mentation generally indicates a progression of the underlying pathology. An acute onset of seizures in a metabolically unstable patient suggests that the underlying metabolic abnormalities (i.e., hypoglycemia) have either not been diagnosed or have not been treated appropriately. A sudden onset of seizures in neurologically abnormal patients, frequent recurrent seizures in hospitalized patients being treated with antiepileptic drugs, or an inability to

control seizures rarely has a positive outcome. Blindness following cardiopulmonary resuscitation is not abnormal and is not a prognostic factor.

### **Airway**

Any increase in audible or auscultable sounds when the patient is breathing can be associated with hypoxia and hypercarbia. Stridor is consistent with an airway that is at least 80% obstructed. It should be assumed that patients that normally have stridor, such as brachycephalic breeds, that present with respiratory difficulty related to their upper airway may be breathing through as little as 5% or less of their normal airway diameter.

### **Breathing**

Respiratory rates less than 18 breaths per minute or greater than 40 breaths per minute (in the animal that is not anxious) suggest an underlying abnormality that may be related to respiratory or cardiovascular pathology or pain. The trachea should be palpated and ausculted. Ventilatory patterns should be closely assessed since any change in pattern usually indicates a problem. Symmetry of chest movement and the presence of any abdominal component to the breathing pattern should be noted. Increased inspiratory or expiratory effort indicates an increased work of breathing that if significant or if progressively worsening can potentially lead to respiratory acidosis, hypoxia or ventilatory failure secondary to exhaustion. Increased expiratory effort usually indicates lower airway disease, often bronchoconstriction. This can be accompanied by wheezes. A restrictive pattern (inability to expand the chest wall) suggests pleural effusion or pneumothorax. These patients usually require an immediate thoracentesis. Paradoxical abdominal movement (the abdomen moves in during inspiration instead of out) usually indicates a severe condition and a patient that may be close to ventilatory failure.

The lungs should be ausculted in at least 4 fields and should always be ausculted prior to ausculting the heart since once the ears have attuned themselves to louder sounds the more subtle sounds can be missed. Evidence of absent or diminished lung sounds, harsh bronchovesicular sounds in an animal that is breathing quietly, crackles or wheezes all suggest pathology. Appropriate thoracic auscultation ideally requires that the patient be in a quiet room; however, this is not possible in most veterinary hospitals. The use of electronic stethoscopes will help minimize the likelihood of missing abnormalities in a noisy environment. Chest percussion in at least 4 quadrants will help detect areas of dullness suggesting pulmonary contusions, hollow sounds indicating a pneumothorax, or a fluid line indicating a hemothorax.

### **Objective Assessment of Airway and Breathing**

Blood gases are indicated in every patient with evidence of stridor, increased audible or auscultable airway sounds, increased inspiratory or expiratory effort or an abnormal ventilatory pattern. Pulse oximetry and capnometry are notoriously inaccurate in awake, nonintubated patients. Venous blood gases can be used for assessing carbon dioxide tension if perfusion is normal but arterial blood gases must be used to assess oxygenation. As with other tests blood gases must be interpreted in light of the underlying disease. A  $PCO_2$  of 36 mm Hg in an animal that has an increased respiratory rate and effort secondary to pulmonary contusions suggests significant pathology. An arterial  $PO_2$  of less than 80 mm Hg at sea level should prompt the clinician to reassess the patient for causes of hypoxemia. Pulse oximetry is notoriously inaccurate except when clips are placed on the tongue. Readings should be interpreted in light of the pulse signal indicated on the oximeter and the patient's mucous membrane colour. Normal oxygen saturation is greater than 97% when breathing room air. A pulse oximetry reading of less than 94% indicates the need for oxygen supplementation. A reading of less than 92% when the patient is receiving supplemental oxygen typically indicates significant hypoxemia

(insufficient oxygen in the arterial blood) and the need for a higher fraction of inspired oxygen ( $\text{FiO}_2$ ) or ventilatory support if the patient is receiving 100% oxygen.

### **Circulation**

Mucous membrane color and capillary refill time should be recorded. Colour should be assessed using a direct light source since overhead fluorescent lights can make pink membranes look pale or muddy. Pale membranes may be due to anemia or vasoconstriction. A delayed capillary refill time (> 2 seconds) indicates decreased perfusion, whereas a rapid capillary refill time (< 1 second) indicates a hyperdynamic state.

Heart rates greater than 140 beats per minute in the dog (no matter what the size of the dog) or 200 beats per minute in the cat are abnormal and can suggest anxiety, pain or hypovolemia. Heart rates less than 60 beats per minute in the dog and 160 beats per minute in the cat suggest a serious condition although high resting vagal tone or sedation in larger dogs can lead to 'normal' heart rates in the 50's.

Pulses should be palpated centrally as well as peripherally. The quality of the dorsal metatarsal pulse is a much more useful physical exam parameter than the femoral pulse. Changes in pulse quality or the presence of pulse deficits should alert the clinician to possible changes in the patient's hemodynamic status. Estimating blood pressure from pulse palpation is exactly that – an estimate – and blood pressure always should be measured in ill or injured patients. Normal blood pressure in the cat and dog is approximately 120/80 +/-10 mm Hg although these numbers will vary somewhat depending on the technology being used. Systolic pressures less than 100 mm Hg or greater than 150 mm Hg typically require a search for the underlying pathology and treatment. Diastolic pressures less than 60 mm Hg typically are associated with hypovolemia, or less commonly excessive vasodilation, and can be associated with poor tissue perfusion. If a Doppler ultrasonic flow detector is being used the flow sounds should be strong. Poor flow sounds may indicate poor perfusion of the area being interrogated.

Venous volume should be estimated by clipping the jugular veins and checking distention. Flat jugular veins that cannot be raised indicate severe hypovolemia. The presence of jugular distention in trauma patients in shock is most likely an indicator of increased intrathoracic pressure or venous obstruction. In the previously healthy animal this may indicate a pneumothorax or pericardial tamponade. If the animal has underlying heart disease jugular distention can be associated with right heart failure.

An electrocardiogram ideally should be assessed in any seriously ill or injured patient. The primary value of an electrocardiogram is in detecting the presence of an arrhythmias; however, the presence of tall T waves (greater than one-fourth the R wave amplitude) is an indication of myocardial hypoxia, commonly related to hypovolemia and/or anemia. Left untreated this will often progress to premature ventricular contractions. Any arrhythmia should be considered abnormal and must be aggressively investigated to ensure it is not affecting perfusion. Ventricular premature contractions are commonly associated with myocardial hypoxia and splenic disease and always should be monitored when either of these conditions might be present. An accelerated idioventricular rhythm (constant premature ventricular contractions but a heart rate that does not exceed 160 beats per minute) may not need treatment but must be closely monitored for signs of tachycardia, multiform complexes, or R-on-T phenomenon, which, in general, will require prompt treatment since these frequently affect perfusion and can be life-threatening.

Capnometry can be used as an indirect assessment of pulmonary blood flow. End-tidal carbon dioxide measurements less than 20 mm Hg typically indicate low pulmonary blood flow unless the patient is being hyperventilated deliberately. Measurements in the 15-18 mm Hg range indicate impending cardiac arrest.

Toe web temperature should be measured and compared with the rectal temperature in patients with altered peripheral perfusion. A difference of greater than 3.5 degrees Celsius is strongly suggestive of poor peripheral circulation.

### Temperature

Hyperthermia in the face of infection is a protective mechanism and it may not be appropriate to attempt to reduce the fever; however, temperatures greater than 40C should be monitored closely because increases may require intervention. Patients with heatstroke or pyrexia due to seizure activity should have the temperatures lowered to a normal range as soon as possible. Temperatures less than 37C typically require supplemental heat and close monitoring. There are two possible exceptions. Animals receiving parenteral narcotics may have slightly subnormal temperatures that usually do not require treatment. Hypothermia in cats frequently is an indicator of poor perfusion and the cause of the poor perfusion should be addressed since providing supplemental heat can cause these patients to pant and potentially worsen their condition since their core temperature may be normal.

Toe web temperature should be taken in all patients in shock or with suspected perfusion abnormalities. The thermometer is placed in between the digital and the metacarpal or metatarsal pad. A difference of greater than 3.5 degrees Celsius between rectal and toe web temperature ( $\Delta T$ ) implies decreased perfusion to the periphery, assuming normal environmental temperature.

### Renal Function

Normal urine output is typically considered to be 0.5 ml/kg/hr or greater in cats and 1 ml/kg/hr or greater in dogs. Values lower than this may indicate dehydration, hypotension to the point renal perfusion is being affected, or renal dysfunction. Oliguria is usually defined as less than 0.3 ml/kg/hr.

Patients that are hydrated typically urinate every 6-8 hours; those on intravenous fluids every 4 to 6 hours. A patient that has not urinated in over 12 hours should have hydration status and blood pressure assessed. If these are both adequate then renal and urinary tract function should be evaluated. A minimum of approximately 0.5 ml/kg/hr of urine should be produced; if the animal is receiving intravenous fluids at a maintenance rate the urine production is usually 1-2 ml/kg body weight per hour. Patients who have difficulty concentrating their urine should produce much higher volumes. Patients with problems such as isosthenuria, polyuric renal failure or postobstructive diuresis may produce 6 ml/kg/hr of urine or more.

Proteinuria (urine protein:creatinine ratio > 1) indicates glomerular dysfunction in a patient that does not have a urinary tract infection. Casts in the urine indicate renal tubular cell hypoxia or ischemia.

### Gastrointestinal Function

The abdomen should be auscultated for bowel sounds and percussed for areas of dullness and tympany. Lack of gut sounds suggests lack of gastric and/or colonic motility. Increased gut sounds especially when an air-fluid interface is frequently ausculted suggests enteritis or enterocolitis. The abdomen should be palpated thoroughly but gently as organs may be

bleeding and additional pressure could cause them to rupture. The caudal abdomen should be carefully palpated for the presence of a urinary bladder.

Blood in the vomit or feces suggests a breakdown of the gastrointestinal barrier. A rectal exam should be performed. This can be related to ulceration (stress, medication-induced, underlying pathology), infection or poor perfusion. Treatment should be directed to any underlying disease process but always to ensuring perfusion to the gut is maximized. Enteral feeding plays a vital role in maintaining the health of the gut mucosa.

### **Musculoskeletal and Skin**

Fractures may worsen with movement and if the animal is recumbent only spinal reflexes and the presence of limb sensation should be assessed until radiographs have been taken. In the severely traumatized patient it should be assumed that fractures are present until proven otherwise and ideally the patient should be restrained to prevent further injury. The most effective method of restraint is to tape the animal to a board with duct tape. Backboards made from Plexiglas are useful because not only are they sturdy but the animal can be visualized on all sides, and radiographs can be taken without having to remove the animal from the board. The entire body should be palpated gently for fractures, swelling, and wounds. The skin should be checked for wounds, petechia, bruises or other abnormalities. Wounds should be covered with sterile saline and a sterile water soluble lubricant prior to being clipped. This prevents further contamination and tissue desiccation, and may help avoid healing complications. After clipping the wounds a sterile dressing should be applied. Fractures of the distal limbs (below the elbow and stifle) should be stabilized following assessment of the injury. Newspaper or bubblewrap splints are placed easily and rapidly and are very effective as temporary splints. Survey radiographs can be taken through both.

### **Laboratory Parameters**

Laboratory parameters that ideally should be evaluated rapidly in ill or injured patients include hematocrit, total solids, electrolytes, glucose, creatinine and venous blood gas values. In more critical patients blood work also should include albumin, coagulation tests, platelet numbers and blood smears to evaluate red and white cell morphology. Packed cell volumes of 27-30% or higher in critical patients are preferred since levels below this may be associated with inadequate oxygen delivery to the tissues. Albumin concentrations that drop acutely to below 20 g/L are usually associated with inadequate oncotic pressure and secondary hypovolemia. In addition to its role in colloid osmotic pressure, albumin plays a vital role as a drug carrier and scavenger of reactive oxygen species. Total solids rarely correlate with albumin levels in sick patients and are not a good assessment of the albumin concentration. Sodium concentrations more than 5 mEq/L above or below the ranges of normal can contribute to muscle weakness and mentation abnormalities. Potassium concentrations less than 3 mEq/L can lead to muscle weakness and when greater than 6 mEq/L can lead to arrhythmias as well as weakness. Glucose is an essential energy source and must be maintained within normal limits in sick patients.

Venous blood gases can be used to assess ventilation status (see above) but more importantly they can be used to assess the presence of metabolic abnormalities. A metabolic acidosis almost always indicates poor perfusion which, in the hospitalized patient, is often a result of inadequate or inappropriate fluid therapy. In simplistic terms a metabolic acidosis should be equated with a global tissue oxygen debt. The longer this debt persists the higher the likelihood of complications and the longer the patient is likely to be in hospital.

A coagulopathic state is not uncommon in the critically ill or injured patient. It can result from loss of clotting factors, hemodilution or excessive consumption. Coagulation tests, which typically do not prolong until clotting factors are only approximately 30% of normal, usually prolong before the problem becomes clinically apparent; it is far easier to correct the problem at this stage. The activated partial thromboplastin time (aPTT) usually prolongs before the prothrombin time unless the patient has ingested an anticoagulant rodenticide, has liver failure or is suffering from rattlesnake envenomation in which the case the reverse usually is true. Minor prolongations in the aPTT should be interpreted in light of platelet numbers as well as the underlying disease and treated accordingly.

White cells should be evaluated for numbers and morphology. Leukopenia in the face of an inflammatory or infectious disease is not a good sign, nor is a degenerative left shift. Red cell fragmentation can indicate oxidant-induced injury (which can lead to a significant hemolytic crisis in cats) or early disseminated intravascular coagulation.

### **Recording the Results**

Results of the primary survey and all measured parameters should be recorded. If necessary, the doctor in charge should be notified when important changes are observed. A strip of 2" tape, stuck to scrub pants on the front on the thigh is a handy "note pad" when used with a good pen.

It is highly recommended that a flow sheet be used for the recording of important monitored data. All monitored I's (inputs) such as drugs, fluids, nutritional products, and all O's (outputs) such as urine, fluid from a nasogastric suction tube, chest tube aspirates are recorded. Masking tape can be used as strips along the side of the bottles of fluids and chest bottles etc., to mark levels at various times. The flow sheet can also be used for the entry of nurse's notes, monitored physical findings and for the recording (by check off of boxes) of treatments required.

IF ITS NOT RECORDED...ITS NOT DONE. This motto is key to the provision of quality, safe, conscientious patient care.

References available on request.

## ENDOCRINE EMERGENCIES - HYPOADRENOCORTICISM

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Hypoadrenocorticism – or Addison’s disease - is a potentially life-threatening condition typically characterized by a glucocorticoid and mineralocorticoid insufficiency. The glucocorticoid deficiency leads to a hypocortisolemia. Cortisol plays an important role in nutrient metabolism, production of catecholamines, regulation of immune function and stabilization of cell membranes. The mineralocorticoid deficiency leads to a hyponatremia and subsequent hypovolemia and hyperkalemia by directly affecting the renal collecting duct’s ability to resorb sodium and excrete potassium.

The cause of this disease is unknown although it is suspected to be most commonly due to an immune-mediated destruction of the adrenal glands. Other causes include neoplasia and hemorrhage. This disease is the “great pretender” in that it can mimic many other diseases, making it difficult sometimes to diagnose.

### History and Physical Exam

These patients often have a vague history of lethargy, weakness, vomiting and diarrhea (with or without blood) and “not doing well”; however, they can also present with no history of signs other than those of the current illness. Patients with a mineralocorticoid deficiency may also have a history of polyuria and polydipsia. Patients presenting on emergency usually have a history of significant gastrointestinal disturbance along with weakness. On physical examination they are usually dehydrated, hypovolemic, hypotensive, bradycardic and hypothermic which is consistent with both a glucocorticoid and mineralocorticoid deficiency. Patients can present appearing relatively stable but may also be close to death at the time of the initial examination.

### Diagnosis and Data Base

The definitive diagnosis of hypoadrenocorticism is based on lack of cortisol elevation in response to an adrenocorticotrophic hormone (ACTH) stimulation test. If in-house testing of cortisol levels is available this can be used for preliminary screening. A patient with a baseline cortisol <2 mcg/dl should have an ACTH stimulation test performed as hypoadrenocorticism is likely. Results between 2 mcg/dl and 6 mcg/dl are considered inconclusive and also should undergo further testing. The patient with a cortisol above 6 mcg/dl is unlikely to have hypoadrenocorticism. A sodium: potassium ratio of less than 27: 1 is suggestive of hypoadrenocorticism although these electrolyte abnormalities can also be present with primary gastrointestinal disease and renal insufficiency. These abnormalities may not be present if the patient only has a glucocorticoid deficiency. Hypoglycemia is often present although patients can be hyperglycemic. Hyperphosphatemia and azotemia may also be present and are usually prerenal in origin due to the hypovolemia. Urine specific gravity is usually low. Hypercalcemia is reported but rare in the author’s experience. The CBC usually reveals the lack of a stress leukogram with the lack of a stress leukocytosis, neutropenia, lymphocytosis and eosinophilia. Anemia may be present.

A lead II electrocardiogram is indicated in every patient with significant hyperkalemia, severe weakness or an auscultable arrhythmia. There are distinct electrocardiographic findings associated with hyperkalemia, although, the order with which they appear is somewhat inconsistent. The abnormalities (in the order they typically appear) include tall or peaked T waves, a prolonged PR interval, an absent P wave, a prolonged QRS complex, bradycardia,

atrial standstill, sine wave complexes, ventricular fibrillation and complete standstill. By the time a sine wave is evident the patient is in imminent danger of dying.

The initial data base should consist of a minimum of complete blood cell count, a chemistry panel, urinalysis, blood gas, and a cortisol. Additional tests such as fecal analysis, coagulation panel, radiographs, abdominal ultrasound and additional cultures may be indicated based on other presenting signs.

### **Treatment**

The goals of treatment are to restore normal electrolytes and normal cortisol levels, to restore perfusion, and treat any underlying additional illness. Oxygen should be provided to patients in shock. Intravenous crystalloid fluids are required and in large volumes to replace the sodium deficit. Normal saline (0.9% saline) typically is infused at high rates although infusion of a buffered electrolyte solution is also a reasonable alternative. Rates should be adjusted based on blood pressure, estimation of central venous pressure (venous volume) and urine output. Depending on the severity of the hyponatremia it may be more appropriate to use a fluid containing less sodium. If a severe hyponatremia is corrected too rapidly ( $> 0.5$  mEq/L/hr) central pontine myelinolysis may result. Synthetic colloids or plasma may be required if the patient is hypoalbuminemic. Transfusion of blood products may be indicated in patients with significant blood loss into the gut.

If the hyperkalemia is significant enough to cause cardiotoxicity then treatment should be instituted. The three primary treatments include infusion of insulin and dextrose, sodium bicarbonate and calcium gluconate. Infusion of 0.5 to 1.5 ml/kg of 10% calcium gluconate has an immediate effect on the muscle because it decreases the membrane potential but will not actually impact potassium levels. Sodium bicarbonate given at a dose of 1 to 2 mEq/kg will cause an immediate transcellular shift of potassium, which once again decreases the resting membrane potential. This treatment can lead to the undesirable side effect of a paradoxical intracellular acidosis. Regular insulin can be injected at a dose of 0.5 U/kg IV which will drive the potassium intracellularly. The insulin injection should be followed by an injection of 2 g of 25% dextrose for every unit of insulin that was given to avoid iatrogenic hypoglycemia. A constant rate infusion of dextrose may be required for a short period of time following the insulin injection and the patient's glucose should be monitored. It is likely that the infusion of dextrose alone should be sufficient to stimulate the release of endogenous insulin, with the same outcome. The advantage of using dextrose alone is that ongoing glucose monitoring is not required. The insulin/dextrose treatment is an effective one without detrimental side effects but may require up to 20 minutes to achieve peak effect.

Dextrose should be administered to patients that are hypoglycemic. A dextrose infusion may be necessary until cortisol levels improve.

Dexamethasone sodium phosphate should be injected intravenously if an Addisonian crisis is suspected. This drug will not interfere with an ACTH stimulation test because the cortisol assay does not detect dexamethasone. It does not have any mineralocorticoid properties so it will not positively impact the sodium balance but it will positively impact the cortisol deficiency. Dexamethasone can cause significant gastric ulceration even after a single dose; therefore, if the patient is not in extremis and already has evidence of hematemesis or melena it may be appropriate to wait until the results of the ACTH stimulation test are available before administering a corticosteroid. Once the ACTH test has been completed hydrocortisone or a prednisone formulation is indicated in order to provide mineralocorticoid activity. Once the diagnosis is confirmed desoxycorticosterone pivalate is indicated. If this is not available then

fludrocortisone should be administered. This is only available as an oral formulation so it often is not tolerated well in the acute crisis situation.

Additional treatment should be instituted as indicated such as gastric protectants, gastric motility modifiers, analgesia as well as treatment of any other concurrent abnormalities.

### **Atypical Hypoadrenocorticism**

Diagnosis of hypoadrenocorticism is often suspected based on electrolyte abnormalities; however, atypical hypoadrenocorticism (AH) is associated with low cortisol levels, normal potassium values and normal or slightly low sodium values. The disease has been well reported in dogs and may comprise up to 45% of Addisonians. It is reported in cats but is a rare disease. It is likely under recognized which can lead to significant morbidity.

Clinical signs in patients with glucocorticoid deficiency are similar to those with both glucocorticoid and mineralocorticoid deficiency. The signs are often non-specific and include lethargy, weakness, anorexia, vomiting and weight loss. Atypical hypoadrenocorticism should be suspected in any sick patient that is not responding to treatment especially with a chronic waxing and waning disease or in any patient that seems more critical than would be expected. Sick patients that do not have a stress leukogram (lymphopenia, eosinopenia) should be suspected of having atypical hypoadrenocorticism. A cutoff of 3000 lymphocytes has been suggested. The definitive diagnosis of hypoadrenocorticism once again is based on lack of cortisol elevation in response to an ACTH stimulation test.

References available on request.

## PARVOVIRUS

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Parovirus is a virus that requires rapidly dividing cells in order to be able to replicate. It originates in lymphoid tissue (oropharynx, thymus, mesenteric lymph nodes) after oronasal exposure to infected feces. Marked viremia occurs within 1-5 days following infection. The virus then infects the rapidly dividing cells of the bone marrow where it destroys white blood cell precursors and the germinal epithelium of the intestines where it leads to necrosis of the intestinal villi. It can affect any age of dog; however, it is typically associated with puppies less than 6 months of age or dogs with inadequate vaccination. It can affect any breed of dog but certain breeds have an increased predisposition. Left untreated it has a high mortality rate but with aggressive treatment outcomes are typically good.

Typically there is a 2-3 day history that starts with anorexia followed by vomiting and then diarrhea that rapidly becomes hemorrhagic. Because of the lack of oral intake and significant losses through vomiting and diarrhea the dogs are always dehydrated and often hypovolemic. These patients are by often septic by definition. The major homeostatic alterations in sepsis include vasodilation, increased capillary permeability, depressed cardiac function, altered hepatic protein synthesis, immune system activation and coagulation abnormalities. This leads to secondary effects such as hypotension, neurologic abnormalities, cardiac failure, acute respiratory distress syndrome, liver failure, renal failure, gastrointestinal barrier breakdown, renal failure as well and disseminated intravascular coagulation. All of these complications need to be considered in this disease.

The virus is very hardy in the environment and can persist for weeks to months and owners should be advised that their home and yard are likely contaminated. Not all disinfectants will kill the virus but dilute bleach is very effective. Whenever parvovirus is suspected the animal should be escorted directly to an isolation unit (ideally through a separate entrance than the main one of the hospital) and personnel should wear gowns and gloves prior to handling the dog. A foot bath should be placed at the entry to the isolation unit.

### Diagnostic Tests

The diagnosis is often made presumptively based on history and age of the animal. Presenting status can vary from stable to being close to death from septic shock. On presentation a primary survey examination (evaluation of level of consciousness, airway, breathing, and circulation) should be completed within 30 to 60 seconds. Abnormalities should be treated as indicated. Depending on the severity of the patient's condition resuscitation may need to be instituted prior to performing a complete physical exam. A very brief history is obtained at this time if possible; however, resuscitation should not be delayed in the critical patient while a complete history is obtained. Instead permission should be rapidly obtained from the owner to allow treatment to be started.

A secondary survey, or complete physical examination, is completed once the primary survey is completed and resuscitation is instituted as indicated. Vital signs are taken at this time. The jugular vein should be clipped and evaluated for distention since this will provide a crude estimate of central venous pressure. Patients may have concurrent pneumonia (aspiration or secondary infection) and close attention should be paid to the ventilatory pattern, presence of cough, and bilateral auscultation of the thorax. The abdomen should be palpated and ausculted.

Auscultation should precede palpation since palpation can cause gut sounds to diminish. The abdomen may be distended as loops of bowel are usually fluid-filled. Abdominal pain is almost always present but can vary in its severity. A rectal exam should be performed and the following should be evaluated: consistency of stool, evidence of blood, and dilation of the rectum. In the author's experience, rectal dilation in puppies with hemorrhagic diarrhea is almost always parvovirus enteritis.

Blood pressure should be considered one of the 4 vital signs and should be assessed as part of the initial exam since fluid resuscitation will depend to a large extent on the patient's blood pressure. A Doppler ultrasonic blood flow detector or an oscillometric device can be used; however, the Doppler is preferred since it allows the clinician to evaluate perfusion or flow as well as blood pressure. Ideally a separate monitor is used in the isolation unit. If this is not an option the unit must be disinfected.

Diagnostic tests are required frequently in order to determine the extent of the disease and to confirm the diagnosis. Resuscitation of the critical patient should not be delayed while tests are being performed unless those tests are required to guide resuscitation. Blood tests including packed cell volume, total solids, and glucose should be part of a STAT database. Many of these puppies are hypoglycemic and require a bolus of dextrose followed immediately by dextrose supplementation in the fluids. Ideally a creatinine, electrolytes and a blood gas also should be assessed as part of a STAT data base. A complete blood count with microscopic evaluation of a blood smear for the differential is essential as leukopenia is associated with a more guarded prognosis. A lymphopenia of <1000/ul within 48h of admission was found in one study to be a negative prognostic indicator. A complete chemistry profile is also indicated. Close attention should be paid to the albumin as well as other liver function tests since some of these puppies have other congenital diseases. Coagulation parameters (prothrombin time, activated partial thromboplastin time) are indicated in severe sepsis and in those with significant hypoalbuminemia.

Feces should be evaluated. The in-house fecal ELISA test for parvovirus is accurate with a high specificity but a lower sensitivity. This can lead to false negative results early in the infection. Concurrent parasitic infections are not uncommon including worms and coccidia.

Abdominal radiographs are indicated; however, care should be taken to ensure minimal contamination of the radiology room. Chest radiographs are indicated if pneumonia is suspected.

### **Treatment**

The goal of resuscitation is to reverse the signs of shock and provide sufficient oxygen delivery to the cells. Resuscitative efforts should be aimed at maximizing hemoglobin levels (oxygen-carrying capacity), blood volume and cardiac function. Patients presenting with signs of shock should have oxygen administered via flow-by. This can be followed with nasal oxygen supplementation if longer-term support is indicated.

One or two large bore peripheral intravenous catheters should be placed. Ideally a central line should be placed to allow for easy blood sampling. In some situations measurement of central venous pressure may be useful in guiding treatment although it may not be appropriate to place this until after resuscitation has been started. Resuscitation will often require a combination of both crystalloids and colloids. Colloids frequently are required due to the low oncotic pressure associated with the hypoalbuminemia. Buffered solutions such as Normosol-R, or Plasmalyte-A should be administered. Lactated Ringer's solution is less preferable due to its low sodium

content. These patients are often acidotic and administration of saline, which has a pH of 5.4, should be avoided.

Crystalloids rapidly redistribute to the interstitial space and only approximately 20% is left in the vascular space after 1 hour. Crystalloids should be considered interstitial rehydrators and not intravascular volume expanders. Infusion of excessive volumes of crystalloids may lead to tissue edema and a barrier to oxygen diffusion. Synthetic colloids such as tetrastarch, or pentastarch, should be considered in any patient showing signs of hypovolemia. Colloids are large molecular weight compounds that are not capable of diffusing across intact membranes and are effective intravascular volume expanders.

Fluids should be infused to achieve or maintain a systolic blood pressure of 100-120 mm Hg, a diastolic blood pressure of 60-80 mm Hg and a heart rate that is in a normal to high normal range. This goal should be reached in a timely manner; ideally within an hour. If patients do not respond to infusion of fluids and volume is assessed to be adequate then a norepinephrine infusion may be indicated. Fresh frozen plasma should be administered with a goal of maintaining an albumin greater than 20 g/L and to provide clotting factors to any patient with a coagulopathy. Although administration of plasma to achieve an albumin of at least 20 g/l is not a reasonable clinical decision in larger dogs it is often feasible in puppies. Albumin also has other beneficial biologic properties including helping to maintain the glycocalyx layer and scavenging of reactive oxygen species.

Parvovirus is associated with significant third-spacing of fluids and ongoing losses of albumin into the gut until the infection has started to subside. Constant rate infusions of synthetic colloids may be needed for several days. Maintenance rates of crystalloid fluids in puppies should be estimated at twice the adult rate. Rates of crystalloid infusion should be decreased based on the infusion of colloids but must be closely monitored to ensure the fluid requirement is not being underestimated.

Electrolyte abnormalities should be corrected based on blood test results. Potassium supplementation is almost always required. Hypomagnesemia should be suspected in the face of intractable hypokalemia.

Patients may present with hypothermia. Or they may become hypothermic during resuscitation secondary to intravascular infusion of large volumes of room temperature fluids. Hypothermia interferes with normal metabolic functions leading to vasodilation, cardiac dysfunction, and interference with the coagulation cascade. Core rewarming should be instituted since peripheral rewarming may lead to worsening of the vasodilation and subsequent worsening of the hypothermia. Artificial warming devices should be insulated from the patient since they can cause burns. Means of rewarming patients includes the use of warm water bottles, warm water circulating blankets, oat bags, warm blankets, and hot air circulating devices. Fluids should be infused at 40C in the hypothermic patient.

A nasogastric tube should be placed for gastric decompression and initiation of early enteral feeding. Gastric distention with fluid is one of the reasons patients with parvovirus vomit. Active gastric decompression is a very effective 'antiemetic'. The tube should be suctioned every hour until volumes decrease then performed every 2-6 hours based on the patient. Early enteral feeding has been associated with a faster resolution of clinical signs. Initially clear liquids with electrolytes and dextrose should be infused. Once it is established that the animal is tolerating this can be switched to a liquid enteral product. Microenteral nutrition (0.1 to 0.25 ml/kg/hr of an electrolyte solution with dextrose) is often tolerated when full enteral nutrition is not. The

addition of a constant rate infusion of metoclopramide also appears to help improve motility in certain patients with upper gastrointestinal dysmotility. Partial parenteral nutrition using an amino acid solution and dextrose or glycerol (Procalamine®) can be used to supplement enteral feeding. This can be administered via a peripheral catheter.

Antiemetics are always indicated. Maropitant is an effective antiemetic with the additional benefit of providing some visceral analgesia. Serotonin antagonists such as ondansetron and dolasetron are useful alternatives and sometimes need to be used in addition to maropitant.

Intravenous broad spectrum antibiotics should be used to 'cover' the range of pathogens that could be contributing to endotoxin and exotoxin effects in the seriously ill patient. Often the number of classes of antibiotics administered is based empirically on the severity of the illness as well as the animal's white blood cell count. Antiparasitics may be indicated and it may be necessary to administer an oral deworming agent in the face of vomiting.

Analgesia should be provided to all animals with evidence of abdominal pain – even if it is mild. Non steroidal antiinflammatory drugs should be avoided. Opioids should be administered intravenously until it is evident that the dog has a functional gut at which time the dog can be switched to oral drugs such as tramadol and gabapentin.

Milder cases of parvovirus enteritis may be able to be treated as outpatients. Survival rates have been reported at 75-80%. Treatment involves once or twice daily injections of subcutaneous fluids, oral dextrose, oral potassium, antiemetics, antibiotics and syringe feeding. The primary reason for proposing this course of action was financial; however, this may actually be more costly in many hospitals. It may; however; be useful in situations where 24 hour hospitalization is not an option.

### **Monitoring**

The Rule of 20 is a checklist developed by Dr. Rebecca Kirby. It was designed to ensure that all vital patient parameters were addressed and that nothing was forgotten. It requires that the clinician evaluate, assess and formulate a plan for each of the items. The 20 items are: 1) fluid balance, 2) oncotic pull, 3) glucose, 4) electrolytes and acid-base balance, 5) oxygenation and ventilation, 6) mentation, 7) blood pressure, 8) heart rate, rhythm and contractility, 9) albumin, 10) coagulation, 11) red blood cell/hemoglobin concentration, 12) renal function, 13) immune status, antibiotic dosage and selection, white blood cell count, 14) gastrointestinal motility and mucosal integrity, 15) drug dosages and metabolism, 16) nutrition, 17) pain control, 18) nursing care and mobilization, 19) wound care/bandage change, 20) tender loving care.

Specific to parvovirus enteritis, the abdomen should be palpated every 6-8 hours to ensure abdominal pain is being controlled but also to ensure the animal is not developing an intussusception. Gut sounds should be auscultated at least twice daily. The animal should not be fed solid food if gut sounds are not auscultated.

References available on request.

## HE CAN'T PEE – MANAGING THE BLOCKED CAT

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Feline urethral obstruction can be caused by crystalline plugs or calculi. Almost all feline urethral obstructions can be relieved using retropulsion and flushing; however, on rare occasion this will not work necessitating temporary decompression via cystocentesis or possibly emergency surgery. Trauma and neoplasia can also cause obstructions; however, these patients will generally require surgical management and will not be discussed in this lecture.

Diagnosis of urethral obstruction is usually based on history and a physical examination finding of a large bladder than cannot be expressed. Cats, if diagnosed early, may have a small bladder, but one that is very turgid when palpated. A painful turgid bladder on palpation is a consistent finding in the obstructed cat. The tip of the urethra of the cat is usually discoloured and occasionally the obstruction can be seen near the tip. An inability to express the bladder does not always mean the cat is obstructed. In obese cats bladder palpation can be problematic.

Every cat suspected of being obstructed should be triaged to ready area of the hospital and the bladder should be palpated. This can be done by a skilled nurse or a doctor. If there is any concern the cat is obstructed the doctor should examine the cat immediately.

In moribund cats an attempt should be made to relieve the obstruction immediately without additional sedation or anesthesia using a 22-24 gauge over the needle catheter with the stylet removed. Using a 3 cc syringe distal urethral obstructions often can be relieved. Ideally an intravenous catheter should be in place and hyperkalemia should be treated prior to performing this procedure but on occasion immediate relief of the obstruction will be lifesaving.

If the patient is more stable ideally diagnostic tests should be performed prior to sedating or anesthetizing the animal. Laboratory diagnostic tests include a hematocrit and total solids, electrolytes, glucose, blood gas and renal parameters. A baseline blood pressure is indicated along with an electrocardiogram if the cat is bradycardic, an arrhythmia is ausculted or the cat is diagnosed with a significant hyperkalemia. A urinalysis can be collected following relief of the obstruction.

Flow-by oxygen should be provided to critical patients. A peripheral catheter should be placed and the patient should be started on a balanced electrolyte solution. Perfusion deficits should be corrected using a combination of crystalloids and colloids, although crystalloids are usually adequate. Once perfusion deficits have been corrected additional fluid therapy may need to be provided judiciously until the urethral obstruction is relieved. Hyperkalemia should be treated if it is lifethreatening as determined by evidence of significant electrocardiographic abnormalities in combination with clinical signs. Sometimes relieving the obstruction rapidly is more effective at treating the hyperkalemia than administering drugs that either do not correct the problem or take time to take effect. This decision needs to be tempered with the side effects of the hyperkalemia as some of these patients may die without administration of medications.

Electrocardiographic abnormalities associated with hyperkalemia include bradycardia, peaked T waves, shortened QT interval, and a wide QRS complex. Eventually the P wave will disappear and when severe hyperkalemia will cause a sine wave appearance to the electrocardiogram. There are three treatment options available for hyperkalemia. Calcium gluconate at 0.5-1.0

ml/kg intravenously will counteract the effects of the hyperkalemia immediately but the effects will be only last 15-20 minutes. Sodium bicarbonate at 1 mEq/kg will drive the potassium intracellularly and provide a rapid, but short lived effect. Insulin give at 0.5 u/kg will drive the potassium intracellular and will have a more prolonged effect. Dextrose should be supplemented immediately at 1-2 g per unit of insulin given. A constant rate infusion of 2.5% dextrose may be required for a few hours until the effects of the insulin have worn off. Alternatively it may be sufficient to simply give the dextrose since the endogenous insulin response should be sufficient unless the patient has underlying diabetes or severe pancreatic insufficiency.

These patients are uncomfortable and often painful. Except in the moribund animal an opioid should be administered followed by a sedative or short acting anesthesia. The obstruction can be relieved in most cats effectively using heavy sedation. The author prefers a combination of butorphanol and midazolam with low dose ketamine or propofol as needed. Dexmedetomidine should be avoided in these patients as should general anesthesia unless the plan is to intubate and provide positive pressure ventilation and close blood pressure and electrocardiogram monitoring. If the patient is anesthetized positive pressure ventilation should be instituted to avoid compounding the metabolic acidosis with a respiratory acidosis, which will worsen the hyperkalemia. Epidural anesthesia can be highly useful especially a sacrococcygeal block.

Expulsion of a distal urethral obstruction in the male cat may be able to be relieved by massaging the urethra rectally. If this is not possible then a stiff catheter (an open-ended tomcat is preferred by the author) or an olive tipped catheter should be used to attempt to retropulse the obstruction. Gloves should be worn and an attempt to keep the procedure as aseptic as possible. Clipping may be required in longhaired cats. The catheters should be liberally covered in sterile water-soluble gel. The prepuce should be manipulated such that the urethra is pushed dorsally and straightened. This will facilitate flushing of the urethra. The tip of the catheter is placed up against the obstruction, a 12 cc syringe is attached and the plunger is flushed and withdrawn in a fairly rapid rhythm. The tip of the urethra should be digitally occluded to attempt to dilate the urethra. The procedure should be done gently and the catheter should never be forced because the urethra can tear easily.

If the urethra is very edematous a single antiinflammatory dose of dexamethasone sodium phosphate should be given intravenously. This can be continued every 12 hours for 2-3 days if the edema is severe. With difficult obstructions the use of lidocaine in the gel or in the flush may help. The total dose of lidocaine should not exceed 2.5 mg/kg. Freezing the catheter, which increases the stiffness may be useful.

Urethral obstruction relief in the female cat is done blind using digital palpation. Typically female cats will need to be anesthetized.

Once the obstruction is relieved a urine sample is saved for urinalysis and possible culture. Any calculi retrieved should be saved for analysis. The bladder should be flushed until the effluent is clear and then a soft indwelling urinary catheter should be placed and attached to a closed collection system. The catheters should be sutured to the prepuce. Catheters made of polyurethane or silicone are preferred in cats to minimize urethral irritation. The catheter should not be larger than 3.5 Fr in cats. Elizabethan collars should be placed.

Radiographs should be taken following relief of the obstruction to attempt to determine if calculi are present. Due to the size of most calculi and the superimposition of the pelvic bones on the urethra calculi may be present but may not be able to be visualized. Catheter position should

also be evaluated. Ideally the tip of the catheter should be just proximal to the trigone. Catheters that are inserted too far can lead to problems with bladder drainage and will occasionally spontaneously tie themselves in a knot creating a surgical emergency.

If the obstruction cannot be relieved there are several options. Cystocentesis can be performed. Ultrasound-guidance is recommended, in part to monitor for bladder rupture which is a risk. Emergency surgery may be required. If the patient is hyperkalemic or unstable there is a risk for death under anesthesia. In the critical patient a temporary cystostomy tube may be more appropriate than surgery to definitively correct the problem.

Fluid therapy must be closely tailored to the animals needs and should be based on cardiac status, volume status (central venous pressure), dehydration, sodium concentration, potassium concentration, albumin, and degree of kidney injury

Dehydration should be restored using crystalloids. Generally buffered electrolyte solutions are indicated. Fluids containing potassium should be used with caution in the hyperkalemic patient until urine output is restored and the potassium level is starting to decrease. Fluid requirements based on dehydration should be calculated and fluids administered over a period of 4-8 hours. Hourly maintenance requirements should be added to this amount along with ongoing losses. Postobstructive diuresis is not common but can lead to significant morbidity if it goes unrecognized. Urine output should be monitored on an hourly basis initially to ensure fluid requirements are being met then every 4 hours until the catheter is removed. Anuric patients may not even tolerate the amount of fluids required to restore dehydration until urine output is restored. If diuresis is the end goal then fluid will need to be adjusted to ensure urine output is greater than fluid input and usually greater than 2 ml/kg/hr. The bladder should be palpated every 4 hours to ensure the catheter is functioning.

As an example a polyuric 5 kg cat that is estimated to be 10% dehydrated has a fluid deficit of 500 ml. Maintenance requirements are approximately 10 ml/hr. Based on an assumption of 2-4 ml/kg/hr if the patient is polyuric adds an additional 10-20 ml of fluid required per hour. Assuming the patient's hydration is to be restored over 12 hours this cat has a fluid requirement of 60-70 ml/hr to meet the desired goals.

On occasion blood loss in the urine can lead to significant anemia and a blood transfusion may be required. If there is significant hematuria the packed cell volume should be monitored at least every 12 hours.

Laboratory tests should be repeated based on the status of the patient and the preprocedural results. Patients that were hyperkalemic need to have their potassium level rechecked within 2-4 hours. Additional tests should be performed at 4-12 hour intervals depending on the status of the patient. The uremia will resolve in most patients once the obstruction is relieved; however, some patients may require treatment for renal failure.

Infection is unusual in cats with lower urinary tract obstructions. Care should be taken when interpreting the presence of bacteria in the urine since bacteriuria does not always equate with infection. Antibiotics ideally should be based on urinalysis and culture results. Prophylactic antibiotics should be avoided in animals with indwelling catheters.

Analgesics should be provided to all patients showing signs of pain. Nonsteroidal antiinflammatory agents should be avoided in azotemic patients. Opioids should be given intravenously to effect initially. The animal can be transitioned to oral medications when

appropriate. Opioids and gabapentin are commonly prescribed and many animals appear to benefit from multimodal therapy.

Urethral spasm is not uncommon in cats and antispasmodics are typically recommended. Two common options for treatment include prazosin and phenoxybenzamine. Prazosin is an alpha-1 adrenergic receptor antagonist. Phenoxybenzamine is a non specific alpha adrenergic receptor antagonist. Although these drugs have been shown to reduce intra urethral pressures, clinical studies have yielded limited proof that they are truly effective. Diazepam has been described as being useful in treating urethral spasm, however, due to the potential for hepatic necrosis associated with diazepam the author does not recommend this.

The ideal length of time a catheter should remain indwelling is unknown; however, removing the catheter when the urine is not grossly clear (no major hematuria, no crystals, no mucus) is not recommended. The urinary catheter should remain in place in the cat for 24-48 hours depending on the doctor's preference. If the bladder is atonic the catheter may need to remain in place for up to 5 days. Instant removal and treatment of the cat as an outpatient, while sometimes required for financial reasons, is not recommended as there is a high likelihood of reobstruction in the first week. If calculi are present ideally the catheter should remain in place until surgery has been performed.

References available on request.

## REVIEW OF THE NEUROLOGICAL EXAMINATION AND LESION LOCALIZATION WITH A CLINICAL APPROACH

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Despite the advancement of diagnostics techniques in veterinary neurology, lesion localization remains the most valuable tool for the clinician. A thorough neurological examination with a practical approach provides significant information in regards to aetiology and outcome of the patient.

When assessing the neurological patient it is helpful to identify if the patient belongs to the Intracranial, Spinal or Peripheral group.

The main components of the neurological evaluation include:

1. Mental Status
2. Cranial Nerves
3. Gait and Posture
4. Spinal Reflexes
5. Presence of Pain or Dysphoria
- 6.

A systematic approach is helpful to become proficient with the performance and interpretation of the neurological evaluation.

**Mental Status:** The main purpose is to determine if the patient is experiencing abnormalities related to cognitive function and ability to interact appropriately with the owner and the surroundings. Typically cognitive impairment is seen with patients with cortical lesions. The patient may be less interactive, confuse, disoriented and having urinary or fecal accidents. Alteration of mentation related to disorders of the brainstem typically manifests with somnolence and increased sleepiness. This results from involvement of the ARAS (ascending reticular activating system) that is located in the brainstem. It is essential to interrogate the owner in detail about the mental awareness and status of the patient. Observing the patient in the examination room can also provide helpful information. The behaviour should be appropriate for the species (feline or canine) in a novel environment. Often compulsive behaviours such as circling, or inappropriate response to fear on a timid animal can be noted by observing the patient act/walk in the examination room.

### Cranial Nerve Evaluation:

There are 12 pairs of cranial nerves:

I. Olfactory, II. Optic, III. Oculomotor, IV. Trochlear, V. Trigeminal, VI. Abducens, VII. Facial, VIII. Vestibulo-chochlear, IX. Glossopharyngeal, X. Vagus, XI. Spinal accessory and XII. Hypoglossal.

The majority of disorders that are clinically relevant affect the Optic nerve, Trigeminal nerve, Facial nerve and Vestibulo-chochlear.nerve. An adequate menace response with a normal pupillary light reflex with good ability to navigate in the room without bumping into objects confirms the normal function of cranial nerve II- the optic nerve. The motor component of the trigeminal nerve is evaluated by assessing the muscle of mastication (masseter and masticatory) for symmetry. Three branches compose the sensory portion of the nerve. The ophthalmic, maxillary and mandibular branches. The ophthalmic branch is assessed by

touching the medial canthus of the eyelid; The maxillary branch by touching the lateral canthus and the mandibular by touching the base of the ear. A blink is expected as a response. Evaluation of the nasal sensation provides an afferent stimulus through the ophthalmic branch. The test is used in practice to assess the cortical function. Usually an asymmetric response is indicative of a cortical lesion that is on the opposite site of the deficit. Some animals will not respond to the nasal septum stimulus. The most important practical information obtained from this test is if the response is equal or asymmetric. A cotton-tipped instrument should be used for this test to detect subtle changes. To evaluate the facial nerve the face of the patient is observed carefully. The position of the upper and lower lips, eyelids, ears and nostrils should be compared for symmetry. The ability to blink is tested by eliciting the palpebral reflex. Facial paresis or paralysis causes a decreased palpebral reflex. The facial nerve is also involved in lacrimation (parasympathetic component). When evaluating the vestibulo-chochlear nerve the position of the head is observed, preferably from behind the patient to assess for the presence of a head tilt. Abnormal nystagmus, and leaning to the side are common signs found in patients with deficits of the vestibular portion of CN VIII. The client may also report signs of hearing loss that can be attributed to involvement of the chochlear portion of the nerve.

The oculomotor nerve, CN III it is less frequently affected in clinical practice than the other 4 nerves already discussed. The nerve is evaluated together with the trochlear IV and abducens VI because they control eye movement. The presence of physiological nystagmus is confirmed by moving the head from side to side. In the normal patient, the nystagmus has a fast phase in the direction of the head movement. CN III also mediates pupillary constriction (parasympthetic function), which is evaluated by the pupillary light reflex. Strabismus may be obvious with ventral deviation of the eyeball. This signs is commonly noted in vestibular patients.

The glossopharyngeal CN IX, Vagus CN X, Spinal Accesory XI and Hypoglossal XII are usually evaluated in bundle by asking the owner if the patient has any difficulty swallowing or prehending food. Symmetry of the muscle of the tongue can also be evaluated. In practice, disorders of these cranial nerves are suspected from the history obtained by the owner. Usually the presenting complaint includes regurgitation, dysphagia, inspiratory stridor or voice change.

**Gait and Posture:** Analysis of the gait can be the most challenging part of the examination. Often, by observing the animal relaxed on the examination room, subtle postural deficits can be detected. The position of the limbs may be inappropriate with a wide base stance. There may be rigidity of the limbs and spasticity on the spinal patient. The purpose of gait analysis is to answer three basic questions:

1. Is the gait normal or not? 2. Are front and hind limbs affected? 3. Is there ataxia present and what type of ataxia? Ataxia is defined as the inability to perform normal, coordinated motor activity that is not caused by weakness, musculoskeletal problems, or abnormal movements such as a tremors.

The types of ataxia include proprioceptive, vestibular and cerebellar. When a head tilt is present **vestibular ataxia** is confirmed. Usually a patient with this type of ataxia tends to lean or walks sideways. In severe cases the patient is recumbent and rolls. The presence of a head tilt and nystagmus helps in confirming that the problem is affecting the vestibular system.

**cerebellar ataxia** manifests with intention tremors and this type of ataxia is characterized by alterations in the rate, range, direction and force of movement. Hypermetria is commonly observed in these cases. There is an overreaching and hyper stepping type of gait.

**Proprioceptive ataxia** is the type of ataxia seen in patients with spinal disease. The ataxia results from the loss of the sense of limbs and body position due to interruption of ascending proprioceptive pathways. The ataxia is characterized by clumsiness, incoordination, swaying

gait and wide base stance. Usually the limbs slide to the side when the patient is walking. The steps are inconsistent and the limbs land in different areas when the patient takes steps. The stride of the affected limb tends to be longer than normal and the toes often drag or scuff the ground.

Careful analysis of the gait is critical in creating a correct differential diagnostic list. Once you establish if the patient is ataxic or not, the selection of diagnostics tests will be completely different. A spinal patient is usually worked up with advanced imaging. The peripheral patient or metabolically weak patient requires other diagnostics procedures (eg. arthrocentesis, tensilon test).

Postural reactions test the same neurological pathways involved in the gait, proprioceptive and motor pathways. Their main value is detecting asymmetry that may not be obvious during the observation of the gait. It is important to remember that postural deficits should be interpreted in the light of gait analysis. Systemic weakness, fever, orthopaedic disease and fear can affect the knuckling response. Proprioceptive pathways are often compromised on the early stages of neurological disease so the deficits may be detected prior to observing signs of weakness. Once all the elements of the neurological exam are combined, it can be established if the deficit seems relevant for the patient that is being evaluated. Cortical lesions are usually characterized by postural deficits that are opposite to the side of the lesion. In a patient suspected to have a cortical lesion the menace response, and nasal septum are evaluated carefully for symmetry to establish if there are contralateral deficits on these tests as well as proprioception.

### Spinal Reflexes:

Spinal reflexes assess the integrity of the sensory and motor component of the reflex arc and the influence on the descending UMN pathway. The most relevant information that needs to be determined is if the reflex is present or absent. It is important to keep in mind that when there is alteration of the muscle mass and extreme rigidity, it can be hard to establish if the reflex is present. In this case the rigidity should be overcome by flexing the limb slowly multiple times. When evaluating the withdrawal reflex in these spastic limbs, it is recommended to start pinching the toe once the limb is successfully flexed. An absent reflex indicates that the lesion could be present in the peripheral nerve, nerve root, spinal segment, neuromuscular muscle and muscle. These are the components involved in the reflex arc. SPINAL SHOCK is a temporary paralysis and loss of reflexes caudal to the lesion that can last for 12-48 hrs after severe spinal injury. Therefore, trauma patients should be evaluated sequentially to determine if spinal shock is present and affecting the results of the examination. If the gait analysis indicates that there is proprioceptive ataxia a spinal lesion is investigated. The next step to localize the area affected is determined by the limbs affected and the presence or absence of reflexes. Spinal lesions affecting **C1-C5** involve all 4 limbs and have normal reflexes. Abnormalities of **the C6-T2** spinal segments manifest with involvement of all 4 limbs and decreased reflexes on the forelimbs. Lesions that compromise the **T3-L3** region tend to present with abnormalities of the hind limbs only but intact reflexes. Lastly spinal lesions affecting the **L4-S2** enlargement will have decreased reflexes and only the hind limbs will be affected. Muscle tone is a valuable element to corroborate the suspected lesion localization. Lesions of C1-C5 and T3-L3 have normal to increased tone. Lesion affecting the C6-T2 and L4-Ss enlargements can present with decreased muscle tone.

**Pain and Dysphoria:**

To evaluate the patient for presence of spinal pain downward pressure is applied over the spinal canal keeping both fingers close to each other and pressure should be applied between the spinous processes. One hand should be placed under the abdomen to detect tensing of the abdominal muscles as the affected area is palpated. Often cervical pain can be detected by observing the posture of the patient and the presence of muscle cervical fasciculations.

History is an essential part of the neurological examination. Occasionally there will be neurological patients that present with vocalization. Owners will attribute the vocalization to presence of pain. However, is not uncommon that a dysphoric patient, presenting with brain disease such as brain tumor, encephalitis or hydrocephalia will vocalize constantly as a manifestation of dysphoria and not pain.

Similarly intracranial tumors can present with pain. It is believed that stretching of the meninges could be responsible for pain in these patients.

*References available upon request*

## CHARACTERIZING OF EPISODIC EVENTS IN VETERINARY NEUROLOGY. ARE ALL OF THEM REALLY SEIZURES?

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Movement disorders in people.

The prevalence of movement disorders in humans have been estimated at 14.5% for tremors, followed by restless legs syndrome (10.8%), parkinsonism (7%), dystonia (1.8%), and chorea and tics (<1% each). Recently awareness about similar conditions in veterinary medicine has gained interest and classification of movement disorders is starting to advance rapidly.

Contrary to what was believed in the past, not all episodic events in veterinary medicine are the result of seizure activity. Movement disorder and vascular events can present similarly. This talk is a review the clinical approach to episodic events in veterinary medicine. Movement disorders are clinical syndromes with either an excess of movement or paucity of voluntary and involuntary movements, unrelated to weakness or spasticity. Movements can be classified as:

### Voluntary

#### Semi voluntary

Induced by inner sensory stimulation

Ticks

#### Suppressible

#### Involuntary

Less suppressible (tremor, dystonia)

Dyskinesia = abnormal involuntary movement during movement or rest on a conscious animal

The pathophysiology underlying movement disorders in dogs remains unknown. A proposed classification of is considered a work in progress that will continue to advance with genetic studies. Links to gluten hypersensitivity in Border terriers with so-called **canine epileptoid cramping syndrome (CECS)** was one of the movement disorders that have been currently characterized in veterinary medicine. **Myoclonus** is defined as a pacemaker like depolarization of local motor neuron. The abnormality is seen in cases of distemper, lead toxicosis and other CNS inflammatory disorders. Myoclonus tends to be present under general anesthesia. **Tremors** result from the contraction of muscles with opposing functions (biphasic nature). Tremors indicate dysfunction of the basal ganglia or substantia nigra. Essential tremors/geriatric (senil tremors) are considered an exaggerated form of physiological tremors and when they occur later in life are considered senile tremors. These are usually not clinically significant. Tremors are present in the veterinary patient as a result of systemic weakness and sporadically in an individual with neuromuscular disorders. Tremors associated with these disorders, however, are often of short duration, episodic, and present during attempts at muscle activity.

Some of the accepted classification of movement disorders in humans includes:

### **Hyperkinesia**

Chorea, dystonia, myoclonus, tics, tremor, asynergia, ataxia, dysmetria, ballism, stereotypy, athetosis, akathisia, myokymia

### **Hypokinesia**

Parkinsons

Apraxia, cataplexy, blocking tics, catatonia, freezing

In veterinary medicine recognition of dyskinesia has improved significantly in the last 5 years. Dyskinesia is characterized by an impairment of the power of voluntary movements resulting in a fragmented and incomplete action. Dogs reported with these abnormalities may exhibit abnormal postures, such as holding a limb up, adopting a kyphotic posture of the spine without been able to initiate movement. The impaired movements can appeared as a muscle cramp. Cramps are defined as paroxysmal prolonged and severe muscle contraction that may be painful. Examples of diseases associated with cramps which maybe dyskinesias include **Scotty cramp, episodic falling of the Cavalier King Charles** and **epileptoid cramping of the Border Terrier**. **Scotty cramps** are clinical episodes of dystonia noted between 6 wks to 3 yrs of age, and may be elicited by stress, excitement or exercise. The thoracic limbs are initially affected becoming abducted shortly after exercise begins; this is followed by arching of the lumbar spine and pelvic limb stiffness and falling. **The episodic falling of Cavaliers** affects dogs between 3-7 months and up to 4 yrs of age. The condition manifests by exercise induced bouncing pelvic limb gait, limbs are abducted and appeared stiff. This may progress to bunny hopping, arching of the spine and collapse. Therapy with Clonazepam 0.5 mg/kg q 8 hrs, is effective but tolerance to the benzodiazepine eventually develops.

An extreme **generalized muscle stiffness in Labrador Retriever** signs are noted between 2-41 months of age. The condition seems to first affect the pelvic limbs and then progresses to the forelimbs causing the patient to present for exercise intolerance. The hallmark of the clinical presentation is muscle stiffness that persists at rest and results in restricted joint movement. An X- linked genetic component is suspected.

**Myokymia and neuromyotonia.** Myokymia is one of the clinical signs of neuromyotonia. Neuromyotonia is clinically characterized by a combination of muscle twitching or myokymya, persistent muscle contraction, muscle stiffness or cramps, and impaired muscle relaxation. Abnormalities of the K channel are implicated in the disease. Dogs- **Jack Russell** and cats have been reported to suffer from the condition.

Some dyskinesias are difficult to differentiate from seizure activity. **Chinooks** exhibit a condition that is characterized by inability to stand or ambulate, head tremors, and involuntary flexion of one or multiple limbs, without autonomic signs or loss of consciousness. Episodes duration varies from minutes to an hour. There has been consideration of whether the condition represents a seizure or a movement disorder. An **idiopathic head tremor** with movement of the head side to side or up and down affecting **Bulldogs** has been seen frequently in practice. **Doberman and Boxers** exhibit a similar tremor. Previously the condition was thought to represent a focal seizure but lately it has been considered a dyskinesia. Antiepileptic therapy is usually

unsuccessful. The tremors tend to be self-limiting and as long as the quality of life is not affected therapy is not recommended. The patient is usually responsive and the tremor resolves with stimulation such as offering food or calling patient’s name.

Discriminator	Paroxysmal dyskinesia	Idiopathic head tremor	Epileptic seizure	Vestibular attack	Syncope	Narcolepsy/Cataplexy	Neuromuscular weakness	Paroxysmal behavior changes (compulsive disorder)
Clinical status between episodes	Unremarkable	Unremarkable	Unremarkable or forebrain signs	Unremarkable	Normal heart rate or arrhythmia, pulse deficits, heart murmur, cyanosis, abnormal lung auscultation	Altered sleep/wake cycle, normal clinical examination	Unremarkable or generalized weakness, muscle atrophy, pain, and decreased reflexes	Unremarkable
Precipitating event or trigger	None or activity, exercise, excitement, and stress	None or stress, fatigue, and overstimulation	None or flashing lights, anxiety, and stress	None	Exercise, excitement, and stress	Excitement and eating	Activity and exercise	Behavioral triggers (e.g., fear)
Pre-event changes	Not applicable	Not applicable	Preictal signs may be observed including anxiety, restlessness, increased affection, contact-seeking, withdrawal, hiding, aggressiveness, and vocalization	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable
Event description	Dystonia, chorea, ballismus, athetosis, tremors, impaired posture, and inability to stand or walk	Vertical or horizontal rhythmic head movement (5–8 Hz)	Depending on seizure focus, focal or generalized, tonic-clonic movements most common	Cardinal signs of vestibular dysfunction present: head tilt, nystagmus, vestibular ataxia, and collapse toward side of head tilt	Brief, sudden collapse, and rapid recovery	Sudden collapse	Stiff, stilted gait prior to collapse	Pacing, barking, licking, chasing imaginary objects or tail, and chewing objects
Level of consciousness	Unremarkable	Unremarkable	Often impaired or unconscious	Unremarkable or disoriented	Reduced to absent	Unremarkable if only cataplexy. Absent (asleep) in narcolepsy	Unremarkable	Unremarkable
Autonomic signs	Not applicable	Not applicable	Possible: hypersalivation, defaecation, and urination	Not applicable	Possible abnormalities of heart rate and rhythm	Not applicable	Not applicable	Not applicable
Muscle tone	Hypertonicity (focal or generalized)	Normal	Typically increased: tonic (hypertonicity) or alternating tonic-clonic movements	Unilateral decrease in extensor muscle tone	Flaccid (all body)	Flaccid (all body)	Often flaccid (can appear spastic with certain myopathies)	Normal
Lateralizing signs	Possible	Not applicable	Possible	Yes	Not applicable	Not applicable	Not applicable	Not applicable
Duration	Seconds to hours	Seconds to hours	Seconds to minutes or >5 min in case of status epilepticus	Seconds to hours	Seconds	Seconds to minutes	Minutes to hours	Minutes to hours
Postepisodic changes	Unremarkable or tiredness	Unremarkable, tiredness, or restlessness	Postictal signs frequently occur including disorientation, aggressive behavior, restlessness, pacing, lethargy, deep sleep, hunger, thirst, ataxia, proprioceptive deficits, and blindness	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable
Further comments	Interaction with the owner can alleviate or interrupt the episode. Consider breed specific disorders and age at onset	Episodes can be interrupted in two thirds of the cases by the owner	Facial muscles often involved during the ictus	Subtle signs of vestibular disease might persist	Clinical signs of heart failure possible, e.g., dry cough and increased respiratory noise	Often occurs in young purebred dogs	May be accompanied by dysphagia, dysphonia, regurgitation, and dyspnea	History of anxiety disorder

Ultimately the use of electroencephalography (EEG) is the goal standard to differentiate seizure events from movement disorders. However performing EEG has multiple limitations. Veterinarian’s awareness of movement disorders been a possible cause of episodic events, will result in further knowledge about the correct management of the conditions.

*References are available upon request.*

## REVIEW OF ANTIEPILEPTIC THERAPY AND SEIZURE MANAGEMENT

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The incidence of seizure activity is estimated to be between 2-5 % of the canine population. This presentation will provide practical tips to approach seizures focusing on antiepileptic therapy. A brief discussion of the use of phenobarbital, potassium bromide, zonisamide, levetiracetam, topiramate will be presented.

Epilepsy is a commonly encountered condition in small animal practice. Despite the demonstrated efficacy of traditional anticonvulsant drugs such as phenobarbital and bromide, a significant proportion of canine and feline epileptics experience inadequate seizure control or intolerable adverse effects associated with these drugs. Alternative anticonvulsant drugs such as gabapentin, levetiracetam, pregabalin, topiramate, and zonisamide have shown promise for the management of veterinary seizure disorders, and their use is becoming more widespread in veterinary neurology. With an understanding of the pharmacology and practical considerations of antiepileptic drugs the clinicians can effectively incorporate these drugs into clinical practice, to improve seizure control and quality of life for epileptic patients and their owners.

Antiepileptic therapy is properly selected when the etiology of seizures, risk of recurrence, seizure type and negative side effects of medication are carefully considered. An International Veterinary Epilepsy Task Force created a Consensus Statement in 2015 covering relevant guidelines and definitions for the management of the epileptic veterinary patient. *Epilepsy* indicates recurrence of seizures of primary brain origin. The diagnosis of **Idiopathic Epilepsy** should be considered likely in dogs between the age of **6 months and 6 years** when metabolic causes are excluded and no neurological deficits are present between seizure events. **Structural Epilepsy** results when the seizures are provoked by confirmed intracranial/cerebral pathology. In the epileptic patient survival is heavily influenced by the anxiety experienced by the family and financial burden of treating the disease. Dogs with epilepsy have an increased risk of premature death due to euthanasia. A major factor to consider when managing any epileptic patient is the significant commitment required from the owner to follow care guidelines, routine rechecks, and therapeutic monitoring recommendations. Therapeutic failures should be anticipated, regardless of the anticonvulsant(s) used, unless the client is properly educated and willing to commit to manage the epileptic dog understanding the chronic nature of the condition.

**Current guidelines to initiate antiepileptic drugs (AED) include** **1.** Identifiable structural lesion present or prior history of brain disease or injury; **2.** Acute repetitive seizures/ Status epilepticus has occurred (ictal event > 5 minutes or three or more generalized seizures occur within a 24 hour period); **3.** Two or more seizures events occur within 6-month period; and **4.** Prolonged, severe, or unusual postictal periods occur.

The frequency of seizure activity in a given period of time should correlate with the intensity of management, quantity of drugs needed, and time to achieve control and subsequent recovery. It is crucial that generalized seizures be controlled rapidly.

Pharmaco resistant epilepsy is inadequate seizure control despite documentation of therapeutic steady-state serum concentrations of one or more traditional anticonvulsants, such as phenobarbital or bromide. Alternative therapies should be considered as a primary therapeutic option when:

\_ Owners have concerns about the common adverse effects associated with phenobarbital or bromide treatment for canine or feline epileptics.

\_ Dogs and cats with secondary epilepsy resulting from malformation.(e.g, hydrocephalus), meningoencephalitis, or a brain tumor. These conditions can affect the level of consciousness. Therefore adding other medications that will not result in further sedation, a common effect of phenobarbital and bromide, will be beneficial for these patients.

\_ Epileptic dogs or cats that have coincidental disease (e.g. liver dysfunction) or are receiving concurrent drugs with known or potential interactions with phenobarbital or bromide.

Intolerance to anti seizure medication is considered to be present in animals that are on phenobarbital and/or bromide and experience clinically intolerable adverse effects, such as sedation, ataxia, polyphagia, polyuria, or polydipsia, at serum concentrations necessary to achieve adequate seizure control.

levetiracetam is a popular choice in this clinical situation because it is rarely associated with adverse effects and rapidly confers anticonvulsant activity.

The following table provides a good review of antiepileptics used in Veterinary medicine

Medication	Steady State	Dose	Side Effects
Phenobarbital	10-14 days	2 mg/kg BID Puppies 6 mg/kg BID	Sedation Ataxia PU/PD Liver toxicity Rarely dermatitis
Potassium Bromide	3-4 months	40 mg/kg SID Loading dose reported 600mg/kg over 2 days	PU/PD Ataxia when combined with phenobarbital
Zonisamide	3-5 days	5-10 mg/kg BID	Vomiting Ataxia, KCS
Topiramate	Unknown	5mg/kg BID	Sedation and urinary calculi
Pregabalin		3-4 mg/ Kg TID	Sedation, Ataxia
Levetiracetam	1-2 days	20mg/kg TID	None

a. Dogs naïve to phenobarbital in status epilepticus:

An IV bolus is administered 2 – 5 mg/kg followed by a phenobarbital CRI at 2 – 6 mg/dog/h. Phenobarbital bolus 2 – 5 mg/kg can be repeated, if necessary, for a total of 16 mg/kg (4 boluses) 20 minutes apart. This high dose of phenobarbital is rarely required. When phenobarbital is given, it should be administered with extreme caution to avoid marked respiratory depression. The cumulative effect of the diazepam administered prior to the phenobarbital should be taken into consideration. Close patient monitoring is required when phenobarbital is given as a CRI.

If the dog is already on phenobarbital for maintenance therapy, the phenobarbital serum levels should be taken prior to IV administration. One phenobarbital 2 – 5 mg/kg bolus can be administered followed by a phenobarbital 2 – 6 mg/dog/h CRI. Note that phenobarbital and diazepam although reported as effective at controlling experimentally-induced focal seizures are not as effective clinically when the focal seizure has been of long duration.

Dogs in the decompensatory phase of convulsive status may benefit from treatment with dexamethasone 0.1 mg/kg q 24h for two to three days.

**Phenobarbital:** Barbiturate with a strong antiepileptic effect and variable sedative effect. Its antiepileptic effect is post-synaptic acting ultimately on GABA by prolonging the opening of the postsynaptic cell membrane Cl<sup>-</sup> channel. Phenobarbital equilibrium is reached in the cerebrum in 5-10 minutes, necessitating a 20 minutes pause between boluses. The metabolism is slow with a half-life of 40 to 80 hours. Repeated dose is cumulative. Barbiturates and benzodiazepines are synergistic. Therefore, careful titration must be used with concomitant use especially in patients with liver dysfunction. Chloramphenicol and cimetidine are to be avoided with phenobarbital. Their inhibition of the phenobarbital metabolism leads to toxic phenobarbital serum levels as early as after one treatment.

**Potassium Bromide:** The medication is typically given as an inorganic salt with a starting dose of 40 mg/kg/day. The median elimination life is 15.2 days. Steady state concentrations fluctuate between dogs due to individual differences in clearance and bioavailability. Dietary factors alter serum drug concentrations. The drug is excreted in the urine without known hepatic metabolism or toxicity. Usually the medication is used as add on therapy. There is very limited scientific data about the efficacy of Potassium Bromide as a monotherapy.

**Zonisamide** can be use at a dose of 5-10 mg/kg orally. Dogs concurrently taking phenobarbital may required the higher dose of 10 mg/kg. Co-administration with phenobarbital increases Zonisamide clearance by about 50%. Since the medication can cause sedation titrating to clinical response is recommended. Zonisamide has been proven to be effective as a single antie-eileptic and also as add on medication. The drug is metabolized by the liver. When Zonisamide levels are evaluated, a trough sample is recommended within 1 hr before the next scheduled administration. The human therapeutic range 10-40 ug/ml is currently also used in dogs.

**Levetiracetam** is an excellent alternative for patients with structural brain disease. The benefit of avoiding sedative side effects allows for accurate monitoring of patient's

mentation. The recommended dose is 20-30 mg/kg PO every 8 hrs. Due to its minimal side effects compared to other antiepileptics levetiracetam has gained increasing popularity. Monitoring of serum concentration is controversial and usually recommended when the patient is poorly controlled and taking phenobarbital concurrently. Phenobarbital will alter the pharmacokinetics of levetiracetam. The reference range is extrapolated from the human range at 12-46 ug/ml.

**Pregabalin:** The recommended dose of is 2 mg/kg every 12 hrs with an increase of oral dose after the first week to avoid excessive sedation. The elimination half-life is 7 hrs. The target dose for epilepsy is 3-4 mg/kg every 8-12 hours. Marked sedation can be noted with this medication therefore its use may be limited.

**Diazepam:** Constant rate infusion (CRI). Diazepam is incompatible with other medications. Diazepam should be added to all protocols to reduce the requirement for other drugs. Initiate diazepam infusion at 0.5 mg/kg/h. It is added to the maintenance hourly fluid therapy in the in-line burette. Prepare, at the most, 2 hours at a time (as diazepam is light sensitive and binds to the plastic tubing). The dosage of diazepam can be safely increased to 1 mg/kg/h for one or two hours if necessary. Once seizure activity has stopped for a minimum of four hours, the infusion can be gradually discontinued over as many hours as it took to control the seizure activity; i.e., if it took 6h to reach control, allow 6h to discontinue the diazepam. Refractoriness to diazepam may be occurring if the seizures continue despite increasingly higher dosages. If generalized seizures are observed following the first hour of diazepam infusion, Phenobarbital CRI is added to the diazepam infusion.

**Gabapentin:** The mechanism of action involves inhibition of the calcium channels and enhancement of the release or actions of GABA in the brain. The medication is excreted by the kidney in people. In dogs 30-40% of the drug undergoes hepatic metabolism. The recommended dose for seizures is 25-60 mg/kg body weight every 8 hrs. The therapeutic range is considered to be 4-16 mg/l.

**Topiramate:** acts by enhancing GABA activity and inhibiting calcium and sodium channels. The elimination half-life is 2-4 hrs. The proposed oral dose is 5-10 mg/kg every 6 to 12 hours. Reported side effects can include sedation and ataxia. Due to its short half-life, frequency of administration is a limiting factor for the use of Topiramate in practice. The drug has been beneficial as add on in few patients that did not responded well to other anticonvulsants.

**Phenobarbital and bromide** both work primarily via postsynaptic-aminobutyric acid (GABA) agonism.

The following diagram summarizes mechanism of action of different anticonvulsants:



## NARROWING THE LIST OF DIFFERENTIAL DIAGNOSIS IN THE NEUROLOGICAL PATIENT BY BREED AND AGE BRACKET.

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When presented with a neurological patient the signalment (breed, sex, age) and history are essential elements in guiding the clinician towards creating an appropriate differential diagnosis list. Once this is established the selection of the tests will be impacted by it. Moreover our ability to properly work up the patient and determine a treatment plan often will depend on the possible prognosis given to the owner from this first assessment. It is important to recognize that the information provided on this presentation should always be interpreted or applied in combination with a properly performed neurological examination.

A table with a summary of suspected or confirmed breed predilections for various neurological disorders can be consulted on Practical Guide to Canine and Feline Neurology 3<sup>rd</sup> edition. Copies will be available at the presentation. An important number of the conditions listed in the table are genetic in origin and rare in clinical practice.

The common neurological disorders affecting certain breeds that are seen at the Mississauga Oakville Referral group will be discussed. It is important to keep in mind that the conditions can vary depending on the geographic location and trend of breeds per period of time.

Seizures and **Epilepsy**. The most common breeds suffering from epilepsy in our region include: **German Shepherd, Golden Retriever, Labrador Retriever, Border Collie, Siberian Husky and Australian Cattle Sheepdog**. Typically genetic/idiopathic epilepsy is suspected when the patient starts experiencing the episodes between 6 months and 6 years of age. The neurological evaluation in these patients tends to be normal and behaviour in between the episodes is also unaltered. It is recommended to start therapy in this group of patients when: **1.** the episodes are acute and repetitive. **2.** The patient presents on Status epilepticus (ictal event that last 5 or more minutes or 3 or more generalized seizures within a 24 hr. period). **3.** There are 2 or more seizures events occurring within 6 months or when prolonged, severe, or unusual postictal periods are seen.

The management of inflammatory brain disease is a complex. The syndrome has evolved significantly over the years. The initial stereotype of GME has created a misleading impression about inflammatory disorders in dogs. It is important to note that dogs with CNS inflammation can have a good prognosis. There are multiple conditions that manifest with inflammation of the brain. Some clinical relevant distinctions that can allow guidance to the clinician include: If the problem is affecting just the meninges? Or are we dealing with parenchymal CNS disease? **Steroid Responsive Arteritis Meningitis (SRAM)** is a meningeal inflammatory disease often seen in **Boxer, Bernese mountain dog, Beagle, Golden Retriever, Pointers and Weimaraner**. Clinical signs usually include severe neck pain, fever. The condition has an excellent prognosis when treatment with immunosuppressive therapy is initiated early in the course of the disease. Affected patients are usually young (less than 2 years). Concurrent autoimmune polyarthritis appears to be common with this disorder. Inflammatory disorders affecting the brain and spinal parenchyma are more complex and presentation and outcome can vary greatly. Previous published data described cases of GME (granulomatous meningoencephalitis) that were confirmed at post-mortem. The condition became a clinical diagnosis when indeed is only a histopathological description of tissue. Moreover a term that became associated with a negative prognosis since the only cases published were the ones that had a post-mortem. Previous experience indicates that a good portion (60-70%) of patients with

inflammatory disease can survive if treated early with temporary remission of clinical signs or full recovery. Currently the most appropriate term to define inflammatory conditions of the CNS parenchyma is **Meningoencephalitis of Unknown etiology (MUE)**. Breeds that are commonly presented for the condition include **Chihuahua, Yorkshire terrier, Miniature pinscher, Maltese and Poodle**. Usually the illness affects patients of a young to middle age bracket. It is important to notice that inflammatory conditions have been recognized with more frequency in larger breed dogs, anecdotally it has been observed that it tends to have a better prognosis in the larger breed dogs. An immune cause is suspected to be responsible for MUE and therapy includes corticosteroids, Azathioprine and Cytosine Arabinoside (Cytosar).

Cervical pain is a common clinical sign in the neurological patient. With the advancement of imaging techniques (CT and MRI) multiple disorders of the craniocervical junction have been described and should be classified under the term **Craniocervical junction abnormalities**. The previously recognized one includes Atlantoaxial anomaly and caudo-occipital malformation syndrome (COMS). Atlantoaxial anomaly typically affects small and toy breed dogs such as **Yorkshire Terrier, Chihuahua and Miniature Pinscher**. Novel anomalies that can present in a similar way include atlanto-occipital overlapping, In this malformation the atlas (C1) is cranially displaced into the foramen magnum, and overlap of the occipital bone and atlas occurs. This displacement tends to compress the caudal aspect of the cerebellum and to elevate and compress the caudal medulla (medullary kinking).

**Caudo-occipital malformation syndrome** or Chiari like malformation refers to a syndrome that consists of hydrocephalus, caudal fossa crowding, cerebellar herniation and syringohydromyelia. All or some of the four anomalies may be present when the condition is diagnosed. The condition affects small breed dogs. Initially it was recognized and extensively studied on the **Cavalier king Charles** and the **Griffon Bruxellois**. Other commonly affected breeds include **Toy poodle, Miniature Pinscher, Chihuahua, Pug, Pomeranian, Maltese, Shih-Tzu** and **Pekingese**. Most dogs affected with COMS present before 1 year of age. However the condition has also been recognized in older patients. It is believed that patients that present before 2 years of age tend to experience more severe clinical signs.

**Cervical spondylomyelopathy** (also known as wobbler syndrome, cervical stenotic myelopathy, cervical vertebral instability) is characterized by static and dynamic spinal compressions. The disease is commonly cause by osseous compressions in young giant breed dogs and by disc protrusion in middle age to older large-breed dogs. The commonly recognized breeds affected by the condition include **Doberman Pinscher, Weimaraner, Bernese mountain dog** for the disc protrusion associated one. The Osseous compressive type of disorder is most commonly observed in **Great Danes, Mastiff and other giant breeds**.

Primary brain tumours include those that originate from brain parenchymal tissue (glial cells and neurons), cells compromising the outer and inner lining of the brain (meninges and ependymal) as well as vascular elements (choroid plexus). Primary **brain tumours** usually occur in dogs 9 years of age or older. A significant higher incidence is noted in **Golden Retriever and Boxer**; meningiomas are the most commonly diagnosed tumour in dogs. Gliomas, hystiocytic sarcoma and lymphoma are other types of CNS neoplasia that are less frequently recognized in practice.

*References available upon request*

## FELINE NEUROLOGY

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The neurological examination can be challenging to perform in cats. Recognizing this limitation is essential so the clinician can select the most relevant tests in order of clinical priority. Depending on the presenting complaint and history, the clinician should elect the order in which the examination is performed, (cranial nerves, gait and posture, spinal reflexes and presence of pain/dysphoria) in case a complete exam is not possible.

More recently, the term Epilepsy of Unknown Cause (EUC) has been adopted to classify seizure disorders in cats where no underlying cause can be identified. The main diagnostic criteria in cats with EUC include absence of pathological findings by diagnostic imaging and cerebrospinal fluid (CSF) analysis and/or a history of seizures with no abnormalities detected on neurologic examination during interictal periods. The median age at onset of seizures in EUC is generally <7 years, but the range varies considerably between 0.4 and 14.4 years, and no breed or sex predilections have been identified. A patient should be classified in this category when other causes of active structural disease have been ruled out. In the patient presenting for **seizures** activity the most important tests include: **mental status, menace response, nasal septum and postural reactions**. Mental status should be investigated based on the behaviour reported by the owners at home. Often cats can be harder to assess for mental status due to their independent nature. Disorders resulting in central nervous system inflammatory conditions can also manifest with seizures. In these cases, frequently lateralized deficits are found on examination. A serious cause of CNS inflammation includes **feline Infectious peritonitis (FIP)**. The condition can affect the meninges, intracranial cavity or the spinal cord. Unfortunately **FIP** carries a poor prognosis.

**Feline meningoencephalomyelitis of unknown origin (FMUO)** can present in a similar way to FIP. Clinical signs included ataxia, proprioceptive deficits, seizures, and spinal hyperesthesia depending on the areas affected. The median age reported for patients suffering from FMUO is 9.4 years. Diagnostics usually reveal CSF mixed pleocytosis and increased microprotein. MRI findings consist of intraparenchymal infiltrative ill-defined lesions. In contrast to FIP, prognosis for FMUO appears to be good with immunomodulatory therapy. Usually the affected patients receive, corticosteroid, lomustine, cytarabine, and anticonvulsant medications. **Cuterebrae larva migrans** can cause a parasitic meningoencephalomyelitis, which is difficult to differentiate clinically from FIP and FMUO. It has been suggested that intraparenchymal haemorrhage and meningeal enhancement on MRI studies are common findings with the parasitic migration.

**Ventroflexion of the neck and head** is occasionally observed in Feline patients presented for neurological evaluation. Differential diagnoses to consider with this presentation include **Myasthenia gravis, Thiamine deficiency, Myopathy and Hypokalemia**.

Thiamine deficiency is a rare clinical problem in pets that are fed a commercial well balanced diet. However, the author has observed cases of outdoor cats that go missing for several days affected with the condition. Thiamine is a necessary co-factor for normal carbohydrate oxidation, and its deficiency results in depleted ATP production in the brain with subsequent neuronal dysfunction. Often patients that present for thiamine deficiency have vestibular signs as well. Seizures and alteration of PLRs can be observed. Fish is high in thiaminase and feeding an all-fish diet to cats can lead to thiamine deficiency. **Myasthenia gravis** is manifested by recurrent and progressive muscle fatigue with exercise. In cats the acquired form of the disease is much more common than the congenital form. Acquired myasthenia gravis is an autoimmune disease in which antibodies (in most cases IgG) are formed against the nicotinic ACh receptors, resulting in decreased number of receptors on the postsynaptic sarcolemma surface. The consequence of the decrease in normal neuromuscular transmission is skeletal muscle weakness. Myasthenia gravis has been associated in cats with presence of thymomas and methimazole therapy. Loss of palpebral reflexes is common in the condition. Therapy is provided with pyridostigmine bromide at a dose of 0.25 mg/kg q 8-12 hrs. Occasionally corticosteroids need to be added at a dose of 1 mg/kg daily. Dose of medication may need to be titrated to clinical response

A **steroid responsive polyneuropathy** has been recognized in cats. Bengals are predisposed. The condition can be difficult to differentiate from neuromuscular disease. The absence of spinal reflexes in a patient with generalized weakness and exercise intolerance help to confirm the diagnosis of polyneuropathy. In contrast, the myasthenic cat, presents with normal spinal reflexes. When treating the polyneuropathy, immunosuppressive therapy (eg prednisone 1 mg/kg SID) maybe required for 6-8 months and occasionally for life. A similar condition can be seen post vaccination and resolutions of clinical signs tend to be within 1 week.

In contrast to peripheral and neuromuscular disorders, where the gait is characterized by weakness and exercise intolerance, **spinal disease** usually manifests with proprioceptive ataxia. Causes of proprioceptive ataxia include **fibrocartilagenous embolic myelopathy (FCEM), myelopathy, intervertebral disc disease (IVDD) and neoplasia**. Intervertebral disc disease is observed with less frequency in cats than dogs. The presentation of IVDD can be challenging because often the lumbar spine is affected without obvious signs of hind limb incoordination. Typically, the presence of a “wobbly” gait is what should alert the clinician to suspect myelopathy. It is important to remember that the lumbar spine has a greater component of gray over white matter and this is the reason why lower lumbar lesions can be harder to differentiate from peripheral neurological disorders and orthopedic pathologies. Fibrocartilagenous embolic myelopathy is an important syndrome that results from embolization of the arterial or venous supply to an area of the spinal cord. The mechanism by which the material reaches the vasculature of the spine is unknown but suspected to be secondary to extruded material entering the venous system or vertebral bone marrow. Most of the reported cats with FCEM tend to be middle age or older. The condition often affects the cervical spine C6-T2. Clinical signs tend to be asymmetric and none progressive.

Although spinal pain was reported as absent in previous literature, currently it is considered that the condition can be painful in some patients at its early stage. Cats can have severe presentation with FCEM and can demonstrate a Horner's syndrome when the cervical spine is affected. Literature suggests a 79% rate of recovery with the majority of the patients returning to normal. The median time to recover ambulatory function in the cats affected by FCEM was estimated at 3.5-11.5 days.

**Feline hyperesthesia syndrome** is a poorly understood condition that has been recognized in practice with increased frequency. Affected cats intermittently display clinical signs of rippling of the skin over the dorsum and muscles spasms of the thoracolumbar region as if this was resulting from an irritative phenomenon. Recent evidence of inclusions bodies found in histopathological samples suggests the origin of the condition maybe a myopathy. Rimmed intramyofiber vacuoles, containing paired helical filaments and beta-amyloid, were found in biopsies from affected cats. The condition can present with violent biting and licking at the flank/back area, agitated behaviour and vocalization. Dilated pupils and aggressive behaviour have also been described. Therapy with corticosteroids, phenobarbital and gabapentin/pregabalin has been attempted with variable success rates but the overall prognosis tends to be poor with progression of clinical signs.

The most common **neoplastic disorder** includes meningiomas of the brain or spinal cord. History and presenting clinical signs reflect the location and secondary effects (haemorrhage, edema) of the tumor. It has been reported that up to 20% of cats with intracranial neoplasia present for lethargy, inappetence and anorexia without any specific neurological dysfunction. Multiple intracranial **meningiomas** have been reported to occur in cats with a frequency as high as 17%. **Lymphoma, gliomas and choroid plexus tumors** are the other malignancies than can affect the feline brain. Meningiomas in cats represent the neoplastic disorder with a hopeful prognosis since the masses tend to be well encapsulated and easy to isolate during surgery from the surrounding tissue compared to other tumors.

*References available upon request.*

## A NEW APPROACH TO CANINE HYPERADRENOCORTICISM

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### Introduction

Canine hyperadrenocorticism (HAC) is a disease that is likely both underdiagnosed and overdiagnosed, depending on the setting. Cases require much more than an increase in ALP to make a definitive diagnosis, however often the additional testing does not result in a definitive 'yes' or 'no'. Once a diagnosis is made, therapy and dosing requires choices and then monitoring. The options for treatment can include medication, surgery, or radiation therapy. The most common therapy, trilostane, has options in formulation and monitoring.

### Diagnosis of HAC

Diagnosing hyperadrenocorticism (HAC) can be a challenge. Only animals with clinical signs of HAC should be tested. Testing is not recommended in animals with an increased ALP as the sole abnormality, and testing should not be performed in a sick animal. Main laboratory screening tests include the urine cortisol:creatinine ratio, ACTH stimulation test, and low-dose dexamethasone suppression test (LDDS). None of these tests are without false negatives and false positives, therefore case selection is important prior to performing a screening test. The main use of the serum cortisol:creatinine ratio is in its almost 100% sensitivity, however 75% of dogs with non-adrenal illness will have an abnormal result therefore additional testing is required if a positive result is returned.

The LDDS test is generally the preferred screening test for hyperadrenocorticism (HAC), whether due to pituitary dependent hyperadrenocorticism (PDH) or a functional adrenal tumour (FAT), as it is considered the most reliable test. In a normal dog, cortisol levels should be suppressed after administration of dexamethasone for the entire 8 hour test period, typically to less than 27 nmol/L. In dogs with PDH, there may be transient suppression at 4 hours, but cortisol levels will usually be back to, or above, baseline levels at 8 hours. Dogs with FAT do not usually show any suppression of cortisol levels. This test is affected by stress, and therefore it is important to try to minimize stress during the test. A negative result likely rules out HAC, but a positive result may warrant additional testing to confirm the diagnosis. The LDDS test is less expensive to perform than the ACTH stimulation test. Endogenous ACTH measurement is theoretically useful, as a low level fits with FAT and a high level fits with PDH. However problems with the stability of endogenous ACTH make results unreliable, therefore this assay is rarely performed and not usually recommended.

The ACTH stimulation test is often performed as it is currently the test used for monitoring after therapy for HAC. The sensitivity of the ACTH stimulation test is 85% for a dog with PDH and 60% for a FAT, with a specificity of 85-90%. The ACTH stimulation test is best performed in a case with a strong suspicion of HAC, and in those cases it has a high specificity. This means that a positive result is consistent with a diagnosis of HAC, whereas a negative result does not necessarily rule out HAC (high false negative rate).

Due to the lack of availability of synacthen, cortrosyn is now used for performing ACTH stimulation testing. This product has a much higher cost than synacthen, and there are protocols that allow for multiple uses of one vial. In addition, there is now a different protocol for

diagnostic and monitoring ACTH stimulation tests, which reduces the cost for dogs having monitoring ACTH stimulation tests performed.

#### *Protocol for Use of One Vial of Cortrosyn in Multiple Patients*

- Reconstitute Cortrosyn vial which results in a concentration of 100 µg/ml (0.1 mg/ml).
- Aspirate 0.5 ml (50-µg) and 0.25 ml (25-µg) aliquots into PLASTIC labelled syringes.
- Freeze the syringes at -20C in a non frost-free freezer for up to 6 months.
- FOR A **DIAGNOSTIC** ACTH STIMULATION TEST (prior to diagnosis of HAC or hypoadrenocorticism): Administer IM or IV at a dose of 5 µg/kg (round up if needed). Administer IV in dehydrated dogs.
- FOR A **MONITORING** ACTH STIMULATION TEST (after a diagnosis, while receiving trilostane or mitotane): Administer IV at a dose of 1 µg/kg (round up if needed).
- Cortisol levels should be measured prior to injection of Cortrosyn (0 hour), and at 1 hour post administration of Cortrosyn.

Abdominal ultrasound can be quite useful in confirming a diagnosis of both PDH and FAT, especially in cases with concurrent illnesses such as diabetes mellitus, liver disease, or hypothyroidism that may affect the ACTH stimulation test or LDDS test. The presence of bilaterally enlarged adrenal glands which retain the typical adrenal gland shape supports a diagnosis of PDH. The presence of a unilateral adrenal mass with a small contralateral adrenal gland supports a diagnosis of a FAT. In rare instances, there have been reported cases of bilateral FATs, PDH with a concurrent FAT, or a FAT with a contralateral, non-cortisol producing adrenal mass. To complicate things further, benign adrenal nodules are quite common in older dogs, and therefore some dogs with PDH (and therefore bilaterally enlarged adrenal glands) will also have an adrenal nodule present. These nodules rarely result in distortion of the entire adrenal gland, however, whereas a FAT will usually obliterate the adrenal gland. Experience with ultrasonography, along with considering the patient's presentation, differential diagnoses, and ACTH stimulation test/LDDS test results can help to navigate these challenging cases.

### **Therapy and monitoring**

Treatment for hyperadrenocorticism includes trilostane or mitotane, and in specific cases, surgery or radiation therapy. Previously recommended selegiline hydrochloride, ketoconazole or bilateral adrenalectomy should not be used for therapy. There are pros and cons to mitotane and trilostane use. Mitotane is effective in most cases and slightly less expensive, however it more commonly causes side effects. Trilostane is successful at treating most cases of HAC, and it is considered safer than in previous years now that the starting dose is reduced. It is now considered the gold standard of therapy for HAC.

Options for obtaining trilostane include use of Vetoryl, and compounded trilostane. There are now several sizes of Vetoryl available, including 5 mg, 10 mg, 30 mg, 60 mg, and 120 mg. This has reduced the need to use compounded trilostane in order to make small changes in dosage. A recent study assessing the quality of compounded trilostane in the US revealed concerns with trilostane from the evaluated compounding pharmacies. While the percent of drug present in Vetoryl capsules was close to 100% (96.1-99.6%), the range of drug present in compounded trilostane varied from 39-152.6%. Using a criteria of acceptability of 90-105% of expected drug present, 38% of the compounded capsules failed acceptable criteria. Compounded capsules also failed in the time expected to dissolution, with 20% of the capsules not meeting expected

criteria. As the cost of Vetoryl is now competitive with compounded trilostane, given the new sizes of Vetoryl available, it is worth considering the source of trilostane used at your clinic.

Both trilostane and mitotane use require monitoring, most commonly via the ACTH stimulation test. A recent study (Vet Record, 2016) reviewed the ability to monitor control of HAC with pre and post pill cortisol levels, as opposed to the ACTH stimulation test. The study included dogs with PDH and dogs with a FAT. This study concluded that in certain dogs, this method provided better evaluation of clinical control than the ACTH stimulation test. Dechra UK has published recommendations for using the pre-pill cortisol level to monitor HAC. However, it is important to note that this is reserved for clinically well dogs, and calm dogs. Aggressive or overly anxious dogs, along with unwell dogs, should not utilize this method for assessing control of hypercortisolemia. My current recommendation is to consider this method in your well-controlled patients. Benefits include a reduction in cost to the client, and the requirement for a single blood sample if you follow the Dechra UK recommendations.

Surgery (hypophysectomy) or radiation therapy should be considered in cases with neurologic signs or large macroadenomas. Functional adrenal tumours are best treated surgically, however both mitotane and trilostane have shown benefit in treatment. Mitotane is more ideal as it is adrenolytic, however a small study showed good efficacy with either therapy. Chemotherapy can also be considered if a definitive diagnosis is obtained; histopathology is required as cytology of adrenal masses has been shown to disagree with histopathology in a significant number of cases. Complications of untreated HAC are uncommon but include hypertension, UTIs, calcium oxalate urolithiasis, thromboembolic disease, and in 15-20% of cases, neurologic signs due to a large macroadenoma.

### **Client education and communication**

An important aspect of the management of HAC in dogs is early and effective communication with owners. Unlike many other medications, there is no set dose for mitotane or trilostane. In order to avoid possible side effects or over-dosing of these medications, a lower starting dose is advocated. This results in many cases needing serial ACTH stimulation tests in order to determine the optimal dosing. For clients, this is perceived as an increase in cost, an increase in visits to the veterinary clinic, and a delay in control of clinical signs. It is vital to explain the reasoning behind this approach, so that clients understand why this approach is taken, and that although it can result in a longer period of time until clinical control, it is in their pet's best interest.

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## NAVIGATING THE DIAGNOSTIC OPTIONS FOR LEPTOSPIROSIS

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### Introduction

Leptospirosis is a zoonotic bacterial disease caused by a spirochetal bacterium of the genus *Leptospira*. The bacteria are maintained in the renal tubules of the reservoir host and excreted in the urine. The two most familiar clinical syndromes of leptospirosis involve renal or hepatic dysfunction; however, patients may also present with conjunctivitis, uveitis, respiratory distress secondary pulmonary hemorrhage, acute febrile illness, pancreatitis and bleeding tendencies. Rapid diagnosis and treatment is imperative for patient survival, as the prognosis is excellent in most cases if antimicrobial therapy is started early in the course of disease.

### Diagnostic Options

There are several options available for the diagnosis of leptospirosis, and selection of the appropriate test or tests is not straight-forward. The vaccination history of the dog must be considered, along with recent use of antimicrobials. Options for testing include polymerase chain reaction (PCR), which detects DNA of the *Leptospira* organism in samples, and antibody tests, which include the microscopic agglutination test (MAT), an ELISA antibody test, and the new in clinic IgM Witness antibody test (Table 1).

	PCR	WITNESS	ELISA	MAT
Qualitative or Quantitative	Negative or Positive only	Negative or Positive only	Negative or Positive only	Provides Titres
Affected by antimicrobial use	Yes	No	No	No
Affected by vaccination	No	For 1-3 months	For months to years	For months to years
Turnaround time	~24 hours	30 minutes	~24 hours	7-10 days
Performance early in course of disease	Fair, some false negative results	Fair to good, may need repeat test (false negative)	Fair to poor	Fair, often need convalescent titer
Cost	\$\$	\$	\$\$	\$\$\$

Table 1: Summary of readily available tests for leptospirosis in Ontario.

Polymerase chain reaction is a relatively rapid test that detects *Leptospira* nucleic acid in blood, urine, cerebrospinal fluid and aqueous humor, although paired blood and urine testing is the most common method of detection. In the first 10 days of infection, organism numbers are

highest in the blood. After that time, organisms are present in the highest concentration in the urine. In clinical infections the time of infection is unknown, therefore simultaneous testing of blood and urine is recommended to increase diagnostic sensitivity. Vaccination should not affect PCR results, however recent antimicrobial therapy can result in false negatives. I would not recommend performing PCR testing if the dog is currently receiving an antimicrobial used for the treatment of leptospirosis, as the result is likely to be negative. Even if a dog has not had recent antimicrobial therapy, a negative result does not rule out leptospirosis because samples may have been obtained when organism numbers in a sample are low. While a positive result confirms infection, a negative test should prompt additional testing to rule out leptospirosis as there is the possibility of a false negative result. Turn around time for PCR testing is usually approximately 24 hours.

Antibody-based tests (MAT, ELISA and Witness) are not affected by antimicrobial therapy, but are affected by vaccination. The administration of a *Leptospira* vaccine prompts an antibody-based immune response. Therefore, a positive antibody test could be detecting antibodies made in response to the vaccine, as opposed to antibodies made in response to an infection with *Leptospira* spp (a false positive). However, the duration to which a leptospirosis vaccine affects the outcome of an antibody test is test-dependent. The MAT and ELISA tests primarily detect IgG antibodies which persist for months to years, therefore these tests are affected by vaccination for months to years. Use of paired titers 2-4 weeks apart helps to differentiate vaccination from infection in the MAT test, as long as vaccination is not very recent. The new Witness antibody test detects IgM antibodies, which are relatively short-lived after vaccination. Therefore, most dogs will have a negative Witness test within 1-3 months of vaccination. Both the Witness and ELISA *Leptospira* antibody tests are qualitative (provide only a positive or negative result) and are not serovar-specific, while the MAT provides a titre for each *Leptospira* serovar tested. However, the serovar which has the highest titre with the MAT is often not the actual infecting serovar, therefore serovar information from the MAT test is not of much benefit. Knowing the infecting serovar does not affect treatment decisions.

The Witness *Leptospira* Antibody Test was introduced in the spring of 2018 in Canada. This test provides a negative or positive, without information on the infecting *Leptospira* serovar. This test has been used in Europe for several years, and was recently introduced in the USA. This is an in clinic antibody test that detects IgM antibodies. As IgM antibodies are produced early in the course of disease, and their production decreases a few weeks after infection, the test is only useful for acute leptospirosis infections. However, acute infection comprises almost all cases. The test is not affected by antimicrobial therapy. The Witness test is affected by vaccination (false positive), but only for a few weeks after vaccination. Because IgM levels begin to rise a few days after infection, it is possible to have a negative result early in the course of disease (false negative). If clinical suspicion remains high, a repeat test 2-4 days later is recommended. Due to the low cost of the test, this is more feasible than with many of the other tests. The Witness test is an in clinic test that can be performed within 20-30 minutes, and utilizes whole blood, plasma or serum.

The microscopic agglutination test (MAT) involves reacting serial dilutions of patient serum with several live leptospiral organisms, followed by assessment of organism agglutination. The highest serum dilution causing 50% agglutination is reported. False negative results may occur early in the course of the disease. Although a single positive titer can increase suspicion for the disease, it does not always confirm a diagnosis, especially if the dog has been vaccinated for leptospirosis at any time in their life. To increase the diagnostic utility of the test, acute and convalescent antibody testing should be performed (an initial 'acute' titer followed by a

'convalescent' titer two to four weeks after the acute titer). A four-fold increase in titer supports recent infection; convalescent titers in vaccinated dogs are generally stable or decreased after 2-4 weeks, unless vaccination was very recent. Turnaround times are 1-2 weeks.

The *Leptospira* ELISA antibody test is provided at Idexx Canada. This test provides a negative or positive, as seen with the Witness test, without information on infecting serovar. This ELISA test is not affected by antimicrobial use, but is affected by vaccination. The test will be positive in dogs for months to years after vaccination, therefore may be positive in dogs with up to date vaccination, and also potentially in dogs with out of date vaccination. A negative result is useful in ruling out leptospirosis unless the dog was infected very recently, in which case a false negative is possible. A positive result in a dog that has *never* received a *Leptospira* vaccine confirms leptospirosis. A positive result in a vaccinated dog indicates that further testing for leptospirosis is needed (PCR +/- Witness or MAT titres if no antimicrobial used, Witness or MAT titres if antimicrobial used). Turnaround time is around 24 hours.

Diagnostic testing for suspected leptospirosis cases is not straight-forward. Testing guidelines are included to help determine which tests to perform. Given the utility of the Witness *Leptospira* antibody test and its low cost, it is worth using as the primary test for leptospirosis. However, other approaches are needed if the dog has been vaccinated in the previous few weeks.

***Dog that has NOT received antimicrobials and is NOT vaccinated:***

- Start with Witness Lepto if you have it in clinic
- Otherwise *Leptospira* spp. Panel (ELISA and PCR)
- If positive on any, you have your diagnosis
- If negative and clinical suspicion remains high, repeat testing in 2-4 days or do MAT; administer appropriate antimicrobials in case of false negative

***Dog that has NOT received antimicrobials and IS vaccinated in the previous 1-3 months:***

- *Leptospira* spp. PCR
- If positive you have your diagnosis
- If negative and clinical suspicion remains high, do MAT titres, administer appropriate antimicrobials in case of false negative on PCR

***Dog that has received antimicrobials and is NOT vaccinated:***

- Start with Witness Lepto if you have it in clinic
- Otherwise *Leptospira* spp. ELISA
- If positive on any, you have your diagnosis
- If negative and clinical suspicion remains high, repeat testing or do MAT titres

***Dog that has received antimicrobials and IS vaccinated in the previous 1-3 months:***

- Acute and convalescent MAT titres

***Dog that has received antimicrobials and IS vaccinated but not in the previous 1-3 months:***

- Start with Witness Lepto if you have it in clinic
- Otherwise acute and convalescent MAT titres

References available upon request

## NEUROLOGY AND VACCINE IN GENERAL PRACTICE

Carolina Duque, DVM, MSc, DVSc, DACVIM (Neurology) Mississauga-Oakville Veterinary Emergency Hospital, Oakville, Ontario

Adverse reactions to vaccination have been recognized for years in dogs and cats. The consequences of vaccine reactions can be serious. However, in the general healthy population, compared to the risks of not vaccinating, the risks associated with immunization are very small. As vaccines are designed to stimulate an immune response, it is not surprising that a predisposed individual may overreact to vaccination due to the production of inflammatory mediators.

### Should I change my vaccination protocol in the neurological patient?

Patients that are affected by primary epilepsy can be vaccinated with routine protocols. **Primary Epilepsy** is considered when the individual develop seizures between 6 months to 6 years of age and a structural cause of the seizures is not present. The patient is normal between ictal periods.

Patients suffering from **Meningoencephalitis of unknown etiology (MUE)** should not be routinely vaccinated. Evidence exists to show that improved survival of CNS inflammatory disorders in these dogs is linked to immunosuppressive therapy (steroids, cytosar and Azathioprine). When vaccines are administered, the body's and specifically brain inflammatory cytokines increase dramatically. It is reasonable to associate the potential development and aggravation of immune-mediated disorders to diseases in individuals that are genetically predisposed. Age of onset of disease and previous vaccines received are other factors to consider. Research has shown that if a modified live-virus vaccine is given after 6 months of age, it should produce lifelong immunity. If another modified live-virus vaccine is given, the antigens of the second vaccine are mostly neutralized. The serum antibody titer is boosted only transiently and additional immune memory cells are not induced; Thus, annual boosters maybe unnecessary. The University of Wisconsin and the Rabies Challenge Fund Charitable Trust are currently conducting research regarding rabies vaccines. The goal is to extend the legally required interval for rabies boosters to 5 and then 7 years. The study is been conducted according to the USDA's vaccine licensing code (title 9 section 113.209 by Dr. Ronald Schultz).

Another way to protect patients with partial or no immunity is through high vaccination coverage in the population. When large groups of individuals are vaccinated, they create "community immunity".

The more antigens administered in a vaccine, the greater the chance of inducing hypersensitivity. Single agent vaccines are recommended in the vulnerable patient. If the vaccination is necessary in a vulnerable patient,(e.g; post encephalitis) pre-treatment with antihistamines and corticosteroids could be helpful.

## Can vaccines induce seizures?

There is no scientific evidence to support that vaccines induce epilepsy in dogs and cats. It is important to remember that a temporal association between vaccination and the development of a clinical sign does not necessarily equate to a cause and effect relationship between the vaccine and the illness. It is possible that stressful situations or specific medications lower seizure threshold in a patient that is already predisposed. When this is the case the vaccine protocol can be modified accordingly. If a patient develops encephalitis, which occurs rarely as a postvaccinal reaction, seizures could occur as part of the clinical manifestation of CNS inflammation. This is a different scenario to the epileptic patient who experiences repeated seizure activity from genetic predisposition.

## Vaccines reactions and neurological manifestation. Do they really occur?

Suspected neurologic reactions were reported to the Canadian Centre for Veterinary Biologics (CCVB) at a rate of 0.459 per 10 000 doses for dogs, 0.249 for cats and 0.478 for rabies vaccines. This data was collected over a 4 year period (January 2010- June 2014). In some cases, animals that are experiencing an allergic reaction or pronounced inflammatory reaction may be reported by the veterinarian as experiencing tremors, or impaired mental status.

Neurological signs which have been reported as possible adverse vaccine events include head tremor/bobbing, encephalitis, head pressing, convulsion/seizure, rigidity, weakness, impaired mental state, abnormal posture, ataxia, high stepping, recumbency, and altered reflexes.

**Table 1:** Suspected adverse reactions for small animals (dogs, cats) vaccines reported to the Canadian Centre for Veterinary Biologics between 2010 and 2014.

Adverse reactions	Number of reactions per 10.000 doses sold	
	Dogs	Cats
Allergic conditions other than ana- phylaxis	2,663	187
Anaphylaxis, circulatory shock	332	29
Dyspnea	155	110
Vomiting	2,511	1,131
Diarrhea	791	402
Loss of consciousness, collapse	141	8
Pain	240	176
Lethargy	1,923	1,473
Fever	132	438
Malaise	17	17
Vasculitis	13	1
Cough	445	16
Other upper respiratory tract disorders	346	85
Injection site reaction other than sarcoma	1,144	689

Injection site sarcoma	2	148
Death	104	161
Suspected lack of efficacy	228	39
Neurological disorders	459	249
Autoimmune disorders	27	8

The **canine distemper vaccine** has been proven to induce neuro-pathologic damage. The histopathological diagnosis was viral non-purulent meningoencephalitis with severe demyelination in all dog cases. This diagnosis was confirmed by positive immunohistochemical analysis of sections of CNS, indicating those tissues were positive for canine distemper virus (CDV). The findings have been attributed to incorrect vaccine protocols or vaccine alteration after improper storage, but also to host factors (immunodeficiency, maternal antibody interference, vaccination during incubation period) or possible mutation of the wild CDV.

Historically, complications following **rabies virus** vaccination have received the most attention. MLV (modified live vaccines) are no longer used in the US for this reason. This may relate to the overt nature of neurologic illness and decreased immunocompetence of the CNS against MLV agents. Vaccine-induced rabies in dogs and cats following MLV vaccination begins with paralysis in the inoculated limb within 7 to 21 days and progresses bilaterally and in an ascending fashion.

Veterinary clinicians are seeing an increase in neurological diseases associated with vaccinosis. The Purdue University School of Veterinary Medicine conducted important studies to determine if vaccines alter the immune system of dogs. The study showed that blood from all of the vaccinated dogs contained significantly elevated concentrations of antibodies directed against proteins. One of the biochemical marker proteins that generated reactive antibodies in the vaccinated population included Anti-cardiolipin. Interestingly this component is frequently found in patients with systemic lupus erythematosus, and other CNS autoimmune diseases in humans. Guillain-Barré syndrome (GBS) is the most common immune-mediated **polyradiculoneuropathy** considered as a severe adverse event following immunization in human. Quiroz-Rothe and coworkers reported a similar syndrome in a 3.5 year-old male Rottweiler dog after receiving an inactivated rabies vaccine (Rabdomun, Pfizer) and other inactivated tetravalent vaccine (Tetradog, Merial) within three months. The presence of antibodies against myelin successfully proves that the case represented a vaccine reaction. The author has observed polyneuropathies presumably associated with vaccines in dogs in cats that are self limiting. In these cases the clinical signs have been noted within 3-10 days from vaccination.

A case of **eosinophilic encephalitis** was also observed by the author in a Chihuahua. The patient developed CNS inflammation in two isolated occasions after 24 and 48 hrs of immunization. CSF and MRI were performed during the second incident to confirm the diagnosis.

Important data about the administration of vaccines against human papilloma virus (HPV) causing severe somatoform and dysautonomic syndromes have been recently published in 2017.

Although it is impossible to completely prevent adverse reactions to vaccines, data supports the view that the benefits of core vaccination against infectious diseases outweigh the low risk of an adverse reaction in the healthy population. Patients with immune disease should be evaluated in individual bases to design an appropriate protocol.

*References are available upon request.*

## PRACTICE PEARL: TOPICAL THERAPY FOR CANINE PYODERMA - REDUCE, REDUCE, REDUCE ANTIBIOTIC USE!

Douglas J. DeBoer, DVM, Diplomate ACVD

Staphylococcal skin infections are increasingly difficult to treat in dogs, because of antimicrobial resistance. Unfortunately, many years of treating pyoderma with repeated courses of antibiotics has led to the recent and increasing appearance of multi-drug-resistant staphylococci. In some areas of the world, more than 50% of skin cultures performed at dermatology specialty practices are methicillin-resistant staphylococci (MRS). These strains include the methicillin-resistant *Staphylococcus pseudintermedius* species (canine infections, referred to as “MRSP”) or methicillin-resistant *Staphylococcus aureus* species (human infections, referred to as “MRSA” and, fortunately, much less common). Veterinarians should endeavor to use correct terminology when discussing these infections with clients; incorrectly referring to a canine MRSP infection as “MRSA” may be alarming to the client. If laboratory testing indicates the presence of MRS, the isolate will be *clinically resistant to all penicillins and cephalosporins*. In addition, some MRS will also be resistant to other antibiotic classes, such as fluoroquinolones or clindamycin.

### What’s the Practical Significance?

- If you treat a dog with staphylococcal pyoderma with a beta-lactam antibiotic (cephalosporin or amoxi/clav) and there is limited or no response, *culture and susceptibility testing is now mandatory*. If you do identify an MRS organism, you should obtain a staph speciation test to determine if the organism is a human or veterinary strain.
- Fortunately, many veterinary strains of MRS are still susceptible to routine antibiotics such as trimethoprim-sulfamethoxazole, clindamycin, or a fluoroquinolone such as enrofloxacin or marbofloxacin. However, it is important to note that it is impossible to predict with any certainty which antibiotics are indicated without performing a susceptibility test. Empirical “antibiotic hopping” is hazardous, as with each cycle of treatment, multiple drug resistance becomes more likely.
- If you have a patient with MRSP (i.e., the canine strain) in your hospital, you need not have the dog under full isolation conditions, but you should isolate the patient to the extent you can and eliminate traffic from this patient to other dogs in the clinic, especially waiting room, surgery, and critical care areas. Especially important to practice good hygiene measures.
- If the organism is a methicillin-resistant, human-origin *S. aureus* (MRSA), the owner should be notified of this fact so they can discuss the situation with their own health care provider, and gloves should be worn when examining the patient. If hospitalized, consider full isolation. This patient is a potential human health hazard and should be considered so until all lesions have completely resolved. The concern here is that without proper precautions, the MRSA could colonize the owner, you, your staff, or others.
  - It is important to understand that merely becoming colonized with MRSA is not inherently dangerous.
  - MRSP or MRSA strains are not really more ‘virulent’ than other staph strains; they are just more difficult to treat.

### Exploring Solutions: Patient Treatment

The emergence of MRS in the veterinary world suggests that we must redouble our efforts to use antibiotics wisely and judiciously and reconsider all efforts to use alternative, nonantibiotic treatments, if possible, especially in the face of recurrent infections.

- Increasingly, dermatologists understand that it is very possible to *eliminate* active superficial staphylococcal infections (even MRS) from the skin by using topical products as the primary treatment without antibiotics. For primary treatment of an existing superficial pyoderma, **daily treatment is necessary until the infection is cleared, which typically takes 3 weeks or more.**
- Antimicrobial topicals are also the first line of defense for *prevention of relapse* in patients with recurrent pyoderma where the cause cannot be found or treated. For preventive maintenance, topicals are typically used 2-3 times weekly.
- Spray-on, wipe-on, leave-on, or mousse products are often preferable to shampoos for frequent or long-term use, as they both provide residual effect and improve client compliance. Whether used daily as primary treatment or every few days as preventive maintenance, the following ingredients are useful in topical products:
  - **Chlorhexidine** is among the best antiseptics. It provides good antibacterial and anti-yeast activity, and good residual activity. Chlorhexidine can cause severe eye reactions, so be very careful around the facial area. Typically, topicals for the skin are 2-4%; there is not convincing evidence that within this range, a higher concentration is better. There is also good evidence that chlorhexidine is synergistic with miconazole for both bacteria and fungi. Thus, the best single antimicrobial shampoo to have in your clinic is probably a 2% chlorhexidine/2% miconazole formulation.
  - **Benzoyl peroxide** is a good bactericidal antiseptic. It also has excellent keratolytic properties, thus can penetrate into the hair follicle and break apart any keratin plugs. This makes it useful in deep pyoderma or in demodicosis. However, it can be quite drying and irritating, so should be reserved for short-term use (4-6 weeks maximum). It will also bleach fabrics in the home.
  - **Iodine, ethyl lactate, triclosan**, and other disinfectants are less effective.
  - **Mupirocin** is a topical antibiotic ointment with excellent activity against staphylococcal organisms. It can be used for localized or focal pyoderma. The common “triple antibiotic ointment” combinations are much less effective.
  - **Nisin** is available in wipe-on formulations. This is convenient for areas such as face or tail-folds, or easy-to-treat bare areas of the skin such as the ventral abdomen.
  - Daily wipe-down (of the pet) with **alcohol-gel hand sanitizer**. Though unstudied, this treatment is reportedly very effective and is used commonly in the Nordic countries.
  - **Use of oxidizing disinfectants** such as very dilute sodium hypochlorite solutions (“bleach baths”) has become very popular in human atopic dermatitis recently to limit bacterial colonization of skin. Veterinary products with similar actions are gaining popularity with some dermatologists (e.g., Vetericyn), although critical studies are lacking.
  - **Peroxide** is an excellent oxidizing disinfectant. The most recently popular products contain “accelerated hydrogen peroxide,” which is simply hydrogen peroxide with added stabilizers and surfactants to enhance its efficacy. We await critical “on-patient” studies of efficacy for these products.

### Exploring Solutions: Sanitation and Public Health

MRS is spreading very fast in both the human and veterinary world. It is time to start taking every precaution to prevent transmission of MRSP strains in your clinic, and prevent colonization of humans by MRSA. Key measures for sanitation have been developed by expert panels, and include:

- Handwashing and disinfection; wearing gloves when appropriate
- Protective clothing, with frequent laundering
- Cleaning and disinfection of premises
- Education of staff and pet owners
- *Explore these very valuable online resources!*
  - Free journal issue containing excellent review articles. In particular see p94, Weese JS, Methicillin-resistant staphylococcal infections in pets [overall review]; and p90, Mateus A, Stewardship of antimicrobials and hygiene protocols in practice; are we there yet? [review of hygiene procedures currently recommended].  
[http://www.fecava.org/sites/default/files/ejcap\\_21\\_1.pdf](http://www.fecava.org/sites/default/files/ejcap_21_1.pdf)
  - Recent international expert panel recommendations for diagnosis and treatment of pyoderma, published April 2014. Great reference.  
<http://onlinelibrary.wiley.com/doi/10.1111/vde.12118/abstract> (click on “get PDF” on the right to see the whole article)
  - A superbly interesting and entertaining blog site about zoonoses; provides great information. Recommended lunchtime reading! Click on “Resources-Pets” to access downloadable client handouts on MRS, etc.  
[www.wormsandgermsblog.com](http://www.wormsandgermsblog.com)

## ABDOMINAL AND THORACIC FOCUSED ULTRASONOGRAPHY TECHNIQUES FOR THE EMERGENT PATIENT

Jennifer Kyes, Bsc. DVM, DACVECC

### The Abdominal FAST (AFAST) exam

The AFAST can detect small volume abdominal fluid and retroperitoneal effusion as well as identifying pleural and pericardial effusion through the diaphragmatic-hepatic view.

Patient positioning should be in right lateral. Dorsal recumbency should not be used as it is not validated for the AFAST-applied fluid scoring system, carries a higher risk for the compromised trauma patient and increased stress for the respiratory-compromised patient.

AFAST views (Figure 1) are in longitudinal orientation and include the:

- Diaphragmatico-hepatic (DH) view
- Spleno-renal (SR) view
- Cysto-colic (CC) view
- Hepato-renal (HR) view

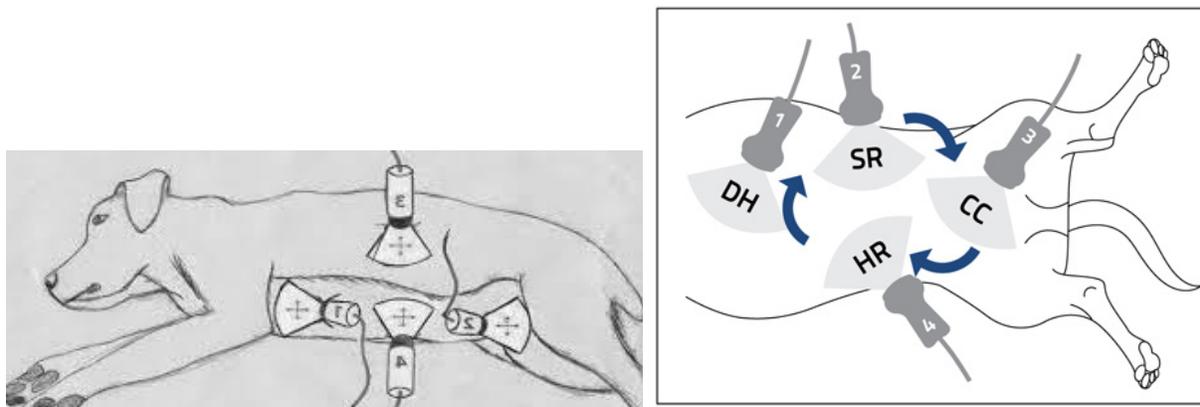


Figure 1a and 1b: AFAST views with patient in right lateral recumbency and with patient in dorsal recumbency.

#### Diaphragmatico-hepatic (DH) view

The probe should be in a longitudinal plane and placement is immediately caudal to the xiphoid process. View the gallbladder adjacent to the diaphragm and fan the probe to visualize small fluid volumes between liver and diaphragm and between the liver lobes themselves. Always take the opportunity to view the thorax for pleural or pericardial effusions.

#### Spleno-renal (SR) view

The SR view includes the spleen and the left kidney. The probe should be in a longitudinal plane with placement parallel to the spine just caudal to the costal arch because the kidney is more recognizable in longitudinal orientation. Fluid is classically identified as anechoic (black) triangles formed between the spleen and colon.

#### Cysto-colic (CC) view

The probe is in a longitudinal plane and placement into the gravity dependent pocket that forms between bladder and ventral body wall. The classic CC view includes imaging of the urinary bladder but does not actually image the colon because it is air-filled and will obscure imaging.

Fluid can be seen as a small anechoic (black) triangle between the urinary bladder apex and the body wall.

#### Hepato-renal (HR) view

The placement of the probe is transverse and just ventral to the umbilicus to image the most gravity dependent area and differentiate small intestinal loops from free fluid.

This view includes loops of small bowel and occasionally the spleen. Ironically, the liver or right kidney are not the target organs nor are they aggressively searched for in this view. The right kidney is generally more difficult to image because of its more cranial location under the rib cage. Fluid can be seen as a “rabbit ear” sign for large volume effusions created by small intestine and omentum wafting in the free fluid.

#### The Thoracic FAST (TFAST) exam

The TFAST can detect the presence of a pneumothorax, pleural and pericardial effusions. A stable patient should be placed in right lateral recumbency and sternal or standing in all compromised patients. Views are taken in a longitudinal orientation and consist of five points (figure

- Right and left chest tube sites (CTS)
- Right and left pericardial sites (PCS)
- Diaphragmatico-hepatic view (DH)

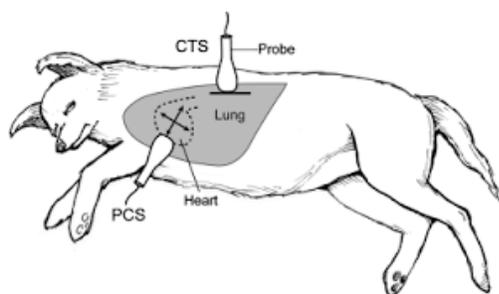


Figure 2a and 1b: TFAST views in right lateral recumbency and standing.

#### Chest tube site (CTS)

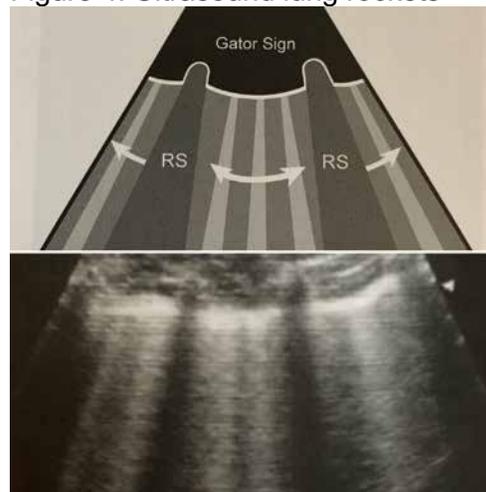
The CTS view is defined as directly dorsal to the xiphoid process between the 8<sup>th</sup> and 9<sup>th</sup> intercostal rib spaces (ICS). If necessary, move an intercostal space cranially to avoid interference from the diaphragm and liver if necessary. The gator sign (Figure 3) is the pulmonary-pleural line interface. The gator's eyes are the 2 rib heads and the gator's nose is the ICS. The bright white line is the pulmonary-pleural line. This is where the lung should glide to and fro with inspiration and expiration along the thoracic wall. The presence of a glide sign rules out a pneumothorax.

Figure 3: Gator Sign



Ultrasound lung rockets (ULR) (Figure 4) are also called B-lines and refer to sites along the lung periphery where water or fluid is immediately adjacent to air. Although ULRs fill the entire ultrasound screen, they only represent a fluid-air juxtaposition within the first 1-3mm of lung's surface. ULRs are easily recognizable and rapidly rule out a pneumothorax. Regional distribution patterns of ULRs can be used to diagnose lung conditions such as edema.

Figure 4: Ultrasound lung rockets



#### Pericardial site (PCS)

The PCS are bilaterally applied in the region of the 3<sup>rd</sup>, 4<sup>th</sup> and 5<sup>th</sup> ICS at the costochondral junctions. The marked should point towards the elbow and then rotate the marker toward the spine. The depth should be adjusted to view the complete heart. Identifying the cardiac chambers prevents mistaking normal chambers for pleural or pericardial effusion. The “mushroom” view allows for subjective evaluation of contractility and volume status. Pleural and pericardial effusion in the majority of trauma cases will be anechoic but other effusive conditions may have various degrees of echogenicity.

#### Diaphragmatico-hepatic (DH)

The DH view begins with placing the probe at the subxiphoid location and having the gallbladder imaged next to the diaphragm. Increase the depth so the thorax is approximately 25-33% of the far field, to image into the pleural and pericardial spaces. The DH view is advantageous as a window into the thorax as it has less air interference than the paracostal view.

#### References

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## RABIES EXPOSURE? TOP 5 SCENARIOS & WHAT TO DO

Maureen E.C. Anderson, DVM, DVSc, PhD, Dip. ACVIM

### When to call...

1) The public health unit (PHU): If there is any concern that you, your staff, a client or anyone else has had contact with a suspicious animal's saliva on broken skin or a mucous membrane, call the local PHU first. This includes but is not limited to any scenario involving an animal bite. Also consider if anyone has been handling the animal's mouth, orally medicating or hand-feeding the suspicious animal, or whether the animal was in contact with young children or anyone who cannot provide a reliable history. It is up to the PHU and the individual's physician to determine if a person is at risk, not you as a veterinarian. *If in doubt, call.*

2) OMAFRA (via the Agricultural Information Contact Centre at 877-424-1300): If you have done a risk assessment for a patient and determined that there is a significant risk that the animal was exposed to rabies, one of three things needs to happen: The suspicious animal needs to be tested, the at-risk animal needs to be vaccinated and confined, or the at-risk animal needs to be vaccinated and observed. Call OMAFRA if either testing or confinement is required, for help with sample submission logistics or providing guidelines to the animal owner. Also call OMAFRA for any unusual cases in which you are unsure of the risk assessment – we can help!

3) Animal control: If a client calls you saying that there is a live animal (typically wildlife) either in distress or acting in an abnormal threatening manner (i.e. not just guarding food or a den), they must be directed to an animal control agency (private or public). There is no other agency that is equipped to handle or otherwise deal with live animals. Local police services should only be called to dispatch an animal if there is no other option available and a significant animal welfare concern. Human safety is of primary importance in any such scenario.

4) Ministry of Natural Resources and Forestry (MNR, via their Rabies Information Line 888-574-6656): If you or a client has the carcass of a suspicious animal (other than a bat) that has clearly not exposed any person or domestic animal, the MNR may be interested in collecting it for surveillance testing, depending on the species, circumstances and geographic area.

### When NOT to call OMAFRA

- Domestic animal altercations (dog vs dog, cat vs cat), unless you have reason to suspect one of the animals is clinically rabid.
- Low risk incidents (see Guide) in which the risk of exposure to rabies virus is negligible (e.g. encounter with normal wildlife in a low-risk area with no visible wounds to the pet).
- When you believe an animal should be tested for rabies because a person is concerned about their own exposure (call the PHU instead).
- A currently vaccinated animal is potentially exposed to rabies but is given a rabies booster within 7 days and will be observed at home by the owners for 45 days. Testing of the suspicious animal (if available) in this scenario is not strictly necessary, unless the observation period will be problematic for some reason. If neither testing nor confinement are required, OMAFRA staff do not need to be informed / involved.

**TOP 5 rabies exposure scenarios****Cat killed a bat!**

Bats are considered a high-risk species for rabies, as the virus is endemic in bat populations throughout Ontario, so the risk is not dependent on the geographical location. A bat bite can be virtually impossible to find or rule out on a pet, so any direct contact with a live bat is considered a potential rabies exposure.

- 1) Determine the likelihood that the cat actually killed the bat. If the bat was found desiccated in an area where the cat does not go, or found hanging on a wall, it is less likely that the cat had direct contact. Simply being in the same house as a bat does not constitute exposure.
- 2) Make sure the bat is dead. Don't be fooled. If there is any doubt, handle the bat with precautions as though it were alive until euthanol can be administered (intraperitoneal). Once it is dead, keep it cool or ideally frozen to preserve the sample
- 3) Contact OMAFRA for instructions on how to submit the bat for testing. The entire bat is sent to the lab, so no sampling is required, and shipping and testing costs will be covered.
- 4) Vaccinate the cat immediately if it is not currently vaccinated, or if test results won't be available for more than 7 days after the exposure occurred.

**Dog fighting with a raccoon!**

There are currently specific areas of Ontario where there is a higher risk of raccoon rabies (see the most recent case map on the OMAFRA rabies website), but even in these areas the majority of animals are not carrying rabies. Evidence of abnormal raccoon behaviour AND a wound on the dog / suspicion of mucous membrane contact with saliva (e.g. a prolonged fight) is typically sufficient to warrant testing or confinement/observation, but in very high-risk areas one or the other may be considered sufficient. Particularly in urban areas, raccoons can be quite brazen, so it often takes some additional probing of the owner to determine if there is really any evidence that the raccoon was abnormal.

- 1) Examine the dog for any wounds that may have been caused by the raccoon, particularly by its teeth. Large dogs with high prey drive can often catch, shake, and kill wildlife so efficiently that the risk of exposure to the animal's saliva is considered negligible. Remember that exposure to blood from the raccoon is not a risk for rabies transmission, but a bleeding wound on the dog that may have come in contact with the raccoon's saliva is.
- 2) If the dog is considered exposed, contact OMAFRA for instructions on how to submit the raccoon for testing. The head of the raccoon will need to be removed, typically by a veterinarian or technician at the clinic – this is most easily done before the carcass is frozen. Shipping and testing costs will be covered.
- 3) Vaccinate the dog immediately if it is not currently vaccinated, or if test results won't be available for more than 7 days after the exposure occurred.

**Dog attacked by a coyote!**

While all mammals are susceptible to rabies virus, some species appear to be more prone to infection than others, likely due to a number of factors ranging from genetics to population dynamics and interactions. Coyotes are considered a low-risk species in Ontario; even in the

1970s and 1980s when the infection pressure was very high in the province, rabid coyotes were very uncommon. It is also not considered abnormal behaviour for coyotes in southern Ontario to attack small dogs, and sometimes even larger dogs. Therefore, even if the dog sustains a bite wound, unless there is evidence that the coyote was abnormal (e.g. showing neurological signs) this is considered low risk for rabies transmission. If the dog is not currently vaccinated, it should be vaccinated as soon as it is medically appropriate, based on the attending veterinarian's assessment (i.e. if the wounds are severe, vaccination should be postponed until the dog is sufficiently recovered to respond normally to the vaccine).

### **Cat came home with a bite wound from...something!**

The most common cause of a bite to a cat is another cat, and rabies is not known to circulate within the feline population, even in feral cats. Wounds from smaller animals that would typically be hunted by cats are also considered low-risk. Even in endemic areas, the vast majority of wildlife are not carrying rabies, therefore in the absence of evidence that the offending animal was a reservoir species or abnormal in some way, a bite from an unknown animal is considered low risk for rabies transmission. If the cat is not currently vaccinated, it should be vaccinated as soon as it is medically appropriate.

### **Cat showing neurological signs – does it need to be tested?**

Cats that hunt can unknowingly be exposed to bats, even if they are indoors, so rabies is a differential for any acutely neurological cat (<7-10 days) in which there is no other apparent cause. Owned cats have often had extensive contact with people, and if there is a risk of human exposure the first call must be to the PHU. If there is known contact or sharing of food / water with another pet, this may also be a potential exposure, and OMAFRA can help facilitate testing as described above. If the cat is the only household pet, or was a stray with no specific domestic animal contact or human exposure, then testing is typically not necessary.

### **What to do in a pinch**

Sometimes there just isn't an opportunity to check the website or make a phone call when the client and animal are right there in front of you, or it's late at night and you need to know what to do even if it's not a true emergency. The important things to remember at these times are:

- 1) vaccination is recommended for any exposed animal (unless the exposed animal has also bitten a person) especially if the animal's vaccination status is already out-of-date
- 2) preserve the required samples from any animal that may need to be tested for rabies (e.g. put the carcass somewhere cold where it won't be scavenged; do not release bats)
- 3) ensure any risk to people is addressed by the PHU (each has a 24-hour contact number)
- 4) minimize the number of people/animals in contact with any animal that may be infected with or exposed to rabies, until a complete risk assessment can be completed.

### **Resources**

- OMAFRA Rabies website (for public and vets)  
[www.omafra.gov.on.ca/english/food/inspection/ahw/rabies.htm](http://www.omafra.gov.on.ca/english/food/inspection/ahw/rabies.htm)

# Rabies Risk Assessment Guide

Veterinarian receives report of domestic animal exposure to a potentially rabid animal (could be report on a single animal or herd).

**YES** Was there any human exposure? (bite, scratch, orally medicating a neurologic animal?) **NO**

**Refer to local public health unit for risk assessment on human exposure. (risk assessment for animals will occur after public health assessment)**

Did a category 2 or higher exposure occur? (see table below for details)

Was the animal displaying abnormal behaviour or neurologic signs consistent with rabies? **NO** Is the offending animal a rabies reservoir species? (bat, raccoon, skunk, fox) **YES/MAYBE** **NO**

Does local rabies epidemiology support an increased risk of rabies in the offending species? **YES**

**Increased Risk. Consider testing or post-exposure management. Recommend rabies vaccination within 7 days of incident.**

What is the local epidemiology of rabies? Did the exposure occur in a higher-risk area for the reservoir species? **AND** Was the animal displaying abnormal behaviour or neurologic signs? **AND** Was the offending animal unprovoked? Was the offending animal's behaviour abnormal considering the situation?

**Negligible Risk. Recommend rabies vaccination if animal not up-to-date.**

**NO** **NO to all** **YES to one or two** **YES to all**

**Negligible Risk. Recommend rabies vaccination if animal not up-to-date.**

**Increased Risk. Consider testing or post-exposure management options if testing not available. Recommend rabies vaccination within 7 days of incident.**

**Significant Risk. Recommend testing if available and post-exposure management options if testing not available or tests positive. Recommend rabies vaccination within 7 days of incident.**

[ontario.ca/animalhealth](http://ontario.ca/animalhealth)

## WHO CATEGORIES

- Category 1:** Touched or licked on intact skin by offending animal.
- Category 2:** Nibbled, minor skin lesions in contact with saliva of offending animal.
- Category 3:** Bitten, deep scratches, broken skin or mucus membranes in contact with saliva of offending animal.

Ministry of Agriculture, Food and Rural Affairs



## **GRAIN-FREE DIETS AND DILATED CARDIOMYOPATHY, AN EMERGING PROBLEM?**

**Jonathan Lichtenberger, DVM, MSc, DACVIM (Cardiology)**  
**Animal Health Partners, North York, ON**

Dilated cardiomyopathy (DCM) is one of the most common acquired heart disease in dogs. DCM is a primary myocardial disease characterized by dilation and reduced systolic function of one or both ventricles. In most cases, the etiology of DCM is genetic, and large and medium sized dogs are most commonly affected, with breeds such as the Doberman Pinscher, Irish Wolfhound, Great Dane, Newfoundland, Cocker Spaniel being overrepresented. However, DCM can also be the result of a variety of other myocardial insults from infectious diseases, toxins, or nutritional deficiencies (including deficiencies in taurine or L-carnitine).

Over the past years, some cardiologists have observed a subjective increase in the incidence of DCM in breeds of dogs that were not typically affected by DCM. It also appears that many of these dogs were being fed grain-free diets, boutique or exotic ingredients diets. In July 2018, the US Food and Drug Administration published a warning stating that most of the cases reported to them were being fed diets that list potatoes or multiple legumes, such as peas, lentils, other “pulses” (seeds of legumes), and their protein, starch and fiber derivatives early in the ingredient list, indicating that there are the main ingredients. High levels of legumes or potatoes appear to be more common in diets labeled as “grain-free”.

### **By which mechanism can grain-free diets lead to DCM?**

Some dogs have low blood taurine levels. This was the case for most of the patients I have personally seen so far. Taurine is an abundant free sulfur amino-acid which participates in many myocardial functions, including osmoregulation, free-radical scavenging, and modulation of contraction strength through regulation of calcium concentration. In 1987, it was demonstrated that inadequate provision of dietary taurine leads to systemic taurine deficiency in all cats and that a high percentage of them would acquire DCM. This is not true for dogs that appear able to synthesize sufficient taurine from dietary methionine and cysteine, and taurine supplementation of commercial diets has only been mandatory in cats. Despite that, some dogs still develop diet-induced taurine deficiency for reasons that are not fully understood yet, but may include the followings: insufficient taurine synthesis, extraordinary loss of taurine or its precursors in urine, accelerated gastrointestinal loss of taurine in bile acid conjugates, or poor bioavailability of sulfur amino-acids. These possible mechanisms can be influenced by dietary factors such as the protein source, fiber type and concentration, cooking and processing methods, or individual dog characteristics such as the breed, or energy requirement.

The problem is likely even more complex than that: some cardiologists report that some dogs eating grain-free diets appear to develop DCM even without evidence of taurine deficiency (i.e. with normal taurine blood levels) and cardiac morphology and function appear to improve with diet change. A recent retrospective study from the Cardiology group at North Carolina State University identified that dogs with DCM fed a grain-free diet had measured echocardiographic parameters suggesting more advanced disease than dogs with DCM fed non-grain-free diets. This suggests that the pathophysiology of DCM may be enhanced by grain-free diets in some dogs. The mechanisms by which grain-free diets may lead to DCM in some dogs in the absence of taurine deficiency are unknown.

Another consideration is that many grain-free, boutique or exotic diets are made by small pet food manufacturers and some of them may have inadequate nutritional expertise or quality control, leading to nutritional imbalances or deficiencies.

### **What should you do if one of your patients is diagnosed with DCM?**

There is no consensus on how to proceed, and the following recommendations are based on personal and other expert opinions.

First it is important to obtain a detailed diet history, to identify risk factors. In addition to standard diagnostics (including an echocardiogram) and treatments for myocardial diseases, I also recommend measuring blood taurine levels, especially in dogs that do not belong to breeds genetically predisposed to DCM or in dogs genetically predisposed to DCM and fed grain-free or atypical diets. In theory, both whole blood and plasma taurine levels should be measured simultaneously. For cost reasons, I often only measure whole blood taurine, which is less subject to change than plasma concentration during the postprandial period or with food deprivation. Moreover, white blood cells are rich in taurine and a plasma taurine level can be falsely normal if the buffy coat was disturbed during sample preparation. I prefer to use the Amino-acid laboratory of the University of California, Davis. All instructions are provided on their website:

<https://www.vetmed.ucdavis.edu/labs/amino-acid-laboratory>

If my degree of suspicion for a diet-induced DCM is high, I usually initiate taurine supplementation after blood sampling, while waiting for the results (typically received within 1-2 weeks). If the dog is being fed a grain-free diet, I also recommend changing diet, progressively once the dog's congestive heart failure is being controlled if this was the case. It is important to choose taurine supplements that match their stated contents and are readily available for absorption. This is the case of taurine supplements from NOW foods (500 mg capsules). I typically recommend a dosage of 1000 mg of taurine every 12 hours in dogs over 15 kg and 500 mg every 12 hours in dogs less than 15 kg. When making diet change, I recommend choosing a diet that does not contain legumes or potatoes as major ingredients or exotic sources of proteins. Ideally, the diet should meet the WSAVA Global Nutrition Assessment Guidelines published as consensus by specialized veterinary nutritionists.

<https://www.wsava.org/WSAVA/media/Arpita-and-Emma-editorial/Selecting-the-Best-Food-for-your-Pet.pdf>

If taurine levels come back low, supplementation with taurine is continued indefinitely, even if the diet has been changed. I typically recommend a recheck echocardiogram at 3 months and if no significant improvement in cardiac morphology and function is seen at that time, I recommend rechecking whole blood taurine levels to confirm adequate supplementation. It may take up to 6 or even 12 months for visible improvement to occur. In some dogs, improved cardiac morphology and function are substantial and cardiac medications can be progressively decreased or even discontinued. If taurine levels are normal, I may still continue supplementation if the breed is atypical for DCM. Some cardiologists add L-carnitine supplementation (50 mg/kg every 8 hours with food). Unfortunately, deficiency in myocardial L-carnitine is difficult to prove since it requires myocardial biopsies and plasma levels of L-carnitine are often normal in deficient dogs.

More data are necessary to better understand the potential link between some cases of DCM and grain-free diets or atypical diets. It is important to report cases to the FDA by following the information found here:

<https://www.fda.gov/animalveterinary/safetyhealth/reportaproblem/ucm182403.htm>

### **What should you do if one of your patient is being fed a grain-free diet and does not show signs of DCM?**

This is a delicate question and again, there is no consensus on how to proceed. I typically recommend avoiding diets with ingredients of concern as mentioned in the FDA warning, unless medically-justified (which is rarely the case). I also recommend using a diet meeting the WSAVA Global Nutrition Assessment Guidelines as indicated above.

If the diet cannot be changed or if there is a high level of concern (for example, another dog of the household eating the same diet was affected by DCM), a taurine level measurement and screening echocardiogram for DCM can be considered.

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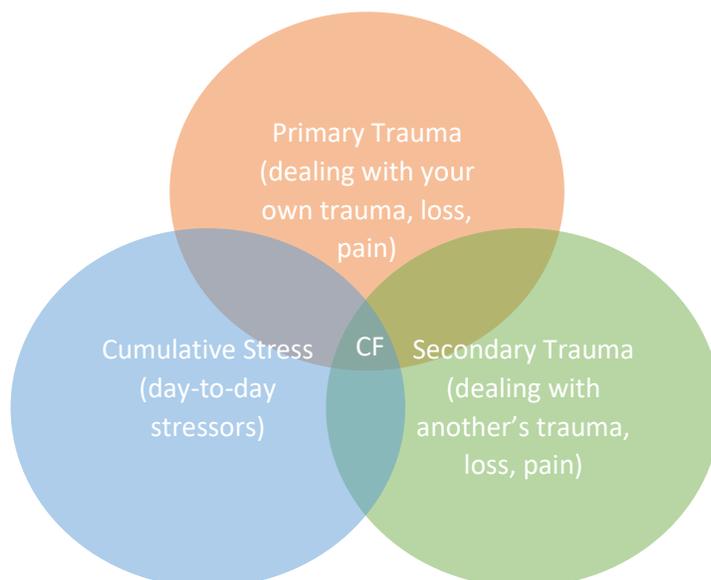
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## “IF YOU CAN’T HELP YOURSELF, HOW THE HECK YOU GONNA HELP SOMEBODY ELSE?”

Sarah Bernardi; RSW, MSW

### Trauma and Compassion Fatigue (CF)



(Voges & Romney, 2003; Gentry, 2005)

Compassion fatigue is considered a form of Secondary Traumatic Stress, however cumulative stress and primary trauma also contribute to its onset.

### Empathy

- ❖ As a person in the helping profession, empathy exists at the center of your work.
- ❖ It is the tool we possess that allows us to understand and bear the suffering of another being.
- ❖ The ability to be empathetic is necessary, in that it allows us to relate to someone’s situation and help them relieve their pain.
- ❖ It is however, a double-edged sword. Too little empathy doesn’t allow for best practice in our work, and too much impacts our own personal mental health and well-being. For this reason, it is believed that empathy exists at the core of compassion fatigue.

### Defining Compassion Fatigue

- ❖ *“Compassion Fatigue has been clinically defined as the formal caregiver’s reduced capacity or interest in being empathic or “bearing the suffering of clients” and is the behavioral and emotional state that results from knowing about a traumatizing event experienced by another person.” (Boscarino, Adams & Figley, 2010)*
- ❖ Exhaustion/fatigue on a biological, social and psychological level and a form of Secondary Traumatic Stress- second -hand exposure to traumatic events

## **Burnout**

- ❖ *“A state of physical, emotional and mental exhaustion caused by long-term involvement in an emotionally demanding situation.” (Pines & Aronson, 1988)*
- ❖ Characterized by hopelessness and inefficiency
- ❖ Burnout is gradual, cumulative process that eventually leads to feeling of ineffectiveness or disconnect to one’s work role. It is often brought on by organizational concerns (i.e. policies and procedures.)
- ❖ CF has a faster onset of symptoms and can be triggered by a single traumatic event.

## **Vicarious Trauma (VT)**

- ❖ A type of Secondary Traumatic Stress that results from exposure to a client or patient’s traumatic material.
- ❖ It affects the way you view your sense of self, relationships and the world. Your symptoms may even be similar to that of the client/patient that you work with.
- ❖ Characterized by a profound, negative shift in world view.

## **Breaking Down Compassion Fatigue- A Closer Look at Moral Stress**

- ❖ Moral Stress (also referred to as moral distress. This term refers to “no win” situations. By definition moral distress is when an individual knows that ethical principles are at stake in a situation but cannot take a course of action to address them due to external constraints.

- ❖ Moral stress in relation to CF/VT shows that their onset is not so clearly defined. CF/VT isn't just about being exhausted, it is a "conflict between our deepest values and the work we are required to do." (TEND, 2018)
- ❖ Moral stress involves emotional consequences. Many find it difficult to let go of these "no win" situations at the end of the day and allows for feelings like guilt, anger and anxiety to manifest.

### The Cumulative Stress Factor

- ❖ Cumulative stress contributes to the onset of CF.
- ❖ For the Veterinary profession stressors have grouped into 4 categories:
  - Occupational (ex. workload, work hours)
  - Environmental (ex. physical environment, interpersonal relationships)
  - Patient/client related (ex. moral stress/decision making, unexpected outcomes)
  - Personal (ex. finances, expectations of performance)

### The Importance of Coping with Stress

- ❖ The use of coping strategies is key to stress management and in turn, addressing CF.
- ❖ Bartram & Gardner highlight two types of coping styles: Adaptive & Maladaptive
  - Adaptive:
    - Targets **long-term** relief of stress
    - Defining the problem: what is the stressor? (i.e. client complaint)
    - Breaking it down to a specific situation: what can be changed and what cannot? If aspects of the situation can be changed then we utilize **(problem-focused)** strategies that actively deal with the problem. If we cannot change certain factors of this situation it is about reframing our way of looking at it **(emotion-focused)**.
    - Refrain from **avoidance** tactics
  - Maladaptive:
    - Focuses on short-term relief
    - Has consequences: may affect physical and psychological health in the long term

Examples of Maladaptive strategies: drug/alcohol misuse, engaging in harmful behaviors to relieve negative emotion, poor nutritional habits, procrastination, Frustration and aggression towards others, self-isolation...etc.

## Signs of Compassion Fatigue

This is not an exhaustive list, and it is important to note that you should look for symptoms that are abnormal to you:

- ❖ **Physical** -poor sleeping habits, insomnia, headaches, exhaustion, susceptibility to illness, sore back/neck
- ❖ **Behavioural** - alcohol/substance abuse, anger & irritability, absenteeism, isolation/ withdrawal, avoiding clients/ patients, impaired decision making, negative gossip/venting, eating habits
- ❖ **Emotional/Psych**- negative self-image, emotional exhaustion, depression & anxiety, eating habits, reduced empathy/sympathy, irrational fears, intrusive imagery. poor self-care, suicidal thoughts

## How Compassion Fatigue Affects Practice

- ❖ Will often have a manifestation of negative psychosocial behaviours, including but not limited to:
  1. **Lack of Empathy**
  2. **Professional Competence**
  3. **Deterioration of Physical Health**
  4. **Poor Work Habits**
  5. **Interpersonal Conflicts**
  6. **Addictions**
  7. **Struggles with Mental Health**

## Self-Care

- ❖ Isn't a quick fix! Allow yourself time to step back and reflect and relax, giving yourself to leave that stressful and demanding space you're in and refuel and regenerate.
- ❖ It is:
  - **Ongoing**
  - **Ethically Responsible**
  - **Balanced**
  - **SMART**- Specific, Measurable, Attainable, Realistic, Time orientated goals.

*References available from the author on request*

## LOSING PATIENCE WITH YOUR PATIENTS?

Nicole Rolfe

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Struggling with your patient to perform routine procedures such as x-rays, bandaging or IV catheter placement is stressful for both the patient and you. Providing sedation/anxiolysis, either orally or by IM injection, to ease the difficulty in performing these tasks is an extremely valuable tool in any veterinary practice. Appropriate 'sedation' can ease anxiety and provides 'chemical restraint' of our patients and can improve safety for both the patient and the handler. It is important to realize the full benefits of sedation and also to be aware of any concerns with any drug protocol you may choose.

Things that improve handling of our patients are still important even when providing sedation. Factors such as providing a quiet environment, gentle handling moving slowly but efficiently and using a calm voice go a long way in making various procedures easier to perform.

Next it is important to know your patient. Factors such as temperament, age, breed medical history and current problem/disease will affect your drug choices. Other considerations include accounting for weight and lean body mass. Larger breed dogs will generally require lower doses than small dogs and cats, for example and lean mass should be considered when choosing doses.

Some of the drugs we use routinely include: Trazadone, Gabapentin, alpha2 agonists such as Dexmedetomidine; opioids such as Butorphanol, Hydromorphone, Methadone; Acepromazine, benzodiazepines- such as Midazolam and finally Alfaxan or Ketamine. More often than not, combinations of the above mentioned drugs provides better sedation at reduced dosages than any single agent. Some of these drugs provide more than just sedation and those other effects are often a benefit. For example, while some opioids are analgesic, they can also be sedating. Benzodiazepines cause muscle relaxation as well as anxiolysis. All of these actions can be of benefit to our patients undergoing medical procedures.

In this presentation, specific sedation protocols will be discussed using a variety of real case examples for both dogs and cats. As well we will review the considerations that went into determining the drug choices and doses.

## Radiographic Interpretation of the Thorax in the dog and cat

William J. Hornof, DVM, MS, DACVR

Eric Herrgesell, DVM, DACVR

When students first start learning radiology, the thorax appears to be a daunting part of the anatomy, compared to the abdomen, but most radiologists I know, are much more comfortable and confident in their radiographic interpretation of the thorax. But why? For the beginning student, interpretation is combined with recognizing anatomy. In the abdomen anatomic structures are surrounded by fat, and in a skinny patient, they may not be visible at all. Finding anatomy can often require a bit of imagination, and not finding it may be completely normal. Thus it is very easy to get lulled into a false sense of security in the abdomen, because so much pathology can hide in a “normal” abdomen. The thorax is totally different, and more precise. Not seeing an organ like the heart or vessels in the lung is always totally abnormal. Beyond that, the reason they cannot be seen is usually also visible. With skill and experience the thorax becomes a window into the pathophysiology of the patient, but for the rookie, it is truly a daunting place, because it is so easy to be wrong.

Unfortunately learning to become skilled and experienced at interpreting thoracic radiographs, cannot be learned by reading books, or looking at pictures. When Dr. Herrgesell and I were on faculty at UC Davis, starting in the Freshman year every radiology exam was a practical examination with radiographic studies to match with diagnoses, and they were all open book. The Freshman always started on the first exam with lots of books and handouts, but the exam was totally case based, and the task was to match a set of cases with their diagnoses. They soon discovered that unless the case they were looking at was the exact case in Figure 5 in their textbook, they did not help. Becoming good at radiographic interpretation requires practice. In a residency that’s what happens. The resident interprets cases under the supervision of a several mentors. The key here is several, not just one mentor, because even the most experienced radiologists are going to incorrectly interpret what’s going on from time to time. For the most part experienced radiologists are very good at not missing any true positive findings, and rejecting false positive ones. The problem comes in combining the abnormalities, with the true negatives, and the clinical course, laboratory findings, etc., to come up with an appropriate diagnosis. Very few radiographic findings, by themselves, are pathognomonic. However, taken in concert with other findings, the underlying pathophysiology can sometimes be accurately deduced. As an example, a radiologist might glance at a thoracic study of a dog and instantly state emphatically: “That patient is in left heart failure!”, without any history or signalment. Unfortunately that conveys the misconception that the radiographic finding was: “Left Heart Failure”, but it was not. Left heart failure is a diagnosis, not a finding, as is pulmonary edema, pyothorax, pneumonia, etc., and each diagnosis has a “Set” of findings. However, often the trained observer can almost instantly recognize a “Set” of findings, that leads to a diagnosis. Another significant difference between the trained radiologist, and the general practitioner, is the radiologist is trained to disciplined and ignore clinical findings, when objectively assessing the radiographic findings. As an example, armed with the fact a patient as being heartworm antigen positive, a radiologist is much more likely to interpret a thorax as having normal pulmonary arteries than a student.

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The important difference between this course and probably any radiology course you have taken is this course is designed to deliberately separate the relatively simple task of finding the individual, and collectively agreed upon radiographic abnormalities, from the clinical signs or the actual diagnosis. Ideally the radiographic findings should be analogous to running a chem panel. The results are not influenced by the clinical signs. Unfortunately, analysis of radiographic findings is totally subjective, and can be heavily influenced by clinical signs. The intent of this course is to break the process of radiographic interpretation down into its component parts. Separating individual radiographic findings from a diagnosis, can help you gain confidence. Like a chem panel the findings are there regardless of the clinical signs or other information. That's not to say there can't be false positives and false negatives, but part of the intent of this course is to help you relate individual findings into a "Set" that makes sense, and helps define the underlying pathophysiology that could possibly cause such changes. Findings that do not fit into a set, are much more likely to be false, findings missing from a set are less likely to be missed. Those sets of findings are then put back together with the signalment and clinical information available, to come up with a list of diagnoses that need to be ruled out. That list then drives the plan for clinical management.

### The learning objectives of this course.

Everyone in this class has had numerous radiology lectures/courses, and likely has copies of numerous books, handouts, and .PPT presentations. Most are filled with pictures of lung patterns, masses, fluid in numerous compartments, cardiac diseases, and more, yet thoracic radiology continues to be a strong draw for CE. After years of teaching radiology we believe lectures are ineffective at transferring the necessary knowledge and experience for practitioners to gain confidence. It requires active coaching. One can only get so far reading a book on swimming, without ever getting in the water. There is also no guarantee that someone who studied massively on swimming, finds that their first time in the water, when they jumped into the deep end, they needed rescuing. Everyone in this class has been in the radiology pool and has some degree of proficiency, and our job is to coach you as you try to improve your form.

### The rules

This course will start out with a short lecture, just to get everyone on the same page, in terms of terminology. The rest of the course will be coaching while you as a group interpret cases. Unfortunately coaching doesn't work unless you are willing to get in the water. Active participation in the interpretation of cases is of paramount importance. We will provide signalment and presenting complaints on each case, along with the radiographic views available. We will then ask for volunteers to point out abnormalities, one at a time, and with each one ask for agreement with the rest of the group. If everyone sits there quietly waiting, we will all wait. It will then be up to members of this class to come to consensus on what the radiographic abnormalities are. Once we have consensus, we will list the abnormalities we saw to compare. Then we will go back to the group for consensus on the differential diagnoses for the case, and then come to consensus on the plan for case management. We will then we

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will disclose the actual outcome of the case, and lead a discussion and answer questions. Believe it or not this course is fun, but only if everyone gets involved.

The findings

Amongst other things we will be looking for findings like:

- 1) Pleural fissure lines
- 2) Silhouette sign
- 3) Lobar borders
- 4) Air bronchograms
- 5) Pulmonary patterns
- 6) Cardiac chamber enlargement
- 7) The shape and size of the aorta, vena cava, pulmonary arteries and veins
- 8) Abnormal pulmonary arteries and veins

Although defining the specific underlying disease will be our goal, our list of findings will be objective, and not include differential diagnoses such as:

- 1) Pulmonary edema
- 2) Heart failure
- 3) Cancer
- 4) Fungal disease

## Radiographic interpretation of the abdomen in dogs and cats

William J. Hornof, DVM, MS, DACVR

Eric Herrgesell, DVM, DACVR

### Introduction:

In our opinion the abdomen presents the greatest interpretive challenge of any body part in small animal radiology. Interestingly, most students initially find the thorax more daunting than the abdomen, but with experience the changes in the thorax caused by specific disease processes, are far more predictable in the thorax than in the abdomen. The main problem with interpretation of the abdomen is there is very little contrast between organs and their surrounding fat, and they are all superimposed. In the thorax where organs are more separated and highlighted by air filled lung, one can be much more emphatic about whether or not something is normal. Not seeing the heart on a thorax radiograph is abnormal, but not seeing such things as the pylorus, pancreas, or right kidney may all be normal. Additionally not seeing a large mass in an otherwise normal thorax means there is no large mass present, while large masses can conveniently hide within the abdomen. The following is an approach to teaching abdominal interpretation that I have adopted over the years.

### Body conformation:

An important part of abdominal interpretation will be the assessment of serosal detail. In order to see serosal surfaces radiographically, it requires fat be in contact with the serosal surface. The increased contrast provided by digital radiography has dramatically improved our ability to detect changes in serosal detail. Thus obese patients tend to have more serosal surfaces visible than lean patients. To me then it is important to determine if the abdomen is distended and if it is, why? Assessing abdominal distension is quite subjective, but as a rule of thumb if the body wall goes in a straight line from the xyphoid to the pubis the abdomen is not distended. If it sags down it is distended. If the abdomen is distended, there are 4 possibilities. Most common is the patient is obese and fat is distending the abdomen, but if there is not a lot of obvious intra-abdominal fat, and there is a lack of serosal details, there are 3 possibilities. The abdomen could be distended with fluid, in which case there will be a complete lack of serosal detail, except where the liver lays on top of the falciform ligament, and the retroperitoneal space. The bowel could be distended with fluid or air and the serosal surface of the bowel are pushed together squeezing out the fat. Lastly there could be an intra-abdominal mass that again is squeezing fat away from serosal surfaces making them appear diminished.

Find your friends:

This incredibly simple statement, summarizes our approach to abdominal interpretation. The goal of abdominal interpretation should be to first inventory those structures that can usually be identified, and probably more importantly, are any of the typically visible structures (your friends) not visible? If a friend is visible, is it normal, and if a friend is not visible, why? We personally do not use a systematic approach to radiographic interpretation, but do systematically identify each of my friends even though the order may vary. The following then are not listed in a suggested order.

Who are your friends:

The Colon:

The nice thing about the colon is it attaches to the anus and the anus is locked in position. That means the end of the colon at least is fixed in position and should be visible. The next step is to carefully track as much of the colon as possible on both views and identify the caecum. At that point it is important to identify any other bowel loops that look like the size the colon should be. If the complete colon is identified and there are other “colons” in the abdomen, they are not colon and they represent dilated small bowel.

The position of the colon is also important. The colon is loosely tethered by the mesocolon and is free to move about the abdomen, but departure from its normal location should be noted as you search for more friends.

The kidneys and retroperitoneal space:

The kidneys are visible because they are surrounded by retroperitoneal fat. On a straight lateral view the dorsal surface of each kidney should be visible, and the retroperitoneal fat of uniform density, without streaks. If the kidneys are not visible on a straight lateral view, with the transverse processes of the lumbar vertebrae exactly superimposed, either the patient is emaciated, in which case there will be no retroperitoneal fat, or the borders of the kidneys are being obscured by fluid accumulation. If there is retroperitoneal fluid, there will be streaks in the retroperitoneal space. Positioning is critically important. If the patient is obliqued the retroperitoneal space and dorsal surface of the kidneys may not be visible, and important information may be lost.

On the VD view the left kidney should be visible, and nestle itself between the spleen, the fundus of the stomach and the spleen. This triad of your “friends” is always visible in normal subjects, and finding one or two of them can help you identify the others.

The bladder and prostate:

On the lateral view at least the caudal bladder and neck should be visible as will the prostate in intact males. The path of the colon and bladder should be compared. If the colon and bladder are displaced ventrally, the cause is most likely dorsal to the colon or lymph node related, but if the colon and bladder are separated the cause is likely related to the genital tract like uterus or paraprostatic cyst.

### The falciform fat:

The falciform fat is a big solid blob of fat that is typically not obscured by abdominal fluid. Even with a fairly massive effusion the falciform fat is typically visible along with the serosal surface of the liver and the ventral body wall in contact with the fat. Like the retroperitoneal space it is a good place to gauge body fat content, and a good place to judge how serosal surfaces should look in the rest of the abdomen.

### The liver and stomach

The liver and stomach are difficult to separate on survey radiographs. However, because they are linked anatomically, the two should be evaluated together along with other clues to their whereabouts. In many cases the stomach is fluid filled and cannot be distinguished from liver. However, much like the left kidney, spleen and fundus of the stomach, the liver and stomach tend to move as a unit. The cranial border of the liver is visible because it sits in contact with the diaphragm, and the ventral border sits in contact with the falciform ligament. The duodenum is reasonably well tethered in position and points to the pylorus, but the pylorus may not be visible unless it contains air. Evaluating the stomach and liver then is a combination of finding those friends you can and deducing the location and contour of the stomach and liver.

### The small bowel:

With the exception of the duodenum. The small bowel is too variable in its location to discern the different parts. However, the pattern of gas within normal bowel, caused by normal peristalsis is characteristic. The luminal gas shadows should be smooth and appear cylindrical. Angular gas shadows and rigid appearing small bowel loops are abnormal and warrant further investigation.

### The pancreas and right upper quadrant

The normal pancreas is not visible radiographically, but typically the relationship between the organs of the right upper quadrant is. If there is a mass in the area of the pancreas, everything gets pushed away. There is more distance than normal between the duodenum and stomach, and both the large and small bowel are pushed caudally and medially, leaving a void in the right upper quadrant. The left limb of the pancreas may also be visible behind the fundus. Again the increased contrast resolution of digital over film has dramatically improved our ability to discern such structures.

## TRAUMATIC WOUND MANAGEMENT & BASIC RECONSTRUCTION

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### Traumatic Wounds

Traumatic wounds are common in cats and dogs and result from a number of causes; hit by automobile accidents, bite wounds from other animals and gunshots to name some more common etiologies. In many cases, these wounds can be difficult to manage due to their level of contamination, location and associated limited, viable tissue available for reconstruction. Consequently, many wounds require open management for some period of time. Open wound management can be a challenge in regards to cost, patient discomfort, requirement of sedation or analgesia and patient fasting due to the frequency with which the wound may need to be dressed. Further, antimicrobial resistance is also associated with prolonged periods of open wound management in both humans and companion animals. To combat these issues efforts should be made to minimize the time that wounds remain open prior to reconstruction and consideration should be given to the degree of invasiveness of daily wound care. The author of these notes prefers less invasiveness in wound care as a general philosophy as long as the main tenants of wound management are adhered to. In general, with contaminated or infected wounds, I prefer an initial anesthetized debridement of all nonviable tissue, followed by daily wound inspection and care until the wound can be closed or nearly closed. If further, significant debridement is indicated after the initial debridement, then this is performed accordingly under anesthesia if needed. If only minimal debridement or care if required (typical), then sedation and analgesia are all that is usually required. Anesthesia requires fasting and many patients with wounds have pre-existing hyporexia or anorexia which is exacerbated by fasting associated with anesthesia. These patients are at significant risk for hypoalbuminemia, malnutrition and acquisition of hospital-associated infections and sepsis.

Traditional wound debridement and application of debriding bandages is almost always indicated at the time of initial evaluation of traumatic wounds. The exception is with traumatic wounds with minimal tissue injury, clean lacerations and when the wound is evaluated within the golden period, typically within 6 hours of injury. My preference for a primary bandage layer has been hypertonic saline. Hypertonic saline is effective, available, inexpensive, clean and exerts an osmotic effect on wound bed pathogens. Hypertonic saline also tends to dry out more reliable than physiologic saline and other dressings. During bandage removal, this results in a gentle peeling effect on the wound bed which is helpful in removing biofilms and superficial pathogens. Medical grade honey and sugar bandages are messy, do not dry out reliably, tend to require additional bandage changes, provide sugar energy to wound pathogens and are therefore less favorable. Negative pressure wound therapy is another option for wounds that may require prolonged debridement and in which case the patient may not be healthy enough to tolerate daily anesthesia and periods of fasting. While negative pressure wound treatment has been demonstrated to be clinically effective in some cases, controlled study demonstrating a significant benefit over traditional wound support has yet to be reported in veterinary medicine. Yet another option that the author of these notes has found to be useful is Nanocrystalline Silver which is being used with increasing frequency in both human and veterinary medicine. The proposed benefits of Nanocrystalline Silver impregnated dressings are:

1. Persistence of an antimicrobial effect
2. Simplicity of use
3. Reduced costs associated with frequent bandage changes
4. Improved patient comfort as a consequence of fewer dressing changes
5. Reduced requirement for sedation or anesthesia for dressing change
6. Improved patient nutrition
7. Lack of development of antimicrobial resistance

Ionic silver exerts its antimicrobial effects by limiting the production of cellular energy by direct interference with electron transport system of the micro-organism.<sup>1</sup> Although silver has been used in wound management (e.g. silver sulfadiazine) for hundreds of years, it has not been until recently that the nanocrystalline form has been used. The major advantage of the nanocrystalline silver impregnated dressings is the ability to maintain a slower, more gradual release of silver ions which are responsible for the bacteriocidal properties.

Currently there is a paucity of literature on the use of nanocrystalline silver as a primary wound dressing in dogs but there are many publications on its use in human medicine, especially in burn victims. The author of this manuscript has used the aforementioned wound dressing in over 30 cases and has found the dressing to be effective and useful in regards to limiting the frequency of bandage changes and in providing adequate antimicrobial control during the early phases of wound healing (**Figure 1**).

A number of silver impregnated dressings are available in the veterinary market. One example is Acticoat (Smith & Nephew Pty Limited) which is available in two main forms, 3- and 7-day. As the names suggest, the 3-day version maintains antimicrobial efficacy for 3 days and the 7-day version, for 7 days.



**Figure 1.** Image of a dog who underwent primary resection of a large truncal Pythium lesion. The large defect was unable to be closed completely primarily. A nanocrystalline silver dressing was applied to the remaining open wound. The silver dressing was replaced after 3 days and the wound went on to heal by second intention over the next 30 days. This dog recovered uneventfully and is now 5 years out with no evidence of recurrence.

### Basic Wound Closure/Healing

*Primary Closure* - Surgical closure of wounds within the golden period (< 6 hours). Primary closure is indicated for clean/sharp wounds e.g. lacerations. Because these wounds have limited contamination and tissue compromise they are amenable to immediate closure. Immediate closure allows wound healing to proceed with a significant reduction in time for all phases of healing.

*Delayed Primary Closure* - Surgical closure of a wound after the golden period but before the appearance of granulation tissue. This method of wound closure is typical of recent traumatic wounds such as dog bites and hit by car injuries. This approach along with efficient open wound

management (e.g. debridement & bandages discussed above), decreases the likelihood of infection and complications. Also, by appositional wound closure, there is a reduction in the time necessary for wound healing; this will minimize the size of the scar and increase the strength of the healed wound.

*Late Secondary Closure* - Closure of a wound by surgical intervention following the proliferative phase of wound healing (i.e. after granulation tissue is present). Generally used for wounds that are more contaminated and have greater soft tissue damage. It has the advantage of a lower risk of infection over primary and delayed primary closure. The disadvantages include greater wound retraction resulting in less mobile skin, which generally requires undermining the surrounding skin edges for complete closure, and often results in greater tension on the suture line. The cosmetic result will not be as good as that of primary or delayed primary closure because more proliferative tissue has been laid down.

*Second-Intention healing* - Leaving the wound open to heal without any surgical intervention. Secondary closure depends entirely on neovascularization and extracellular matrix remodeling to restore tissue bulk, wound contraction to re-establish normal tissue tension and to reduce scar size, and re-epithelialization to provide normal surface coverage. Second intention healing is less ideal as it tends to be time consuming and labor intensive regarding on-going wound support and cosmetically, results are variable.

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## USEFUL SKIN FLAPS FOR DIFFICULT WOUNDS

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### Skin Flaps

Reconstructive cutaneous surgery requires that the surgeon have a sound knowledge of basic surgical principles and is able to apply them to each patient and problematic lesion. The success of the procedure is contingent upon this tenant. Inappropriate wound management leads to increased morbidity in terms of infection, pain, failure, economics, and death. Every dog or cat requiring reconstructive surgery is unique and the whole of the patient must be considered and factored into the therapeutic plan.

Because of these many considerations, there is a need for versatility in treatment and closure techniques. This has led to the creation of a whole host of different skin flaps in dogs and cats. A number of different classifications exist and are typically based on: vascular properties, specific “named” arterial blood supply, anatomic location, and tissue composition (e.g. cutaneous, myocutaneous, gingival). Below is a summary of the most common classifications and practical examples of their use.

#### Vascular flaps

The *Subdermal plexus flap* is a flap that relies on the superficial and middle branches of the cutaneous arteries in the hypodermis and panniculus muscle. An *Axial pattern flap* is a flap that is maintained by a “named” direct cutaneous artery and vein and is more robust in regards to its blood supply than a subdermal plexus flap. An *Island arterial flap* is an axial pattern flap in which the cutaneous pedicle is completely severed, leaving it attached solely by the artery and vein. Finally, a rare vascular flap is a *Free vascularized skin flap* which is basically, an axial pattern flap in which the vascular pedicle is severed, then anastomosed to vessels at a distant recipient site.

#### Non-vascular flaps/grfts

In contrast to vascular flaps, avascular skin grafts have no patent blood supply existing at the time of implantation to the recipient site. Two major variations exist based on the thickness of the dermis - *Full thickness* and *Partial thickness* skin grafts. Full thickness grafts are skin grafts in which no reduction of the dermal thickness has been created. In contrast, partial thickness grafts are grafts in which the dermis has been attenuated, usually with a Dermatome (an instrument similar to a cheese slicer), which effectively splits the thickness of the dermis. Non-vascular flaps/grfts can also be *Meshed* or *Non-meshed*. Meshing is basically the creation of many small (~1cm), full-thickness slits in the dermis which increases the surface area of the graft by up to 5-times. These types of grafts tend to be simple to use but are more prone to dehiscence than vascular grafts. They also rely heavily on the recipient wound bed which MUST be a healthy soft tissue bed and not bone, fascia or infected tissues. A common use of these types of grafts are with appendicular oncologic wounds (e.g. soft tissue sarcomas, mast cell tumors, etc).

In addition to blood supply, skin flaps are also Classified by location. Local flaps are based on tissue that is adjacent to the defect to be covered. Typical local flaps include:

*Advancement flaps* are subdermal plexus flaps that are elevated and advanced directly over a defect. *Rotational flaps* are subdermal plexus flaps that involve rotation (typically in an arc fashion) of a piece of skin that is continuous with one portion of the defect. *Transposition flaps* are subdermal plexus flaps that are elevated and rotated (usually less than 180 degrees) into an adjacent defect. Transposition flaps are similar to rotational flaps although they are usually larger and result in a larger secondary defect. Transposition flaps are more versatile than rotational flaps. *Interpolation flaps* are subdermal plexus flaps that are elevated and rotated into a defect that is not continuous (not touching) with the flap. A portion of this flap must pass over intact skin prior to recipient implantation. Thus these are rare flaps to use in veterinary medicine but can be exceptionally useful for reconstruction of oral and periorbital wounds.

Distant flaps are subdermal plexus flaps that are based on tissue that is obtained from a site that is not continuous with the recipient bed. This tissue can be *tubed* and slowly advanced to a distant site; this is referred to as an indirect distant flap. Tubed flaps require multiple procedures; typically 10-14 days apart. Tubed flaps can be tumbled or caterpillared into the recipient bed. They are rarely used, due to development of axial pattern flaps and free skin grafts which in contrast to tubed flaps, do not require multiple procedures. Another type of distant flap is the *pouch* or direct bipedicle flap. These flaps are simple and effective but do require a second procedure. *Pouch or pedicle flaps* are created by incising the lateral trunk (thorax or abdominal skin), then either advancing the pelvic limb cranially, or the thoracic limb caudally, placing the recipient bed into the *pouch*. As mentioned above, these procedures require a second surgery to “detach” the grafted wound typically 7-10 days later. I have found these wounds very useful for traumatic and oncologic wounds involving the metatarsal and metacarpal wounds in small-to-medium-sized dogs without compromise to the other 3 limbs.



**Figures 2 and 3.** A Boxer dog with a soft tissue sarcoma around the right stifle. The tumor was resected with 3 cm margins and reconstructed in a single stage with a CSE axial pattern flap. Axial pattern flaps are exceptionally reliable and have several advantages for use in wound closure. Due to their robust blood supply, axial pattern flaps can be much longer than subdermal plexus flaps and still maintain viability. The direct blood supply makes axial pattern flaps preferable for tumor resection sites that may require radiation therapy, which would

cause rapid necrosis of a non-vascularized skin graft. Since they are full thickness flaps, axial pattern flaps maintain hair growth and cosmesis and provide relatively durable skin covering. One of the greatest advantages of axial pattern flaps is that they allow instant, complete closure of large defects without the laborious bandage changes required for free skin grafts. The author has found the *caudal superficial epigastric* and *thoracodorsal* axial pattern-type flaps to be exceptionally useful and practical.

The primary disadvantage of axial pattern flaps is that they do not reach the distal extremities in dogs or cats. Another disadvantage of axial pattern flaps is the large donor site incision, which

increases surgical time and patient morbidity. For these reasons, direct bipedicle flaps are often used for distal extremity defects. It is important to make sure that the patient will tolerate the limb held in flexion or extension and that the patient does not have any orthopedic or neurologic deficits. Often, a test bandage to hold the limb along the body wall is placed prior to surgery to make sure that the dog or cat will tolerate the procedure.

There are many reconstructive techniques available to the practicing veterinarian. While most are simple, careful attention to limitations and principles is critical to ensure the best possible outcome.

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## PERINEAL & ABDOMINAL HERNIAS

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### Overview

Perineal herniation (PH) is a common and nearly 100% preventable disease affecting male intact dogs. Although other species and sexes have been reported to develop perineal herniation, the disease in companion animal practice is almost exclusively a disease of older, male-intact dogs. While the exact etiology of PH is unknown, most believe that a mixture of genetic and prostatic-related factors ultimately lead to degeneration and atrophy of the pelvic diaphragm. Once the pelvic diaphragm ruptures a variety of morbid consequences can ensue including displacement of caudal abdominal organs such as the rectum, colon, prostate, urinary bladder and small intestine. Often affected dogs have chronic underlying conditions such as benign prostatic hyperplasia or diarrhea which are made all the more worse once a hernia develops. Perineal herniation is a very morbid condition which can lead to death in some cases. Affected dogs are often very painful, have chronic tenesmus, constipation and diarrhea and lower urinary tract obstruction due to bladder retroflexion. Depending on the status of the dog and the timing of presentation, either emergent or elective surgery may be indicated. In dogs with incarcerated organs or bladder entrapment with urinary obstruction emergency surgery is often necessary. A myriad of surgical options to correct PH in dogs have been described but the most common technique performed is the internal obturator muscle transposition (IOMT) procedure with or without pexy of the urinary bladder and/or colon.

### Presentation & examination

The most common clinical signs at the time of presentation are: swelling in the perineal region, diarrhea, tenesmus and constipation. Physical examination alone often reveals an obvious perineal swelling which can range in size from barely perceivable to very large depending on the organs contained within the hernia. Dogs with urinary bladder entrapment can have a very large perineal swelling due to the expansive nature of the urinary bladder. Caudal abdominal palpation may also reveal an absent urinary bladder in the case of bladder retroflexion. In dogs without bladder retroflexion, an enlarged prostate can be palpated at the cranial brim of the pubis. This is due to gradual cranial displacement of the prostate which occurs in most intact dogs as they age. The combination of a digital rectal exam and caudal abdominal palpation with the opposite hand is useful to assess these structures. Digital rectal exam allows for palpation of the pelvic diaphragm, prostate and urethra in most dogs although other organs can also be palpated depending on the case. It is common to find either bilateral or unilateral PH but it is uncommon to palpate a unilateral PH with a “normal” contralateral pelvic diaphragm. Most dogs with a unilateral PH have thin and palpably weak contralateral pelvic diaphragm muscles which may rupture in the future despite successful management of the ruptured side. Owners should be warned of this possibility at the time of surgery. Because most affected dogs are older, a thorough physical assessment should be made prior to performing anesthesia and PH surgery.

### Diagnostics

A complete diagnostic assessment is necessary in most dogs with PH. In general, a complete blood count, serum biochemistry and urinalysis are indicated for systemic and for pre-anesthetic assessment of the patient. Diagnostic imaging should include imaging of both the abdomen, perineum and thorax. In general, abdominal radiographs including the perineal region are

adequate to assess for major organ herniation although ultrasound is useful due to the improved textural and spatial resolution it provides. Thoracic radiography is indicated to assess for cardiopulmonary abnormalities which are common in aged dogs. Computed Tomography including the chest and abdomen is also a good option and may be useful in some challenging cases of PH.

### Initial treatment

Initial treatment of dogs with PH can vary depending on the status of the dog. For example, in dogs with bladder retroflexion, emergent stabilization including urinary catheterization or paracystocentesis (urethral catheter can't be passed) should be performed initially and any fluid deficits corrected with intravenous crystalloids. Other dogs with PH may be systemically stable without organ herniation and may not require any specific preparation prior to surgery. General recommendations prior to surgery include: withholding of food for 12 hours and digital rectal evacuation immediately prior to surgery. A purse-string suture is then placed carefully avoiding the anal sacs. Perioperative antibiotics are up to the discretion of the surgeon but broad spectrum, including both gram- negative and positive are recommended. Cefazolin is a commonly utilized antibiotic and can be administered at 22 mg/kg IV q 90 mins during surgery. The second generation antibiotic cefoxitin is also a good choice and is administered at 22 mg/kg IV q 90 mins during surgery.

### Surgical approach

Once the patient is clipped and prepared aseptically, the dog is positioned in sternal recumbency with the tail pulled forward and secured to the front end of the table. In the figure, a dog with a left-sided perineal hernia is correctly prepared and positioned for IOMT



herniorrhaphy. Notice the padding under the caudal abdomen and inguinal region as well as the tail taped to the front edge of the table. It is helpful to tilt the operating table about 30 degrees with the head directed downward (Trendelenburg position). My preference is to sit during PH surgery and Trendelenburg position facilitates an ergonomic position for the procedure. Perineal hernia surgery can be challenging for a variety of reasons and should not be undertaken if the surgeon is not prepared for the many contingencies which may exist. Although many different procedures have been described and are sometimes needed in complicated PH, the most commonly used and predictable procedure is the internal obturator transposition herniorrhaphy in conjunction or after castration. Other procedures which may be indicated include: cystopexy, colopexy or contralateral semitendinosus muscle

(STM) transposition. The author does not use nonabsorbable implants such as mesh due to the risks of infection and the availability of autogenous tissues such as the IOM and the STM. Colopexy is also not performed due to the lack of a demonstrated benefit and the risk of colonic perforation and septic sequella.

Once the patient is positioned, prepared and draped for surgery, an adhesive dressing such as loban should be applied over the perineal region to protect the exposed surgical wound from contamination. A skin incision is made over the hernia approximately 2-3 cm lateral to the anus and extending from the tail base to the ischiatic tuberosity. The adhesive dressing is then sutured in to the cut skin edge nearest the anus. Caution should be exercised when incising the skin as the hernia sac and underlying viscera are immediately under the relatively thin perineal skin which could result in visceral and neurovascular injury with an overzealous cut. Once the hernia sac and/or displaced organs are identified, they should be inspected and reduced

through the hernia back into the caudal abdomen. It is often necessary to place a laparotomy or gauze sponge into the hernia to help prevent re-displacement of the herniated viscera during the herniorrhaphy. Identification of the internal pudendal and caudal rectal neurovascular structures should be performed so as to avoid inadvertent transection or injury during the surgery.

Once the pelvic diaphragm muscles have been identified, the internal obturator muscle is palpated and assessed for suitability for transposition. While some IOM are robust and thick, others are thin and seemingly less strong. This being said, the author has never identified an IOM that could not be used for PH at least at the time of the first surgery. The author has used the STM in rare cases when revision from a failed IOMT procedure occurred. Once the IOM has been identified, its tendinous insertion is carefully incised with a number 15 blade along the most caudal aspect then gently elevated off the ischium using a Freer periosteal elevator. It is unnecessary to transect the muscle tendon as transposition cranially and medially is not impeded by this structure. It is rare to identify any robust remnant of the levator ani muscle and this muscle is not necessary for most PH repairs. However, it is critical to correctly identify the coccygeus, external anal sphincter and internal obturator muscles as these muscles will need to be sutured securely to perform the herniorrhaphy.

Although nonabsorbable and absorbable suture can be used, the author prefers a long-lasting absorbable suture such as polydioxanone or polyglyconate between 2-0 and 3-0 depending on the size of the dog. Simple interrupted sutures are preplaced between the three muscles to reconstruct the pelvic diaphragm. Once the sutures have been preplaced and the surgeon is satisfied with the apposition of the muscles, individual sutures are tightened. The area is lavaged with sterile saline and the wound closed in two layers; subcutaneous and skin. The author prefers intradermal skin sutures in this region to provide acute snug apposition which may minimize contamination of the wound. The purse string suture is removed and a digital rectal exam is performed to assess the repair.

### **Postoperative care & monitoring**

Dogs should be managed for postoperative discomfort and provided both opioid and nonsteroidal analgesics assuming there is no contraindication. Laxatives such as lactulose are administered for up to 4-6 weeks after surgery to reduce straining and stress on the repair during healing. The continuation of prophylactic antibiotics is debatable but the author prefers to continue broad spectrum antibiotics for 7 days after surgery while the skin is healing.

In the hands of an experienced surgeon, complications with perineal herniorrhaphy are infrequent but can be significant in some cases. The risk of infection and abscessation of the wound is around 10% and is likely minimized by strict adherence to aseptic technique including the use of an adhesive dressing such as Ioban. Fecal incontinence is also rare and is avoided by careful visualization and protection of the internal pudendal and caudal rectal neurovascular structures during surgery. Immediate severe pain and sciatic deficits should prompt the surgeon to consider if accidental sciatic entrapment occurred during the surgery. This is rare but can be seen when the sacrotuberous ligament is used in the repair. Use of the sacrotuberous ligament is rarely necessary in PH repair in the author's experience. Continued tenesmus and recurrence of the hernia can also occur and should prompt the veterinarian to explore for an underlying cause. If the repair is intact but signs continue, the contralateral side should be assessed and/or another underlying cause of persistent tenesmus should be explored.

### **Prognosis**

The prognosis is typically good following surgery although high recurrence rates have been documented, especially with primary herniorrhaphy alone (not recommended). Other factors that negatively affect outcome are: lack of surgeon experience, not performing castration and tension on the repair.

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## CHALLENGING AIRWAYS

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### Background

Brachycephalic Airway Syndrome (BAS) is a purpose-bred condition in which rostral to caudal compression of the skull and airway results in significant obstruction to airflow through the upper airway. Typically affected breeds include: French and English Bull Dogs, Pugs, Pekingese, and Boston Terriers.<sup>1,2</sup> Clinical signs range from mild to severe and include: stertor, “snoring”, dyspnea, exercise intolerance, as well as gastrointestinal signs (regurgitation, vomiting, diarrhea). Clinical signs associated with BAS tend to be progressive with age. This is likely due to the deleterious effects of chronic pharyngeal and laryngeal obstruction, which result in weakening and ultimately collapse of the arytenoid cartilages.

Laryngeal Collapse (LC) typically starts as eversion of the laryngeal saccules (grade 1 laryngeal collapse), which further obstructs airflow through the ventral aspect of the larynx. Consequently, larger pressure differences are required during inspiration to move air through the larynx and into the trachea. Because a more negative airway pressure develops (Venturi effect) caudal to the larynx during inspiration, there is greater cyclic stress on the arytenoid cartilages, which likely leads to the initiation and progression of LC. Eventually, adduction of the cuneiform (grade 2 laryngeal collapse), and corniculate processes (grade 3 laryngeal collapse) occurs, which results in severe laryngeal obstruction.

The most common findings in BAS dogs are: an elongated soft palate, stenotic nares, and everted laryngeal saccules.<sup>1,2</sup> Consequently, surgery is most often directed at shortening the soft palate, widening the nares, and removing the everted saccules (grade 1 and 2 laryngeal collapse). Prognosis is typically good in dogs with grade 1 laryngeal collapse and guarded to good in dogs with grade 2 collapse. However, in dogs with grade 3 laryngeal collapse, permanent tracheostomy or arytenoid lateralization is most often required to obviate the severe airway obstruction. \*\*\*It is important to note that surgical difficulty and severity of complications increases significantly in dogs with grade 2 and 3 laryngeal collapse\*\*\* Therefore, it is strongly recommended that early surgical intervention be undertaken (when indicated) to reduce the progression and clinical signs associated with BAS.

### Preoperative management

All owners should be counseled thoroughly regarding the risks of upper airway surgery prior to intervention. The most important preoperative considerations are systemic, cardiovascular and pulmonary health. In addition to a complete physical examination, a complete blood count, serum chemistry, urinalysis, and survey-thoracic radiographs are indicated. Common concurrent conditions include: valvular insufficiency, pneumonia, gastrointestinal erosion and hiatal hernia. In more complicated cases, consideration should be given to preoperative CT and upper GI endoscopy. Preoperative CT is useful to diagnose less common abnormalities such as aberrant nasal turbinates, nasopharyngeal stenosis and soft palate thickening which can affect surgical indications and outcome. Endoscopy is useful for assessment of hiatal herniation and GI erosion and ulceration. Dogs presenting for surgery are fasted at least 12 hours prior to the procedure if possible. Use of sedative and opioid drugs is minimized due to the potential for decreased gastrointestinal motility and aspiration pneumonia. If regurgitation or vomiting are

reported or if blood work is consistent with gastric ulceration, H<sub>2</sub>-blockers (e.g. famotidine 0.5 mg/kg po BID) or proton pump inhibitors (e.g. pantoprazole 1 mg/kg po q 24 hours) are indicated. Anti-inflammatory doses of steroids can be used perioperatively in dogs with laryngeal edema and swelling. I prefer to use a single dose of dexamethasone (Dexamethasone-SP; 4 mg/ml, MWI, Meridian, ID 83680) 0.15 mg/kg intravenously at the time of surgery. Repeated dosing is avoided due to the risk of gastric ulceration. My preference is to perform laryngeal examination at the time of anesthesia induction in contrast to the initial examination as the diagnosis of BAS is based predominantly on signalment and clinical signs.

### Surgical procedures

**Soft palate** – The soft palate is a fleshy muscular organ and is lined by mucosal epithelium. It serves to divide the nasopharynx from the oropharynx. The soft palate prevents nasopharyngeal reflux during swallowing by occluding the nasopharynx. As such, inaccurate assessment of length and/or overzealous shortening must be avoided. An elongated soft palate is diagnosed when the caudal margin of the soft palate extends past the tip of the epiglottis or when the caudal margin extends further than the caudal aspect of the tonsillar crypts. The tonsillar crypts tend to be more reliable landmarks as their position does not change with rostral traction of the tongue. This is in contrast to the epiglottis, which is pulled forward and may artificially exaggerate the length of the soft palate, potentially leading to over-shortening and subsequent nasopharyngeal reflux.



Once the diagnosis of an over-long soft palate is made, the patient is positioned and the amount of palate to be resected is planned. My preference is to put the dog in sternal recumbency with the maxilla suspended using an ether stand or 2 intravenous poles (**Figure 1**). The mandible is left free and the endotracheal (ET) tube is tied to it. This allows manipulation of the mandible, ET tube, and tongue together during the procedure. An Army-Navy retractor works nicely to retract the ET tube and base of the tongue ventrally during the procedure. \*\*\* “Must have” instruments include a pair of long DeBakey forceps, long Metzenbaum

scissors, and long needle drivers.\*\*\*\* I start by placing a 4-0 monofilament absorbable suture at the level of the planned resection on the left side of the palate, adjacent to the junction with the oropharyngeal mucosa. This suture is left long and then set aside. Next a stay suture is placed centrally at the caudal aspect of the soft palate. This suture is used for manipulation of the palate during resection. The stay suture is grasped and the palate pulled rostrally. The long Metzenbaum scissors are then used to transect the soft palate from left to right (right handed surgeon) just distal to the first tagged suture along the previously planned margin. At this point, the stay suture and excised soft palate are discarded. The nasopharyngeal and oropharyngeal mucosa are closed in simple continuous fashion from right to left using the originally placed suture. **Figure 1.** a) Notice the stenotic nares and b) elongated soft palate.

*Stenotic nares* – Stenotic nares are diagnosed when narrowing or obliteration of the nostrils by adduction of the paired ala and dorsolateral nasal cartilages is present. The result is significant obstruction of airflow through the nostrils and nasopharynx. Consequently, large negative pressures must be generated to move air through the nasal passages and into the larynx. Chronic exposure to these large negative pressures is hypothesized to significantly contribute to the progression of clinical signs and eventual deformation of the larynx (LC).

There are a number of previously reported techniques for correction of stenotic nares, but my preference is to perform vertical wedge resection. With the patient in sternal recumbency, a number 15 blade is used to create a vertical wedge in the nasal planum and dorsolateral alar cartilage. The nasal cartilages have an abundant blood supply stemming from the maxillary artery. Gross hemorrhage is expected but can be controlled by gentle tamponade using cotton tip applicators. Once the wedge of tissue has been excised, simple-interrupted apposition using an absorbable, 4-0 monofilament suture on a fine-tapered needle, is performed. Four or five interrupted sutures are all that is required per nares.

*Everted laryngeal sacculles* – Everted laryngeal sacculles are relatively avascular outward eversion of the laryngeal ventricle mucosa. Eversion is a sequela of upper airway obstruction (stenotic nares and elongated palate) and ultimately becomes part of a vicious cycle whereby complete obstruction of the ventral larynx results. There are a number of methods by which they can be removed but simple excision using long Metzenbaum or Stevens tenotomy scissors is preferred. Because bleeding is minimal to absent hemostasis is generally not required. The main challenge with laryngeal sacculle excision is that the dog must be extubated during the procedure. Because of this, I prefer to perform this correction after both soft palate and stenotic nares correction are completed.

*Laryngeal collapse* - As discussed above, laryngeal collapse is plastic or permanent deformation of the arytenoid cartilages secondary to chronic upper airway obstruction. Because many dogs with grade 2, and all dogs with grade 3 laryngeal collapse, do not improve with the surgical techniques described above, permanent tracheostomy or arytenoid lateralization are required to reduce upper airway resistance enough to improve clinical signs.

### **Postoperative management and outcome**

All dogs undergoing upper airway surgery are monitored closely for at least 24 hours postoperatively. Anti-inflammatory corticosteroids (discussed above) can be used but their impact is limited. Nasotracheal oxygen supplementation is useful and effective during recovery from anesthesia. In some cases, temporary tracheostomy for 24–48 hours is required due to laryngeal swelling and collapse. If a patient cannot be weaned off of the temporary tracheostomy tube, arytenoid lateralization or permanent tracheostomy are considered if the owners elect to proceed with further treatment. Permanent tracheostomy requires a huge commitment on the part of the owners and that fact should not be underestimated. Cricoarytenoid lateralization has been reported to be effective in some dogs with grade 2 and 3 laryngeal collapse although the author of these notes has not found it to be effective in the few cases I have attempted it in. Prognosis following surgery is good to excellent for young or early stage (less than grade 1 laryngeal collapse) brachycephalic dogs.<sup>1,2</sup> However, dogs with grade 3 laryngeal collapse or those that require permanent tracheostomy or arytenoid lateralization have a guarded prognosis. \*\*\*Consequently, early surgical intervention is recommended to avoid or reduce the risk for developing laryngeal collapse later in life.\*\*\*

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## WHEN TO NEUTER AND WHY

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### The Big Picture

In the United States, gonadectomy in the form of ovariohysterectomy (OHE) and castration of dogs and cats are the most common elective surgical procedures performed in veterinary practice.<sup>1</sup> In addition to reducing pet overpopulation, elective neuter also prevents spread of disease and animal suffering.<sup>2</sup> Between 3 and 4 million unwanted dogs and cats are euthanized annually in the United States alone. Dogs and cats are efficient and prolific in regards to reproduction. For example, female cats are polyestrous and typically enter into estrus in the Spring when days become longer. Because of their polyestrous nature, queens may cycle multiple times until they become pregnant. Additionally, because they are induced ovulators, and produce large litters, fecundity tends to be high. This reproductive efficiency is in part responsible for feline overpopulation and subsequently, a demand for spay and neuter clinics and programs.

In addition to preventing pet overpopulation and animal suffering, the practice of gonadectomy is also indicated to prevent or reduce the risk of certain acquired diseases, for example, endometritis/pyometra, uterine and ovarian neoplasia, and prostatic disease. However, as with any intervention, potential adverse consequences also exist. As a professor of small animal theriogenology at the University of Minnesota, College of Veterinary Medicine recently stated, “we change animals when we spay and castrate them, both in good and bad ways”.

This session focuses on trying to answer the following questions from a soft tissue, orthopedic and oncologic perspective: **should** we be recommending gonadectomy for the **majority** of non-breeding animals; and if so, what is the **ideal timing**?

### Traditional age at gonadectomy

It seems that most US veterinarians are advocating elective gonadectomy between 6-9 months of age, and before the first estrus in bitches and queens. It is historically unclear what this recommendation is based on. Performing the procedure prior to skeletal and sexual maturity of the patient (generally under 12 months of age for most breeds) is considered “early”.

As previously stated, pet overpopulation remains an ever-increasing problem locally and worldwide. As a result many pet shelters have adopted a policy of only adopting out pets that have undergone gonadectomy, which may be performed as early as 2 months of age.

## SOFT TISSUE/GENERAL PERSPECTIVE

### Considerations for male dogs

*Prostatic hypertrophy and perineal herniation* - Castration prevents benign prostatic hypertrophy and significantly reduces the risk for developing prostatitis or prostatic cysts. Castration is also therapeutic and an essential component to the treatment of these diseases. Fifty percent of intact male dogs have prostatomegaly and histology consistent with Benign Prostatic Hypertrophy (BPH) by 5 years of age.<sup>3</sup> Further, castration significantly reduces the risk for other associated acquired diseases like perineal herniation, which leads to significant suffering and morbidity in intact dogs. For example, clinical signs and complications associated with perineal herniation include: dyschezia, obstipation, bladder retroflexion, tenesmus, rectal prolapse,

incontinence, infection, surgical wound dehiscence and recurrence. In the author's opinion, castration is a mandatory component of surgical treatment for perineal herniation and is associated with an increased risk of failure if not performed in conjunction with herniorrhaphy. The increased risk of surgical failure likely has to do with persistent obstruction of the pelvic canal by the enlarged prostate as well as continued production of androgens, which are responsible for weakening of the pelvic diaphragm muscles. Perineal herniation in castrated males is exceptionally rare and is a major rationale for the neutering of male dogs, especially those breeds at increased risk.

*Testicular/prostatic neoplasia* - Castration prevents the development of testicular neoplasia (e.g. sertoli cell tumor, leydig cell tumors, and seminomas), orchitis and testicular torsion and significantly reduces the risk for secondary prostatic disease. Risk for prostatic neoplasia (both carcinoma and adenocarcinoma) in neutered dogs has been determined to be up to 2.8 times greater than that of intact male dogs but the older age of neutered dogs may be a confounding factor in some of these studies.<sup>4-6</sup>

### **Considerations for female dogs and cats**

*Urinary Incontinence* - Post-spay urinary incontinence is reported in up to 75% of dogs following gonadectomy as documented via urethral pressure profilometry. The proposed mechanisms include: alteration of hormones such as estrogen and gonadotropin, decreased urethral smooth muscle mass and blood flow, as well as a reduced functional urethral length. Gonadectomy prior to 3 months of age was associated with an increased risk of urinary incontinence compared to dogs spayed between 3 months and 1 year.<sup>7</sup> Consequently, for female dogs the recommendation to delay gonadectomy until at least 3 months of age has been recommended and may be beneficial. However, a recent systematic review of the veterinary literature has brought into question the power of these studies and concluded that the evidence is not consistent or strong enough to make firm recommendations on the effect of neutering or age at neutering on the risk of urinary incontinence.<sup>8</sup>

*Lower urinary tract disease in cats* - Few studies have examined the association of lower urinary tract disease in cats based on neuter status. A study by Howe et al. in 2000 demonstrated no increased risk for lower urinary tract disease in early neutered cats with follow-up out to 3 years. Alternatively, neutered male (versus intact) and obese cats had an increased risk of lower urinary tract disease in another study.

*Endometritis/Pyometra* - Pyometra is defined as septic suppurative inflammation of the uterus and is a consequence of a primed uterus, which requires the influence of progesterone. Progesterone levels increase during diestrus. Increasing progesterone levels cause 1) reduction in uterine motility 2) increase in glandular secretions and 3) constriction of the cervix. These effects create an ideal environment for growth of bacteria should the region become inoculated. Inoculation of the vagina and uterus occurs regularly from skin flora and perineal and rectal microflora. The risk is approximately 25% for intact dogs greater than 10 years of age. Therefore, gonadectomy is preventative or curative for pyometra. Pyometra is more rare in female cats but uterine disease in queens was found to be more likely after 5 years old in one study. Because of the relatively high risk and potentially lethal nature of pyometra in dogs, gonadectomy is advised.

### **MUSCULOSKELETAL PERSPECTIVE**

Historic studies of the skeletal development in eunuchs have shown us that removal of hormonal influences in the immature animal results in delayed physal closure.<sup>9,10</sup> The impact of this conformational change on later development of orthopedic disease is most notable in

canine cranial cruciate ligament disease, canine hip dysplasia, and feline capital physeal fractures.

### **Cranial Cruciate Ligament (CrCL) Disease**

A retrospective study by Duerr et al. (2007) found that in a population of large breed dogs, those that had early gonadectomy had a 3-fold increased risk of excessive tibial plateau angle and subsequent predisposition for early CrCL injury. Numerous other studies across all breeds have shown that neutered male and spayed female dogs have 2-3 times the incidence of CrCL disease vs their intact counterparts.<sup>12,13</sup> A recent study in 750 golden retrievers had no CrCL disease in their intact dogs vs 5% and 7.7% incidence in the early neutered males and females respectively.<sup>14</sup> This study did not see a significant difference between the BCS of the neutered male and female population with and without CrCL disease, suggesting that conformation change and not just increased body weight associated with gonadectomy is responsible.<sup>14</sup>

*Hip dysplasia (HD)* - Gonadectomy, and the timing of surgery, also appears to have an influence on the development of canine HD, although the effects appear to be somewhat gender and breed specific. In the afore mentioned golden retriever study out of UC Davis, incidence of hip dysplasia in the early neutered male population was double that of intact males (10% vs 5% respectively). Early neutered male retrievers also had an earlier onset of their disease (3.6 years) vs their late-neutered (4.7 years) and intact (4.4 years) counterparts. Similar to CrCL disease, BCS of the early neutered male population with or without HD was not significantly different. Female golden retrievers did not show any significant differences in incidence of HD between the spayed or intact populations.<sup>14</sup>

A similar trend has been noted with neutered boxers having 1.5 times the risk of developing HD, with an average age of 3 years at the time of gonadectomy.<sup>15</sup> Another breed and sex non-specific study showed gonadectomy increasing the likelihood of HD by 17%.<sup>12</sup> Spain et al. (2004) noted that if gonadectomy was performed before 5.5 months of age, the incidence of HD was 6.7% vs 4.7% if the puppies were neutered between 5.5 months and 1 year of age. The older group had a subsequent higher euthanasia rate vs the younger group leading the investigators to suggest that despite the higher prevalence of HD, early-age gonadectomy may lead to a less severe form of HD.<sup>7</sup>

*Fractures* - Overweight, neutered male cats are predisposed to spontaneous femoral capital physeal fractures. This is thought to be a result of delayed physeal closure related to early neutering.<sup>16,17,18</sup> There does not appear to be any association between incidence of other fractures and gonadectomy in dogs or cats.<sup>7,19</sup>

*Obesity* - Obesity plays a large contributing factor in the development and progression of a multitude of orthopedic disease and osteoarthritis. Gonadectomy is the most commonly reported risk factor for obesity. Although the timing of gonadectomy, and the exact cause-and-effect relationship between gonadectomy and obesity is not well defined, the importance of a controlled diet and exercise regime post gonadectomy is clear.

### **ONCOLOGIC PERSPECTIVE**

There has been a lot of recent attention in the veterinary literature to determine whether or not gonadectomy may have an effect on cancer risk. This has created a lot of questions for veterinarians about the common practice and recommendation to spay and neuter dogs and cats early. Studies that evaluate the effect of spaying and neutering retrospectively are inherently difficult to perform because to date, they have all been retrospective. There are many

other factors that may contribute to the development of cancer that are difficult to capture in this type of study. Oncogenesis is multifactorial. Neuter status may play a role, but the patient's genetics and environmental exposure likely are also important factors. One potential confounding factor of evaluating the risk of cancer development with neutering is that spayed and neutered animals are known to have an increased life-span. This alone will increase the risk of developing cancer and it is difficult to separate these factors out retrospectively. Another potential confounding factor is that owners that spay and neuter their pets may have an increased tendency to seek medical attention for their pets and to commit financially to the diagnostics necessary to definitively diagnose cancer. Because most of the retrospective studies on the effect of gonadectomy and cancer are performed via data bases at veterinary teaching hospitals, there is a potential for selection bias because these will tend to be referral cases rather than cases from the larger pet population. Another precaution is the source of the information. The paper that has received the most attention recently is an article in the on-line journal pLOS. This journal is an open-access journal that is not peer-reviewed. This does not automatically mean that the information is not valid. However, this type of journal relies on the readers to serve as reviewers and to scrutinize the information presented carefully. There is, however, some emerging evidence that gonadectomy may increase the risk of developing some forms of cancer.

*Mammary neoplasia* - Most veterinarians can recite by rote the statistics on the preventative effects of early spaying on mammary tumor development. Before the first heat, the lifetime risk is 0.5%, between the first and second heat, this risk is 8%, and after the second heat, the risk is 26%. This original article was authored by Schneider et al and was published in the Journal of the National Cancer Institute in 1969. It has been cited in the veterinary literature > 156 times. The effect of estrogen exposure is thought to be the cause of mammary neoplasia development in intact bitches and queens. We also know that in countries that do not routinely spay their dog population, mammary tumors are extremely common. However, a recent Cochrane metaanalysis in veterinary medicine by Beauvais et al has suggested that the evidence that early spaying prevents the risk of neoplasia in the current veterinary literature is weak. This is most likely because the studies are weak, rather than suggesting that mammary tumors are not prevented by early spaying. In cats, intact queens have a reported seven times increase in developing mammary neoplasia. This risk is decreased by 91% and 86%, if performed before the first and second heat, respectively.

*Reproductive organ tumors* - The removal of the reproductive tract in females will eliminate the risk of ovarian neoplasia and uterine neoplasia. It will also almost completely eliminate the risk of vaginal leiomyomas. In male dogs, there will be a corresponding elimination of the risk of testicular tumors and almost complete elimination in the risk of perianal gland tumors with neutering. Neutering however, has been shown to increase the risk of prostatic carcinoma.

*Other common cancers* - The risk of the development of osteosarcoma has recently been reported to increase in spayed and neutered dogs. This risk has been reported to vary between no risk in some articles to 1.3-1.9 times the risk in spayed or neutered dogs. One study that evaluated Rottweillers exclusively found that neutered males and females were at an increased risk. However, this article also had a significant age differential between neutered (older) and unneutered (younger) patients. Because osteosarcoma often presents in older patients, it is important to be cautious when evaluating this data.

Hemangiosarcoma has also been recently reported to occur with increased risk in females that are neutered late compared to females neutered early or intact females. A study in Vizlas also found that spayed females were at an increased risk. The next step

is to decide what do to with this information. Do we hold off on spaying and neutering, or do we go ahead, but perform a prophylactic splenectomy for high-risk breeds? Other tumor types that have been shown to have an increased risk in the neutered population include lymphoma, transitional cell carcinoma and mast cell tumors.

When evaluating the risk of various cancers with relation to spaying or neutering, the veterinarian will need to look at every patient as an individual and determine with the owner this risks and benefits of the procedure. Currently there is data suggesting that spaying and neutering may carry an increased risk of cancer in some breeds, but the data is inherently weak because it is retrospective and because there is a great deal of selection bias that can not be corrected for. A knee-jerk response to hold off on spaying and neutering will likely lead to an increase of reproductive tract tumors and other health and social problems related to having a pet population that is largely intact. Increased surveillance of at-risk breeds that have been spayed or neuter for the cancers that they are predisposed to may be a more moderate approach to managing this information. There is currently a Morris Animal Foundation study that is following 3000 golden retrievers over their lifetime. <http://www.caninelifetimehealth.org> Because this study is prospective, it may help shed some light on these important questions. We cannot assess neuter status in isolation with respect to the risk of developing cancer because it is only one factor in many that lead to the development of cancer.

## CONCLUSIONS

Many potential benefits and risks exist when deciding to spay or neuter a dog or cat. In an ideal world, each of these benefits and risks would be discussed with companion animal care-givers and a decision made based on an accurate evidence-based body of data. However, given the limitations with the current available veterinary literature as well as the frequency of conflicting conclusions, evidence-based decision-making is tremendously difficult. Further, given the societal concern for animal suffering, frequency of unwanted euthanasia, the limitations of animal shelters, and the greater risk for euthanasia for unaltered animals, a strong movement will persist which advocates for spay and neuter of cats and dogs.

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# **FOCUS ON INFECTION PROGRAM**

## Canine influenza

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### Introduction

Canine influenza has caused much concern in veterinarians, dog owners and public health personnel. From the emergence of H3N8 canine influenza virus (CIV) in the US in the early 2000s,<sup>1</sup> to the ongoing and broad international dissemination of H3N2 CIV,<sup>2-5</sup> this disease has had a significant impact. The scope of disease is not known, but it is estimated that thousands (or, more likely, tens of thousands) of dogs have been infected in the US, with recent introduction of the virus to Canada. Serious infections, including fatal infections, have occurred and while the overall medical and economic impacts are unknown, they are clearly substantial. CIV has also highlighted various areas such as risk of imported pathogens, spread of disease in a naïve population, the need for a coordinated human/veterinary response to this potentially zoonotic pathogen and the need to consider optimal vaccination strategies for an emerging disease.

### Epidemiology/Transmission

Canine influenza refers to a influenza virus that is host-adapted to live in dogs and circulate in the dog population. Dogs are susceptible to a range of influenza viruses, including human influenza viruses,<sup>6</sup> but infections with those are sporadic and not sustained in the dog population. Canine influenza viruses can spread readily between dogs, without the need for any other hosts.

There are two CIV strains of concern. H3N8 is an equine-origin virus that emerged in dogs in the early 2000s, being the cause of outbreaks in greyhounds in Florida.<sup>1</sup> This strain subsequently spread in a patchy manner and was found in sporadic disease and outbreaks in various parts of the US.<sup>1,7,8</sup> It has been rarely identified in recent years and may not be circulating widely (or at all) any more.

H3N2 CIV is an avian-origin virus that emerged in Asia.<sup>9</sup> It is widespread in some Asian countries, especially China and South Korea, and there have been multiple introductions into North America through importation of infected dogs.<sup>4</sup> Currently, this virus is present in many US states, with thousands of dogs likely infected.

As is common for influenza viruses, CIV can be shed for a short time (e.g. 24h) prior to the onset of clinical signs, with peak shedding during early disease. Duration of shedding differs between H3N8 and H3N2, with H3N8 being shed typically for only a few days and H3N2 being shed for up to a few weeks. This may account for the wider and more sustained transmission of H3N2, as longer shedding creates more opportunities for infection.

Transmission of CIV can be via direct contact, droplet transmission (transmission over a short distance from infectious aerosols generated during breathing, barking or coughing) and indirect transmission via fomites or the environment. The relative roles of these routes are not known, but direct contact is probably the most important route. Influenza viruses are not particularly stable in the environment and do not persist for long periods of time, so indirect transmission is probably short term and mainly involving items and surfaces that have direct contact with respiratory secretions (e.g. bowls, human hands).

Cats can be infected with a range of influenza viruses but appear to have low susceptibility to CIV and while infections have been reported, they are rare.<sup>10,11</sup>

### Canine influenza virus in Canada

When H3N8 CIV emerged in the US in the early 2000s, it was assumed that it would inevitably and shortly enter Canada through regularly transborder dog movement. However, this virus did not establish a foothold in Canada. A small and poorly documented cluster of infections occurred in British Columbia, but no other cases were identified. In Ontario, an initial seroprevalence study only identified one seropositive dog, and that dog had originated in the US.<sup>12</sup> Another seropositive dog that had not left the country was subsequently identified; however, housemates of it were show dogs that travelled to the US, so it is assumed that the dog was infected through that route. The lack of emergence of H3N8 CIV in Canada was surprising but perhaps is explained by the relatively short time infected dogs shed the virus, reducing the potential for transmission with moving dogs.

The emergence of H3N2 CIV led to renewed concerns about CIV in Canada, with the potential for increased risk because of the longer duration of shedding of some infected dogs and the increasing amount of importation of dogs from China and South Korea. Indeed, H3N2 CIV was introduced to Ontario at the end of December 2017, with further importation in early 2018. With prompt diagnosis, coordinated response, isolation of infected animals and comprehensive contact tracing, it appears that CIV was contained and (at least for a while) eradicated from Canada, demonstrating the potential impact of a coordinated and aggressive response to CIV, even with a highly transmissible virus in a naïve population.

### Clinical Disease

Canine influenza is often described as having two different syndromes, typical upper respiratory tract infection and severe pneumonia. In reality, it is more of a gradation, with most cases having self-limiting upper respiratory tract disease and a small percentage developing varying degrees of more severe, lower respiratory tract disease. Additionally, subclinical infection can occur, resulting in clinically normal dogs that are actively shedding CIV.

Most clinically affected dogs develop upper respiratory tract disease with varying degrees of cough, nasal discharge, ocular discharge, lethargy and anorexia. Fever may be present but is inconsistent. Secondary bacterial pneumonia may develop in a minority of cases. Severe peracute lower respiratory tract disease has also been reported and can result in rapid deterioration (including death) within a short period of time. How much of that is due to an abnormal presentation of primary viral disease versus severe peracute secondary bacterial infection is usually difficult to discern.

### Diagnosis

Clinical signs are not adequate for diagnosis since CIV infection is not distinguishable from disease caused by other CIRDC pathogens. Suspicion should be raised in some situations, such as high morbidity outbreaks in groups of dogs vaccinated against *Bordetella bronchiseptica* and canine parainfluenza virus and in dogs imported from Asia, but diagnostic testing is required.

PCR is the most widely used test. PCR assays can be broad influenza A tests or specifically target H3N8 or H3N2. It is useful to have strain-specific results as that can help influence infection control measures, particularly isolation recommendations. H3N8 shedding is often fairly short-term,<sup>13</sup> so false negative samples can occur if samples are not collected early in disease. This is less of a concern for H3N2 since it is usually shed for longer durations,<sup>14</sup> but false negative results can occur, so influenza cannot be definitively ruled out on the basis of a single negative test. Testing can involve one or more of nasal, pharyngeal and conjunctival

swabs. Conjunctival swabs are likely the lowest yield and nasal swabs are most often used. Testing of 2 or 3 sites in parallel likely increases sensitivity of diagnosis.

Serological testing can be used for diagnosis, but is less useful clinically because of the need to test convalescent titres. A four-fold or greater increase in serum antibody titre in samples collected ~4 weeks apart is diagnostic in the absence of recent (probably within the past few weeks) vaccination. Single serum samples are not particularly useful. They might be more useful in areas where CIV is known not to be endemic and in an unvaccinated animal. However, diagnosis via detection of seroconversion is most useful when respiratory samples cannot be collected or were not collected early in disease and a false negative PCR results is of concern. Virus isolation is not routinely used clinically because of limited access and turnaround time. It is predominantly a research tool to obtain viral isolates for research, molecular epidemiology and to identify new influenza strains.

### **Treatment**

Treatment, as for other causes of CIRDC, is mainly supportive.<sup>15</sup> Most cases of CIV infection respond well to supportive care, such as cough suppression. The type and level of care that is needed (e.g. IV fluids, oxygen support) is dictated by the severity of CIRDC, not by the pathogen. Most affected dogs are easily managed at home. Secondary bacterial infections are not common but may occur, and should be managed as per standard approaches.<sup>15</sup> Antiviral drugs are uncommonly used in humans and the approach should be similar in animals. While drugs such as oseltamivir might be useful in some situations, they are not likely indicated in the vast majority of cases. They could be considered in severe, acute cases, particularly in high risk (e.g. brachycephalic) patients, but evidence is currently lacking.

### **Infection Control Practices in Veterinary Clinics**

CIV is readily transmissible in situations where there is abundant dog-dog contact or where there is a high likelihood of cross-contamination. These events can occur in veterinary clinics, and clinics have been foci for CIV transmission. Some large outbreaks have resulted in temporary clinic closure or restriction of activities as a means of stopping transmission. Reducing the risk of CIV transmission in clinics focuses on prompt identification of suspects (not waiting for definitive diagnosis), isolation of potentially infectious individuals, use of contact precautions (e.g. gloves, gown or dedicated outerwear) with potentially infectious individuals and the use of good routine general infection control practices (e.g. hand hygiene, proper use of routine protective equipment, cleaning and disinfection) for all cases because of the potential for subclinical shedding. CIV is readily inactivated by routine disinfectants, provided they are used properly.

### **Prevention**

There are two main approaches to prevention, reducing exposure and increasing resistance. Reducing exposure can be a challenge when CIV is endemic in a region. In that situation, basic practices such as limited the number of contacts with different dogs, reducing contact with high risk (e.g. kennel) populations and avoiding contact with sick dogs should be considered. In non-endemic areas, avoiding contact with dogs are higher risk for introducing the virus, particularly dogs imported from Asia, is important.

Increasing resistance is focused on vaccination. Monovalent H3N2 or H3N8, and bivalent vaccines, are available, but at the time of writing, only the bivalent vaccine was available in Canada. It is questionable whether H3N8 vaccination is required since this virus seems to be very rare in North America (if it is even still circulating). As is typical for killed vaccines, an initial two dose series is required. The timeframe required for two doses and subsequent development

of a protective immune response means that vaccination is not a highly effective initial outbreak response tool, since much transmission can happen during this window of developing resistance. However, vaccination can likely play a useful role at both the individual and population (herd immunity) levels, particularly in endemic regions and in high risk animals. While vaccination can be considered for any patient, vaccination is perhaps most important in dogs at higher risk of exposure (e.g. those exposed to imported dogs or dogs from other endemic regions, dogs that have frequent contact with other dogs) and those at increased risk of severe infection (e.g. seniors, dog with underlying respiratory or cardiovascular disease, brachycephalics).

### **Zoonotic Concerns**

Influenza A in animals inherently raises public health concerns. The public health risks with H3N2 CIV seem to be very low, but there are two areas to which attention is being paid. The first is transmission of H3N2 CIV to humans. Human infections with H3N2 CIV have not been reported. While there is endemic H3N2 human (seasonal) flu and human infections with H3N2 swine flu have been reported, these flu strains are quite different and there is little concern about direct transmission of H3N2 CIV to people. However, it cannot be completely ruled out so practical infection control measures to improve hygiene (e.g. hand hygiene) and limit direct contact with respiratory secretions of infections dogs are prudent. The greater concern is the potential for dogs to act as a 'mixing vessel' for influenza viruses,<sup>16</sup> since dogs can rarely be infected with human influenza strains. In this type of scenario, concurrent infection with CIV and a human influenza virus could result in recombination of the two viruses. If this resulted in a new virus that was able to infect humans, cause disease and be transmissible between humans, a new influenza strain would result. If this strain was antigenically different enough from human influenza viruses that there was no cross-immunity was present, the result would be a new virus to which the human population has no protection.

**References are available upon request**

## Antimicrobial Stewardship: A Human Health Perspective

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### Introduction

Antimicrobial stewardship is a relatively new term, coined in 1996 by Drs. John McGowan and Dale Gerding to describe attempts to make better use of antimicrobials by reducing unnecessary overuse and optimizing necessary use. Over time, antimicrobial stewardship in human health has taken on increasing importance, as it is juxtaposed with increasing antimicrobial resistance, increasing healthcare costs, ongoing struggles with healthcare-associated infections, and a shrinking antimicrobial development pipeline.

Antimicrobial stewardship, as formally defined, is coordinated interventions designed to improve and measure the appropriate use of [antibiotic] agents by promoting the selection of the optimal [antibiotic] drug regimen including dosing, duration of therapy, and route of administration. Put simply, antimicrobial stewardship is making sure that patients get the right antibiotics when they need them, and only when they need them. Recognizing that the term was coined just over 20 years ago, antimicrobial stewardship remains in its infancy.

### Metrics in Antimicrobial Stewardship

Because antimicrobial stewardship is, at its core, a quality improvement initiative, gauging success (or failure) requires being able to measure. Antimicrobial stewardship is meant to serve a variety of purposes.

#### Purposes of Antimicrobial Stewardship

Some of the purposes are specific to human health, and include improving the appropriateness of antimicrobial prescribing resulting in:

- improved patient clinical outcomes
- decreased drug side effects
  - allergic reactions
  - adverse reactions/side effects (e.g. liver, kidney, and bone marrow toxicity)
  - antibiotic-associated infections, such as those due to *Clostridium difficile*, and *Candida* species
- reduced the development of antimicrobial resistance
- preserved patients' microbiomes (i.e. their endogenous microbial flora)
- decreased drug (and other) costs

Other purposes include animal health and the environment, and include:

- reducing environmental antimicrobial contamination
- reducing antimicrobial drug resistance in the geosphere and hydrosphere (i.e. the ecosphere), including land animals and aquatic life
- reducing antimicrobial resistance acquisition via the food/water supply

#### Antimicrobial Appropriateness

Accordingly, antimicrobial stewardship metrics should be focused on "appropriateness". Unfortunately, there are no reliable methods to ascribing appropriateness. There are currently two approaches to assigning appropriateness:

- i. compared to accepted standards or clinical practice guidelines
- ii. compared to peers (i.e. looking at outliers)

Comparing to clinical practice guidelines is challenging in Canada because there are no accepted sets of clinical practice guidelines for infectious diseases. Similarly, there are few opportunities to compare antimicrobial stewardship practices to peers. Finally, any other attempts to ascribe appropriateness are based on “expert opinion” rather than a standard. Antimicrobial appropriateness therefore remains, largely, a distant dream in human healthcare.

### **Antimicrobial consumption**

The most common approach to measuring antimicrobial use is to report on antimicrobial consumption. Metrics include defined daily doses (DDD)/1000 patient-days, where a DDD is a standard daily quantity of an antimicrobial. For example, 2g of ceftriaxone each day is 1 DDD. If 500 out of 1000 people get 2g ceftriaxone daily, their DDD is 500 DDD/1000 patient-days. However, if they only get 1g ceftriaxone daily, their DDD is 250 DDD/1000 patient-days or—for outpatients—DDD/1000 patients, or DDD/1000 inhabitants/year. The other method of measuring antimicrobial consumption is days of therapy (DOT)/1000 patient-days. Finally, for outpatients, no. of prescriptions/1000 patients or 1000 inhabitants/year is also a common method. Each method has its strengths and weaknesses, including accounting for body mass, renal function, etc.

### **Antimicrobial cost**

Perhaps the easiest metric to track and report is antimicrobial cost or expenditure. Costs are readily available, and can be used for inpatients or outpatients. They do not, however, properly account for changes in pricing, and so are weighted against on-patent medications that tend to be substantially more expensive than generic medications.

### **Measuring Outcomes from Antimicrobial Stewardship Efforts**

As mentioned above, there are various stated purposes for stewarding antimicrobials, that include reducing the Darwinian selection of antibiotic-resistant organisms (i.e. either resistant variants of common organisms or the emergence of organisms with constitutional drug-resistance), reducing the adverse effects of antibiotics, and improving patient outcomes. Each of these are ideally followed in antimicrobial stewardship efforts. However, the challenges are:

- a) reporting mechanisms
- b) measuring outcomes such as “resistance” (we end up tracking rates of antibiotic-resistant organisms, but those aren’t the same as measuring resistance).

### **The Four Forms of Inappropriate Prescribing**

Antimicrobial prescribing can be inappropriate for 4 basic aspects:

1. Patient needs an antibiotic, but it is either not prescribed or does not cover the infectious agent(s). This is **UNDERTREATMENT**.
2. Patient needs an antibiotic, but the antibiotic is prescribed too high a dose, too long a duration, is unnecessarily broad-spectrum, or is administered via the incorrect route. This is **EFFECTIVE BUT UNNECESSARY**.
3. Patient needs an antibiotic, and the choice, duration, dose, etc. is optimal. This is **APPROPRIATE**.
4. The patient does not need an antibiotic but is being prescribed an antibiotic, or is being prescribed an antibiotic but shouldn’t because of allergy. This is **INAPPROPRIATE**.

Measuring appropriateness, however, must be manual, making this a difficult metric.

## **Sports Analytics**

Analytics have transformed a variety of elite sports. It has the potential to transform antimicrobial stewardship, but we need to develop systems of making the data available. At present, we don't have the data systems in place.

The analytics in sports era was transformed by Miky Tamir and Gal Oz, whose firm, SportVU, allowed NBA teams to receive an exorbitant amount of data and then analyze that data to their advantage. It changed so much that it created scoring "geniuses" like Stephen Curry who stopped shooting mid-range shots and instead started shooting shots from far out, that were worth 50% more if made. It allowed coaches and players to understand what to target, what to improve upon, and what to ignore. We need similar data in healthcare, but there is currently no system like SportVU that allows us to perform these kind of analytics. Hospitals are starting to get this information, as are some healthcare systems, but it is still in its infancy.

## **Behavioural Economics and Nudging Behaviour**

We are now recognizing that human prescribing behaviour is complex, but is motivated in a similar manner to other consumer-based behaviours like purchasing, eating, etc. Accordingly, looking at prescribing as a behavioural economic challenge has gained both greater traction and greater success more recently. This includes looking at a framework of behaviour that is dependent on motivation, capability, and opportunity. These can each be influenced by a variety of mechanisms, including education, training, coercion, modeling, restrictions, etc. The Behavioural Change Wheel is a useful tool for this.

## **Civil Society**

Learning from other initiatives requiring a massive mobilization of resources and effort (e.g. HIV, climate change), we are only now starting to realize that science and health care providers alone are not going to change the status quo. We need to engage civil society to support behaviour change efforts and to motivate government to resource efforts appropriately.

## HONEY BEE HEALTH IN ONTARIO: HOW BEEKEEPERS AND VETERINARIANS CAN WORK TOGETHER

Paul Kozak (Provincial Apiarist / Apiary Specialist – Ontario Ministry of Agriculture, Food and Rural Affairs) and Les Eccles (Lead Technology Transfer Program Specialist – Ontario Beekeepers' Association)

Apiculture (beekeeping) is a diverse agricultural industry that combines elements of livestock husbandry, entomology and crop management. While beekeeping has parallels with other sectors that veterinarians are involved in there are some key differences (invertebrates, the concept of managing units of colonies that function as a superorganism). And like all sectors in agriculture, where there parallels there are key differences that make each unique. Furthermore, there are key differences within the beekeeping industry – internationally, within North America and within Canada whereby the Ontario beekeeping sector is its own unique industry. Pest and disease status, demographics, environmental and geographical influences, overlap with the other sectors of agriculture and the general public, and regulatory requirements and services are among many of the details that are unique within beekeeping within Ontario.

This session is not intended to train veterinarians in apiculture, rather to provide an overview of apiculture in Ontario and highlight future training opportunities, resources and recently developed models for how beekeepers and veterinarians may work together. This is especially important as the antibiotics that beekeepers use will fall under a veterinarian prescription on Dec 1, 2018.

### Beekeeping in Ontario:

There are just over 3,000 registered beekeepers in Ontario operating approximately 100,000 honey bee colonies at over 6,000 registered locations (apiaries) across Ontario. Most of the colonies (~80%) are operated by commercial beekeepers (beekeepers with 50 or more honey bee colonies and up to ~10,000 colonies). Commercial beekeepers may be focused on honey production or diversified (production of honey bee queens and colonies, pollination of domestic or out of province crops, value added colony products, sales and retail of honey bees and bee related equipment). Commercial beekeepers may operate numerous bee yards (3 to >100) with numerous colonies at each location (10 to 1,000). While small-scale beekeepers account for the other ~20% of the honey bee colonies they account for most of the beekeepers (~2,800 beekeepers) some may only operate less than five colonies. However, they are important part of the industry, for purchasing bees from beekeepers, additional honey production and distribution and as a risk for honey bee pests and diseases.

All of these figures vary from year to year and rely on beekeeper registration with the province through the Ontario Apiary Program. In Ontario, like most jurisdictions in North America, honey bees are regulated. The Bees Act of Ontario and associated regulations (see appendix 1) are the primary legislation that regulates beekeeping in the province. Some key details of the act and requirements under the act: beekeepers must register with the Provincial Apiarist, A Provincial Apiarist is appointed under the Bees Act as well as Apiary Inspectors (both under the Ontario Ministry of Agriculture, Food and Rural Affairs), beekeepers can be inspected for pests

and diseases specifically named under the act or regulations and that are specific to honey bees, beekeepers require permits for selling or transferring honey bee colonies, material and used beekeeping equipment, Apiary Inspectors and the Provincial Apiarist may issue orders of treatment, detainment or destruction of honey bee colonies and / or equipment.

Most beekeeping legislation in North America is specific to honey bees as their biology is unique and their health issues are very species specific (most of their pest and diseases are species specific and do not impact vertebrates). While there are a few honey bee pests named under the regulations that have the potential to injure or kill livestock and / or people (species of wasps that are pests of honey bees, Africanized honey bees) the majority of the Bees Act and regulations is focused on the health of honey bees, primarily through the transmission of honey bee diseases.

#### Resources for Apiculture:

In addition to regulation of the beekeeping sector there is a lot of advisory and extension information available to Ontario beekeepers. The main delivery agents are: the Technology Transfer Program (Ontario Beekeepers' Association)(2); the University of Guelph – Honey Bee Research Centre(3); The OMAFRA Apiary Program (4) and more recently Niagara College – Commercial Beekeeping Program. These programs also collaborate and produce guidelines, best management practices (5), treatment recommendations (6), workshops and training to beekeepers (both commercial and small-scale and experienced and novice). This information references materials from CFIA (National Biosecurity Standard for Honey Bees), Health Canada (Pest Management Regulatory Agency for registered treatments for honey bees pests – e.g. varroa mites), recent research with applied and foundational research represented from Ontario, grey literature, program statistics and beekeeper input.

While there are other sources of information for beekeepers, including researchers at Universities, government programs and specialists, many of these resources are delivered through the OBA or local beekeeping associations. This is becoming increasingly important as the distribution of information and resources is expanding through the internet, often with information that is not always accurate, properly sourced or even regionally relevant. This is not a phenomenon unique to honey bees, as there are many sources of information on human and animal health that the public is using where ease of access is more heavily weighted.

More recently, the upcoming requirement for beekeepers to get a prescription from a veterinarian for antibiotics resulted in the formation of an Ontario working group that selected membership included Ministry, industry and academic specialists in apiculture, regulators, representative veterinarians, representative commercial beekeepers. This group worked closely with OMAFRA, the Ontario Veterinary Medical Association and College of Veterinarians of Ontario to ensure the process and guidelines. This strategy developed options for how veterinarians can work with beekeepers in a manner that is straightforward, yet address the main requirements of each side – a meaningful VCPR for veterinarians that addresses requirements of such as relationship and a meaningful delivery of information on honey bee health and management between the two sides.

**Listing of References:**

- 1) Ontario Ministry of Agriculture, Food and Rural Affairs – Apiculture:

Regulation of the Beekeeping Industry

<http://www.omafra.gov.on.ca/english/food/inspection/bees/apicultu.html#regulation>

Bees Act of Ontario

<https://www.ontario.ca/laws/statute/90b06>

Regulations Under the Bees Act

<https://www.ontario.ca/laws/regulation/900057>

Honey Bee Registration Form

[http://www.omafra.gov.on.ca/english/food/inspection/bees/info\\_registration.htm](http://www.omafra.gov.on.ca/english/food/inspection/bees/info_registration.htm)

- 2) Technology Transfer Program – Ontario Beekeepers' Association

<https://www.ontariobee.com/outreach/ttp>

- 3) University of Guelph – Honey Bee Research Centre

<http://www.uoguelph.ca/honeybee/>

- 4) OMAFRA Apiary Program

<http://www.omafra.gov.on.ca/english/food/inspection/bees/apicultu.html>

- 5) Best Management Practices in Advance of Winter

<http://www.omafra.gov.on.ca/english/food/inspection/bees/bmpwinter.htm>

- 6) Ontario Treatment Recommendations for Honey Bee Disease and Mite Control

<http://www.omafra.gov.on.ca/english/food/inspection/bees/2017-treatment.htm>

## EXPANDING RANGES AND CHANGING RISK: WHY ECOLOGY MATTERS WITH TICKS AND TICK-BORNE DISEASE

Katie M. Clow, DVM, PhD

Over the past two decades, there have been notable changes in Canada's tick populations. Both the diversity of species and abundance of ticks has increased, in part due to climate change and other ecological changes [1-3]. Many of these tick species are vectors for pathogens of human and animal health significance. An understanding of the ecological factors that influence tick populations and tick-borne disease is vital to assessing and predicting risk to animal and human health.

### Tick Ecology

Ticks are within the class Arachnida and Order Ixodidae. They are further divided into three families: Ixodidae (hard ticks), Argasidae (soft ticks) and Nuttalliellidae. Ticks develop from eggs into larvae, then nymphs and finally adults, which is the mature reproductive stage.

In Canada, there are over 40 species of ticks – 32 of which are hard ticks and 8 of which are soft ticks [4]. Hard ticks are characterized by their hard dorsal shield. They are active during the day, and feed once per life stage for an extended period of time (days). In contrast, soft ticks lack the hard dorsal shield, are generally active at night and feed multiple times per life stage for short periods of time (minutes to hours) [4].

Ticks can be labelled as one-host, two-host, three-host or multi-host ticks, depending on their feeding patterns. A one-host tick spends most of its life on one host, taking a bloodmeal during each life stage from the same host. The only one-host species of tick in Canada is the moose tick, *Dermacentor albipictus*. Three-host ticks take a bloodmeal on a different host during each life stage and spend the rest of their life (~98%) in the environment. There are no two-host ticks in Canada, and all multi-host ticks are soft ticks [4].

When considering the tick species in Canada that pose a risk of pathogen transmission, all are three-host hard ticks. Each of these species has specific abiotic and biotic factors that influence its distribution, including climate, habitat and hosts.

Climate can be considered at two-levels: microclimate and macroclimate. The microclimate refers to the immediate area where a tick lives and quests, and therefore has a direct impact on the tick's activity, development and survival [5]. The macroclimate describes climate patterns that happen on a larger geographic scale and influence the microclimate. In general, temperature and humidity are the most relevant climatic variables for a tick.

Temperature is a basic requirement for tick survival and development. If the environment does not reach a basic temperature threshold for a specific duration of time, a tick cannot complete its development prior to exhausting its energy stores [6,7]. Also, temperature extremes can lead to increased mortality [7]. Some species of ticks are highly sensitive to low humidity. If humidity drops below a basic threshold, ticks will not be active, and this may also contribute to increased mortality [8].

Other climatic factors, such as precipitation and extreme weather events can influence tick populations, both directly and indirectly. Indirect effects refer to impacts on the tick's habitat and hosts [9].

Tick habitat and hosts are highly variable and should be considered specifically for each species. Most tick species have a specific habitat in which they can survive and thrive. Habitat needs to provide protection, a substrate on which to quest and access to hosts [10]. For hosts, some ticks are generalists and will feed on a variety of species. Other ticks are highly host-specific and will only take a bloodmeal on a select number of species.

The most notable changes in tick populations in Canada have been seen with the blacklegged tick, *Ixodes scapularis*, the groundhog tick, *I. cookei*, the American dog tick, *Dermacentor variabilis*, and the lone star tick, *Amblyomma americanum*. The longhorned tick, *Haemaphysalis longicornis*, has not yet been detected in Canada, but poses a risk of invasion. The ecology of these species will be explored further, with specific emphasis on how ecology is relevant to the changing risk of these species.

### **The blacklegged tick (*I. scapularis*)**

Northward range expansion of the blacklegged tick has been noted in the recent past. Previously, Long Point was the only known population of the blacklegged tick in Canada. Now there are established, reproducing populations present in many areas along the northern shores of Lake Erie and Lake Ontario, a large part of eastern Ontario, the Rainy River area, southern Manitoba, southwestern Quebec, Nova Scotia and New Brunswick [11]. Ongoing range expansion has been detected within a short time frame and is predicted to continue [2].

Several pathogens are transmitted by the blacklegged tick. These include *Borrelia burgdorferi* and *Anaplasma phagocytophilum*, the causative agents of Lyme disease and Anaplasmosis in dogs, respectively [12,13].

The blacklegged tick is a forest-dwelling tick [10]. It can desiccate quickly in areas of low humidity, so it requires a place with leaf litter and protective understory for survival [14]. The primary hosts for blacklegged ticks are small mammals and ground foraging birds for larvae and nymphs, and white-tailed deer and other larger mammals for adults [15].

Each year, millions of blacklegged ticks are introduced into southern Canada by migratory birds [16]. Not all of these ticks will survive and reproduce, but with climate change, many areas where they were previously introduced and could not survive are now becoming climatically suitable for this tick [1]. Climate change may also contribute to the expansion of highly suitable Carolinian forest habitat and the range of white-footed mice [17,18].

### **The groundhog tick (*I. cookei*)**

The groundhog tick is a nest dwelling tick that predominately feeds on groundhogs. It is widely distributed in eastern Canada [4]. It has received attention lately as it is one of the vectors of Powassan virus, which can lead to a fatal encephalitis in humans.

From 2007-2015, the number of submissions of this tick through passive surveillance has more than doubled in Quebec [19]. Similar increases have not been noted in other provinces. More research is needed to determine the reasons why this tick has become more prevalent in Quebec, with climate change being explored as a potential explanation.

### **The American Dog Tick (*D. variabilis*)**

The American dog tick has long been an inhabitant of many areas of eastern and central Canada [4]. Despite documented transmission of several pathogens in the southern United States, including *Rickettsia rickettsii*, no pathogen risk has been detected in the Canadian population of ticks [20].

In Alberta and Saskatchewan, there is evidence of northward and western range expansion of this tick species [21]. It was previously thought that *D. variabilis* and *D. andersoni* (the Rocky Mountain wood tick) inhabited different ecological niches with *D. andersoni* inhabiting hot, drier areas and *D. variabilis* inhabiting warm, humid environments. Now, their ranges overlap with *D. variabilis* being found in environments that were previously thought to be unfavorable (i.e., little to no vegetative cover, sandy soil) [21].

More research is needed to understand why there is a lack of pathogen carriage and transmission by this species in Canada and what ecological factors are facilitating the range expansion in the west.

### **The lone star tick (*A. americanum*)**

Infamous as an aggressive feeder, the lone star tick is the most common adventitious tick in Ontario [22]. Despite regular detection via passive surveillance, there is no evidence that established populations exist in Ontario, or anywhere else in Canada [23]. This species poses a risk as it is the vector for *Ehrlichia chaffeensis* and *E. ewingii* in dogs and *Cytauxzoon felis* in cats [24].

The lone star tick is found in second-growth forests with dense understory, which provides a moist habitat to prevent against desiccation. Although this tick actively feeds on white-tailed deer and birds, it will indiscriminately feed on a large number of species [23].

Northward spread of this tick species has occurred from the southern United States across many areas of the northeast. It can now be found in many of the states bordering Canada. Model predictions indicate that areas as far north as Montreal, Quebec provide a suitable climate for *A. americanum* populations [3].

### **The longhorned tick (*H. longicornus*)**

In November 2017, an invasive tick species was detected on a sheep farm in New Jersey. Native to Asia and also found in Australia and New Zealand, it is not known how the longhorned tick ended up in the United States. By the spring, the tick was detected in more than half a dozen states and may have gone undetected in the US for over 5 years [26].

The invasion potential of this tick is high due to the ability of a female to lay viable eggs without the presence of a male tick. This means a female that is transported into a new area can set up a colony quickly on her own [26].

This tick species has not been found in Canada (at least at the time of preparation of these proceedings, October 2018), but the risk is potentially high. Besides being a pest of many livestock species, it is also the vector of bovine theileriosis and babesiosis. Pathogens including

*A. phagocytophilum*, *E. chaffeensis*, and Powassan virus have been detected in field-collected ticks in Asia [26].

### Future Considerations

Major and ongoing changes in the tick population in Canada illustrate that we must remain vigilant in tick research. An ongoing effort to understand how ecological changes are influencing native and invasive tick populations will be of value in assessing current risk and predicting future risk to our companion animals and their owners.

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## VETERINARY MEDICINE BEYOND BORDERS

Katie M. Clow, DVM, PhD

Every day, we are provided with examples of the interconnectedness of human, animal and environmental health. This is evident in veterinary practice – diseases like leptospirosis, Lyme disease and rabies quickly come to mind. In developing countries, the importance of animal health in overall community health cannot be overlooked.

Veterinarians without Borders – Vétérinaires sans frontières Canada (VWB-VSF) works in some of the poorest communities across the global south and Canadian north to foster improved animal health, human health and environmental health by applying an Ecohealth approach [1].

### Ecosystem Approaches to Health (Ecohealth)

Ecosystem Approaches to Health or Ecohealth examines health with a holistic view, looking at how various factors (ecological, social, economic) impact human health. It also considers how human activity can impact the health, function and sustainability of our ecosystems [2,3].

There are six core principles or patterns that guide the Ecohealth approach: (1) complexity and systems thinking, (2) transdisciplinarity, (3) participation, (4) knowledge to action, (5) social and gender equity, and (6) ecosystem sustainability. These principles are not mutually exclusive, nor do all need to be present in order for something to be considered within the Ecohealth framework [2,3].

Complexity defines a situation in which there is a deep level of uncertainty with multiple perspectives and no right answer. Systems thinking is a tool that is needed for complex problems as it helps generate a greater understanding of the entire problem. When applying systems thinking, various dimensions (e.g., ecological, social, economic, political) are considered, as well as multiple scales (e.g., temporal, geographical) [2,3].

Transdisciplinarity refers to bringing together multiple perspectives, including scientific knowledge and community knowledge, to generate new ideas and understandings. This helps build a common way forward with a more fulsome view of the situation [2,3].

Participation is a process that involves stakeholder engagement. It is intricately related to transdisciplinarity and involves not only those who are being impacted by the problem, but those who are contributing to the situation as well as working towards a solution [2,3].

Knowledge to action means that the work being conducted generates tangible outcomes that contribute to ongoing improvement of health. It is dynamic – meaning knowledge is being used as it is generated - and multidirectional - highlighting that knowledge flows from and to all stakeholder [2,3].

Social and gender equity are vital considerations in health, since health risks and access to health care are not equal across all members of society [4]. When we acknowledge this and explore these realities, we can work towards a more equitable approach [2-4].

Ecosystem sustainability is core to much Ecohealth work. We must strive to incorporate practices that are environmentally sound in order to promote long term improvement of health.

Indeed, many developing countries are the first to experience the negative effects of climate change [2,3,5].

Examination of several case studies will further illustrate these principles and highlight the unique role that veterinary medicine plays in the improvement of animal, human and environmental health in communities around the world.

### **Goat Pass-On Project, Isingiro District, Uganda**

The Isingiro District is located in the southwestern part of Uganda and is home to 362,000 people. This is a multiethnic society in which the majority (71.9%) of people practice subsistence crop farming, with the main crop being plantain bananas. Many livestock are also produced in this area, predominately goats, cattle and sheep [1].

HIV/AIDS, malaria, tuberculosis, malnutrition and diarrheal diseases are major human health threats. The economic and social determinants of health are vast and complex and require local perspectives and understanding to generate a set of tools and approaches for improved health [1,6].

The main animal health risks include foot and mouth disease, brucellosis, tuberculosis and East Coast fever. Livestock losses are high, sometimes reaching 100% [6,7].

In partnership with local organizations, VWB-VSF has contributed to the Goat Pass-On Project. Through this initiative, women, predominately widowed grandmothers with many orphans (due to HIV/AIDS epidemic) are provided with education and resources to begin farming. Each woman receives a goat as a loan and this goat can change the social and economic status of that family. When the goat has her first offspring, this kid is provided back to the program to be given to a new family, thus continuing the program [7].

Paraveterinary ('paravet') training is a vital component of this program [8]. Community members receive animal health training and conduct animal wellness campaigns for vaccination against *Brucella melitensis* and clostridial diseases and deworming for enteric parasites. Many of these paravets have gone on to train other community members, which allows for continued community capacity building. Significant reductions in livestock mortality have been observed in these communities [7].

There is a wealth of evidence that empowering women through education and training is a key driver for poverty reduction [4]. VWB-VSF is a strong advocate for gender equity and involving women in agriculture and animal health.

### **Dairy Cow Care, Meru Region, Kenya**

In Kenya, a large proportion (~79%) of the population are farmers – either growing crops or raising livestock. A combination of devastating droughts and floods as well as environmental degradation has made it very hard for these farmers to support their families and meet their basic needs. Malnutrition, water-borne diseases and malaria pose major health threats [1].

A single dairy cow has the ability to change the prospect for a family. For example, they now have access to nutritious milk and can generate income from milk production that can cover school fees [1,9].

VWB-VSF is working with two local dairy organizations, as well as Farmers Helping Farmers (another Canadian NGO), to provide training to local farmers and animal health workers, including women, in proper dairy cow care. Small changes in management can dramatically improve the overall health and thus milk production of a cow [10].

### **Cricket Farming, Central Laos**

Laos has one of the highest rates of malnutrition among countries in Southeast Asia. Malnutrition is recognized as the most important risk factor for disease in developing countries [11].

Edible insects have excellent nutritional value, especially as a source of protein and numerous micronutrients. They are also an acceptable food source in many cultures, including Laos. Consumption of insects has dropped though, as most are collected from the wild, and wild insect populations have declined due to environmental changes and anthropogenic habitat modifications [11].

Farming of insects is a relatively new practice and can be completed with little training and infrastructure. VWB-VSF was involved with an 18-month trial program that introduced cricket farming to 20 families in two rural villages in Central Laos. Families were provided with training and supplies to go through 5 cycles of cricket production [11].

Cricket farming was successfully adapted. Participants noted that they were easy to raise, helped generate income, provided good nutrition to the family and could be used as gifts to family and friends. Several challenges were encountered though that require additional research in order to increase the viability of cricket farming. These included inconsistent availability - as crickets took 60 days to produce and only generated food for one week - and high mortality of crickets during the cold season [11].

### **Community Dog Health, Remote Northern Canada**

In Canada's remote, northern communities, there is significant need for effective, sustainable, community-based animal health services. Across the three territories, there are only six full-time veterinary clinics, with at least 50 communities of 500 or more residents that lack access to basic veterinary care [1]. The situation is similar for the remote, northern areas of many provinces.

Many of these communities are Indigenous and dogs are the primary domestic animal. Dogs play an important role in hunting, transportation, protection and companionship. However, some of them have become a danger in many of these communities. Attacks by free-roaming dogs are not an uncommon occurrence, with elderly citizens and children under the age of 10 especially vulnerable. Risk of zoonotic diseases, such as rabies and intestinal parasites, is high due to a lack of vaccination and parasite control [1,12].

VWB-VSF recognizes this substantial need and has committed to contributing to efforts to improve animal and human health in Canada's remote, northern communities. Significant work has been undertaken to develop a sustainable program based on the principles of Ecohealth for the delivery of culturally sensitive veterinary care in these communities. This is a highly collaborative initiative involving all 5 veterinary colleges in Canada, as well as other animal health organizations including the Northwest Territories Society for the Prevention of Cruelty to Animals and the Alberta Spay and Neuter Task Force [1].

## A Veterinarian's Role

Veterinary medicine provides us with a unique and highly valuable skillset that is not only vital to improving the lives of animals and humans in our own communities, but around the world. Opportunities to share this skillset exist through many outstanding organizations, including VWB-VSF. For more information, visit [www.vetswithoutborders.ca](http://www.vetswithoutborders.ca).

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# EQUINE PROGRAM

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### THE NEUROLOGICAL EXAMINATION:

The clinical history often provides important clues to the source of the problem as one hears the owner or trainer's description of the primary complaint, the duration of the problem, and its progression, as well as the effect of interventions that have been used. Most causes of neurologic disease do not cause pain, with the notable exception of trauma and some forms of cervical vertebral stenotic myelopathy (CVSM). Some painful orthopedic conditions (foot abscess or laminitis) can cause a non-weightbearing lameness that is very similar to a horse with a radial or femoral neuropathy, but the horse with the neuropathy generally will be more willing to move and attempt to use the limb, whereas the horse with the painful lameness will be more reluctant to move.

The author prefers to start at the head and proceed caudally to the tail. In general, an equine neurologic exam can be divided into 4 parts: (1) Evaluation of mental status; (2) Cranial nerve (CN) exam; (3) Evaluation of posture, spinal reflexes and muscle while horse is standing; and (4) Evaluation of gait, posture, and postural reflexes while horse is moving. At the onset I examine the horse standing quietly in its stall looking at its posture, mental awareness and whether any odd behaviors are exhibited. Next is to approach the horse to evaluate its cranial nerves, looking for abnormalities or asymmetry between sides of the horse. Next is to watch the horse walk in straight line, trot horse in straight line, walk in circles, walk with head elevated, walk backwards and then walk the horse while pulling tail in each direction. I sometimes walk the horse in a serpentine, spin it in tight circles, walk on uneven ground and walk up and down hills.

Things I pay particular attention too are stride length, stride height, regularity of foot placement and whether the horse is dragging a toe. Horses with spinal cord disease affecting the upper motor neuron and general proprioceptive tracts show a long-stride length with a floating gait. Diseases that affect the lower motor neurons or neuromuscular system tend to cause a short choppy gait that sometimes mimics an orthopedic problem. The rate, rhythm, and regularity of foot placement can be very revealing. Horses with ataxia tend to have irregularly irregular foot placement, while horses with orthopedic disease have regularly irregular foot placement.

Proprioceptive deficits suggestive of neurologic disease are most easily seen when the horse is changing directions, circling tightly, or walking up and down a hill with the head elevated. Practitioners should watch carefully for toe scuffing, foot dragging, delayed protraction, knuckling or buckling, abnormal limb crossing or interference, pivoting on the inside limb or circumducting the outside limb when circling, and stepping on the opposite foot.

If evidence of ataxia is subtle or not observed, a more extensive lameness examination is indicated, which could include trotting in circles, flexion tests, and working on the lounge or under saddle. When an abnormal gait is recognized but its origin is not clear, the next step is again often diagnostic local or regional analgesia to see if the abnormal gait will 'block out', in which case musculoskeletal disease is assumed. If the abnormal gait is not considered 'blockable,' involves multiple limbs, or there are other reasons not to perform diagnostic analgesia, a systemic analgesia trial with phenylbutazone or similar non-steroidal anti-inflammatory drug might yield useful information. Repeated neurologic and lameness examinations are important, particularly after analgesia trials. Some of the most confusing cases are those with neuromuscular disease, or those that have both neurologic and orthopedic problems. Diagnostic testing geared toward neuromuscular disease might include pre- and post-exercise muscle enzyme activity, electrodiagnostic testing (e.g. electromyography), and muscle (or nerve) biopsy.

### **Neuroanatomic localization:**

Localizing lesions is the most important step in accurate diagnosis is localizing the problem to a specific part of the nervous system. This will help guide the differential diagnoses list and the diagnostic test selection. Treatment plans can then be formulated based on the working diagnosis which is obtained. Here are some typical clinical signs seen with lesions in these neuroanatomical areas. The forebrain or prosencephalon encompasses the cerebral hemispheres and diencephalon. Common signs associated with lesions in these regions include obtundation, central blindness (loss of menace response contralateral to lesion), seizures, proprioceptive deficits (contralateral to lesion), and decreased nociception (reduced response to stimulation of nasal septum or body contralateral to lesion). Behavioral changes frequently occur and might include head-pressing, circling, maniacal behavior, or unusual aggression.

Diseases that cause forebrain signs include metabolic encephalopathies (hepatic, renal/uremic, or intestinal/hyperammonemia) and viral encephalitides (Eastern Equine Encephalomyelitis, West Nile Virus, Rabies, and (rarely) Equine Herpes Virus-1). Trauma and space-occupying masses, such as tumors, granulomas (cholesteatomas), and abscesses, can affect the prosencephalon. Bacterial meningoencephalitis is rare in immunocompetent horses but more commonly seen in horses with immune deficiencies. *S. neurona* and *N. hughesi*, the causative agents of Equine Protozoal Myeloencephalitis (EPM), can affect the prosencephalon and mimic other encephalopathies. Though most horses with EPM show spinal cord or brainstem signs.

Horses with cerebellar disease show ataxia that is often characterized as spastic and dysmetric along with head and neck tremors that worsen with intention, normal vision but loss of menace (ipsilateral to lesion). Cerebellar abiotrophy is a genetic disorder that occurs in young Arabian horses. All the viral encephalitides as well as EPM can affect the cerebellum. Trauma rarely affects just the cerebellum, but space-occupying masses might.

Horses with brainstem lesions often show changes in mental status, obtundation to stupor, due to interference with the ascending reticular activating system; general proprioceptive (spinal) ataxia due to interference with UMN/GP tracts; and multiple cranial nerve abnormalities due to lesions in the cranial nerve nuclei. The viral encephalitides and EPM can affect the brainstem. Trauma secondary to flipping over backwards with fracture of the basilar skull bones or pathologic fracture/meningitis due to temporohyoid osteoarthropathy can cause brainstem signs. Less commonly, space-occupying masses affect the brainstem.

Spinal cord lesions between C1 and C6 result in upper motor neuron paresis and general proprioceptive (spinal) ataxia in thoracic and pelvic limbs. This region is the most common spinal cord localization and most common overall neurolocalization in horses due to the prevalence of cervical vertebral stenotic myelopathy (CVSM). Pelvic limb signs are often more pronounced than thoracic limb signs. Diseases to consider include CVSM, EPM, trauma, and Equine Degenerative Myeloencephalopathy (EDM)/Neuroaxonal Dystrophy (NAD). Foals sometimes develop discospondylitis or vertebral abscesses.

Horses affected with lesions between C6 and T2 show lower motor neuron paresis and general proprioceptive (spinal) ataxia in the thoracic limbs with upper motor neuron paresis and general proprioceptive (spinal) ataxia in the pelvic limbs. Less common site of a lesion in horses are found between T3 and L3 in the spinal cord. Affected horses have normal thoracic limbs but upper motor neuron paresis and general proprioceptive (spinal) ataxia in the pelvic limbs. Horses with EHV-1 myeloencephalopathy often present with thoracolumbar spinal cord signs predominating. EPM can also cause predominantly thoracolumbar signs. Tumors such as metastatic melanoma can compress the spinal cord in this region, and trauma causing vertebral fractures or dislocations can also occur. Lesions between L4 and S2 will result in horses with normal thoracic limbs but lower motor neuron paresis and general proprioceptive ataxia in the pelvic limbs, possibly urinary and/or fecal retention/incontinence, and sometimes weak anal tone. Potential causes include EPM and trauma to the sacral region. Finally lesions in the caudal sacral regions cause normal thoracic limbs and often normal or only mild pelvic limb lower motor neuron signs. Cauda equina signs, such as urinary and fecal incontinence, weak tail tone, and decreased to absent perineal sensation, are present. Potential causes include polyneuritis equi and trauma.

Diffuse neuromuscular disease induces generalized weakness, difficulty supporting weight, base-narrow stance, muscle tremors, and tendency to become recumbent. The two most common diffuse neuromuscular diseases of horses are Equine Motor Neuron Disease (which does not have cranial nerve involvement) and Botulism (which almost always has cranial nerve involvement such as weak tongue tone and dysphagia). It is also possible to have a multifocal or diffuse neurologic disease, in which case any combination of brain, spinal cord, and neuromuscular dysfunction might be seen. EPM, should be considered strongly when multifocal disease is present.

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## EQUINE PROTOZOAL MYELOENCEPHALITIS (EPM)

EPM is a common infectious cause of ataxia in horses. This disease has two recognized causes in horses the most common cause is by the protozoan parasite *Sarcocystis neurona*, while less frequent but equally devastating can be another protozoan parasite such as *Neospora hughesi*. General proprioceptive ataxia is the most common clinical sign of disease. Signs are often asymmetric, with a mixture of upper and lower motor neuron paresis. Horses with lower motor neuron involvement, show muscle atrophy which is asymmetric and often multifocal indicating damage may be in both brainstem nuclei as well as in the spinal cord.

Antemortem diagnosis of EPM can be challenging and is often presumptive because though many horses are exposed to the parasites and make antibodies, detectable in blood and CSF, against these organisms only a small portion actually develop clinical signs. Therefore, performing serology, regardless of test chosen, will only reveal whether the horse has been exposed to *S. neurona* or *N. hughesi* and does not indicate the current disease status of the CNS. The best way to confirm a diagnosis of EPM in the live horse is to identify the presence of neurologic disease; rule out other causes for the clinical signs, such as cervical vertebral stenotic myelopathy; and identify specific antibodies in the CSF and blood.

Currently available tests are based on differences in their methodologies and which antibodies each detect. The platforms include (Western blot (WB), indirect fluorescent antibody test (IFAT), or enzyme-linked immunosorbent assay (ELISA). Several commercially available tests detect antibodies against *S. neurona*, including WB, IFAT, SAG2, 4/3 ELISA, and SAG 1, 5, 6 ELISA. All but the WB are quantitative. IFAT and ELISA are available as testing options for antibodies against *N. hughesi*.

Following recognition of neurological disease in a horse a guideline for interpretation of EPM test results should be: regardless of the magnitude of the titer a positive serum test indicates exposure to the organism but does not confirm CNS infection. A negative serum test usually indicates that the horse has not been exposed to the organism. Although rare, in some recently infected horses' clinical signs are seen prior to seroconversion (repeat the test in 10-14 days). A positive CSF test is more likely to correlate with an EPM diagnosis than a positive serum test, however, false positives occur. A negative CSF test usually means EPM is not the cause of disease, though as mentioned above some recently infected horses can show clinical signs prior to developing a measurable antibody level in CSF. In the end the best way to diagnose EPM is to submit serum and CSF for quantitative testing and calculation of a serum: CSF titer ratio (or specific antibody index), which allows detection of intrathecal antibody

production. Validation studies for the *S. neurona* SAG 2,4/3 ELISA showed that the serum:CSF titer ratio increased overall accuracy to 93-97%, as opposed to serum alone, which had an overall accuracy of 54-56%.

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## UPPER AIRWAY CONDITIONS IN THE STANDARD BRED AND THOROUGHBRED RACEHORSE

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Upper airway conditions of the racehorse are many and can either be obvious or subtle, silent or dynamic, and are often subject to a variety of interpretations. Diagnosis by resting endoscopic examination has been the gold standard for decades, however, the dynamic nature of many of these abnormalities has given rise to the tremendous popularity and value of the dynamic respiratory exam (DRE) or overground endoscopy.

### EPIGLOTTAL ABNORMALITIES

By far the most common epiglottal abnormality observed in the racehorse is entrapment by the aryepiglottal folds (AEF). This is almost always a surgical condition and uniformly these cases do extremely well following correction. There are a few key things to remember about making this diagnosis and recommending a course of action.

1. Entrapments come in all shapes and sizes and it is important for the practitioner to know the character of the tissue (i.e., fresh and elastic vs hard and fibrotic) when choosing the type of surgical approach (laser or hook).
2. Many entrapments are extremely swollen, or even infected at the time of diagnosis and preoperative medical therapy can be very beneficial to ensure a successful surgical outcome. Additionally it is critical to be able to differentiate an entrapped epiglottis from a case of epiglottitis or abscessation of the AEF.
3. The 10-14 days of postoperative care is critical to the success of surgical repair and the importance of the practitioner's oversight here cannot be overstated.



*It is critical to know the difference between and epiglottal entrapment (left) and epiglottitis (right). The case on the right is NOT surgical and a misdiagnosis could result in potential irreparable harm to the epiglottis.*

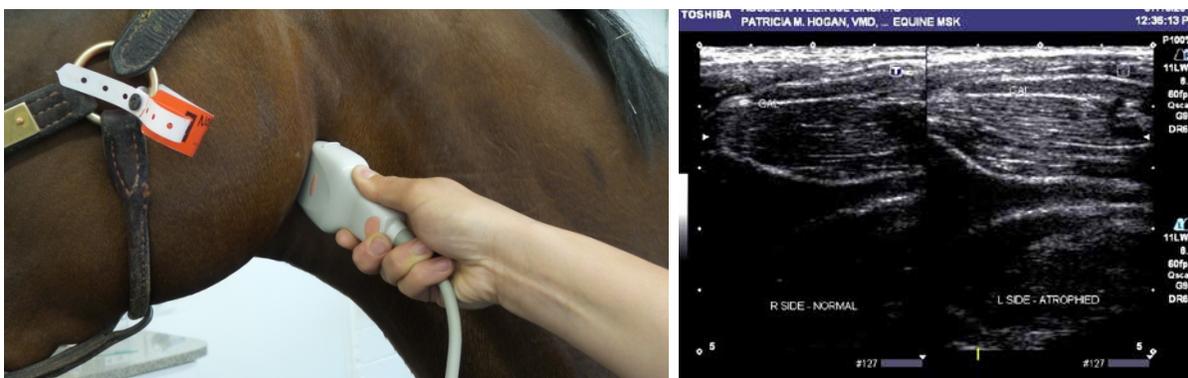
There are several other significant epiglottal abnormalities affecting the racehorse to be discussed including intermittent entrapment by the AEF, epiglottitis, epiglottal retroversion, chronic ulceration of the underside of AEF, axial deviation of the AEF, and subepiglottal cyst formation.

## DORSAL DISPLACEMENT OF THE SOFT PALATE (DDSP)

The more surgery I have done to address the complaint of intermittent dorsal displacement of the soft palate, the more I believe that this is primarily a medical disease and not a surgical one. It is my opinion that many horses displace their palate or experience other temporary upper airway obstructions when fatigued as a direct result of increased upper airway pressure due to lower inflammatory airway disease (IAD). I have found that by addressing the IAD, performance issues generally resolve and the clinical signs of upper airway obstruction dissipate.

## LEFT LARYNGEAL HEMIPLEGIA (LLHP)

Paralysis of the left arytenoid (and rarely the right) remains a relatively common airway abnormality, primarily in the Thoroughbred racehorse. Remarkably, the surgical procedure for correction of this condition, a laryngeal prosthesis or “tieback”, has remained relatively unchanged in technique since its development nearly 50 years ago. What has changed is our ability to detect the most subtle cases using either dynamic endoscopy or laryngeal ultrasound. Laryngeal ultrasound (*below*) has been a game-changer in my practice and has been a very valuable technique not only in detecting grade 2 or subclinical grade 3 paralyse, but also to differentiate and manage early cases of arytenoid chondritis.



In general, clients tend to have a very poor impression of tieback surgery. There is some substance to that impression due to the innate nature of the abnormality and the challenges associated with the surgical correction. However, there have been some small but very significant improvements in the technique and there is a definite learning curve for the surgeon as far as achieving success with this issue. Most surgeons with a concentrated upper airway caseload enjoy success rates appreciatively better than the reported 60-70%.

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## PERIPHERAL NERVES:

Evaluation of peripheral nerve injuries begins with the knowledge of the major peripheral nerves including the origin from the spinal cord. For the thoracic limb, the pectoral and subclavius muscles are innervated by the pectoral nerve which originates from C6-C7 and C7-T1 (picture of Allison Springers horse). The supraspinatus and infraspinatus muscles are innervated by the suprascapular nerve which originates from nerve roots C6-C8. The deltoid muscles are innervated by the axillary nerve C6-C8. The biceps brachii by the musculocutaneous nerve C6-C8. The triceps and extensor carpi radialis muscles innervated by the radial nerve C7-T1. The superficial digital flexor is innervated by the ulnar nerve C8-T2 and the deep flexor is innervated by the median and ulnar nerves originating from C7-T2.

The paravertebral muscles are segmentally innervated by dorsal branches of ventral spinal nerves from L6 to coccygeal 1. Innervation of the pelvic limb the long digital extensor is by the peroneal nerve originating from L6-S1. The gastrocnemius, deep digital flexor by the tibial nerve originating from S1-S2. The semimembranosus by the ischiatic nerve which originates from roots of L5 to S2. The quadriceps muscles including the vastus lateralis are innervated by the femoral nerve from L3 to L5. The biceps femoris receives innervation from the caudal gluteal, ischiatic and peroneal nerves which originate from segments L6-S2 and the middle gluteal muscle is innervated by the cranial and caudal gluteal nerves originating from L5 - S2.

Clinical signs associated with peripheral nerves are most often a result pressure on a particular nerve such as might occur by a traumatic incident, although pressure caused by an internal mass such as an abscess, hematoma, swollen lymph node, or sometimes neoplasia can also put pressure on a nerve and lead to delayed conduction or even loss of both motor and sensory functions. When a nerve is injured or transected from its cell body a series of events occur which leads to loss of myelin, this is referred to as Wallerian degeneration. As the nerve degenerates the first thing to be lost are large diameter myelinated fibers responsible for proprioception causing the horse to show abnormal foot placement or ataxia. This is followed by loss of large myelinated motor nerves leading to weakness or paralysis and with time the horse shows loss of sensory function as these are the smallest fibers and are responsible for pain. Thus, loss of deep pain is the last thing to go and signifies a very poor or grave prognosis. Following transection, a muscle will lose about one-half of its size within 2 weeks. The loss of tone that occurs soon after a nerve injury can cause a muscle to appear smaller almost immediately which is sometimes confusing when the history states the injury occurred "yesterday".

Some examples of peripheral nerve injuries that the author sees fairly often include suprascapular nerve damage with outward rotation of the thoracic limb and atrophy of the supra and infraspinatus muscles (sweeny). Horses showing overextension of the

elbow have suffered injury to the musculocutaneous nerve while horses showing a dropped elbow along with inability to extend or lock the fetlock have radial nerve injury. When a horse shows all of the signs described above it is likely the horse has damaged the entire brachial plexus.

Some examples of pelvic limb nerve injuries can include an inability to bear weight along with knuckling and buckling of one limb followed by loss of sensation to the medial thigh region followed by atrophy of the quadriceps muscles have femoral nerve damage. While a horse showing lateral slipping of the pelvic limbs likely has obturator nerve damage with loss of function of the gracilis and adductor muscles. Injury to the sciatic nerve results in loss of function of the semimembranosus and semitendinosus muscles; signs associated with this include poor limb flexion, extended stifle and hock and a flexed fetlock. Horses with peroneal nerve damage show knuckling of the fetlock along with inability to flex the hock or extend the digits.

Peripheral nerve injuries are quite debilitating and often appear very painful, making it difficult to eliminate a fracture as the cause of the signs. Horses that cannot use a limb quickly become very anxious and may be difficult to handle, thus tranquilization is often an important part of the patient management. Soon after the horse is quieted it is helpful to provide support for the weak limb. This sometimes involves use of external support such as a sling to help hold the horse up while a bandage and splint are applied to the affected leg. In some horses a splint can be applied while the horse is recumbent, however most of the time the limb can be best positioned for splint application if the horse is in a standing posture.

In the acute phase of medical management use of both steroidal and non-steroidal anti-inflammatory medications are indicated. Topical medications such as DMSO as well as topical non-steroidal medications like Surpass® can help reduce inflammation. In the earliest stages use of cold hosing and ice packs may help reduce inflammation and swelling. Following the acute phase and assuming the horse has stabilized to a degree that some use of the limb is possible it is prudent to begin a course of exercise as part of the rehabilitation program. Use of ..... therapies such as acupuncture as well as Transcutaneous Electrical Nerve Stimulation (TENS units) to stimulate either muscle or nerves to fire can be helpful.

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## EQUINE NEUROLOGY

### **Infectious Neurologic diseases: (EHM, EEE, WNV, EPM, Lyme et al)**

Equine herpesvirus-1 myeloencephalopathy: Infectious neurologic diseases which can cause fever along with ataxia or other neurologic signs. One which has been of particular interest and concern over the past ten years is equine herpesvirus-1 myeloencephalopathy (EHM). Whenever this problem is mentioned it often incites fear of serious neurological disease and even death in individual horses as well as and outbreaks with multiple affected horses. The other fear is what happens if my horse or my farm or my show ground or race track is subject to quarantine. Equine herpesvirus myeloencephalopathy is not the only cause of fever and neurologic signs.

Understanding and recognizing diseases that cause fever and neurologic signs and knowing how to properly diagnose and treat infectious neurologic diseases, including knowledge of which diseases can be prevented by vaccination is important. In addition, it is critical for veterinarians to understand when to be suspicious of EHM and how to mitigate potential outbreaks.

Two of the most common neurologic diseases, cervical vertebral stenotic myelopathy (CVSM) and equine protozoal myeloencephalitis (EPM), do not cause fevers.

Therefore, when faced with a febrile and ataxic horse, several possibilities exist. The fever could be due to an unrelated comorbidity; reflective of hyperthermia due to high ambient temperature, pain, or stress; or indicative of an inflammatory, infectious neurologic disease. If there is no evidence for the first two causes, viral and bacterial diseases causing febrile neurologic disease should be considered with particular attention as to whether EHV-1 is a possible cause.

EHV-1 is a ubiquitous alpha-herpesvirus that is readily and primarily transmitted as a respiratory pathogen, either by aerosolization or direct contact between infected horses or fomites. This virus can also cause abortion, neonatal death, and neurologic disease in individual horses or outbreaks. Typical clinical signs of the neurologic form include fever, paresis, ataxia (usually worse in pelvic limbs than thoracic limbs), urine dribbling, fecal retention, and decreased tail and anal tone. Severely affected horses become recumbent or show evidence of brain involvement.

Polymerase chain reaction (PCR) is the most rapid and sensitive test for antemortem diagnosis of EHV-1 and can distinguish neuropathogenic from non-neuropathogenic strains, both of which can cause outbreaks of neurologic disease. However, approximately 85% of outbreaks over a 30-year period were caused by the neuropathogenic strain. Either strain may cause cases or outbreaks of respiratory disease, neonatal deaths, or abortions and neurologic disease. Samples for PCR testing include whole blood (buffy coat leukocytes) or nasal swabs; submitting both

samples yields maximum sensitivity as the post-exposure temporal profiles of EHV-1 in nasal secretions and PBMCs do not completely overlap, and it is possible for one test to be positive while the other is negative.

Treatment of EHV-1 myeloencephalopathy is primarily supportive, with the goals of maintaining the horse's comfort, hydration, and nutrition while allowing it time to recover. Continuous or intermittent urinary catheterization may be required, as well as antimicrobial treatment to prevent or treat urinary tract infections. Anti-inflammatory medications such as NSAIDs or steroids are commonly used at standard dosages. Antithrombotic medications have been used (aspirin, pentoxifylline, heparin) but their benefit is yet to be fully and critically evaluated. Antiviral drugs might be beneficial for EHM treatment. Acyclovir has poor oral bioavailability in horses and subsequently low serum concentration, giving it limited therapeutic potential. Valacyclovir has improved bioavailability, estimated at 48-60% by one group. A treatment protocol of 27 mg/kg valacyclovir q 8 hr. for 2 days (loading dose) then 18 mg/kg q 12 hr. (maintenance dose) has been recommended for 1-2 weeks in the face of an outbreak.

Prompt identification of potential EHM cases and outbreak mitigation is essential. EHM is a reportable disease and state veterinarians should be notified on suspicion or confirmation of disease as required by the individual state. Outbreaks often seem to occur in the winter or early spring, possibly because during those times horses tend to congregate in more closed environments with common airspace. There is often a history of recent horse movement. To control an outbreak, several basic steps should be followed. First, all horse movement should be stopped, and the facility housing affected horses (or those exposed to affected horses) should be quarantined. If exposed horses have left, a trace-out should be performed. This quarantine generally remains in place for at least 21-28 days after all horses are normal (afebrile) although understanding the rules in your state or province is important.

Whenever a quarantine is recommended or entered it is important to have a plan in place for how to get out of the quarantine. This usually involves some form of testing and PCR is most often utilized to confirm cessation of EHV-1 shedding by quarantined horses. Temperatures should be monitored twice daily and any horses with temperatures  $\geq 101.5$  should be isolated. All horses with fevers, respiratory signs, abortions, or neurologic signs should be tested for EHV-1, and any horses with a reasonable likelihood of exposure to those horses are often tested. Affected/suspect horses should be moved into strict isolation, a dedicated facility away from other horses. Personnel should be restricted and barrier precautions (coveralls/plastic gowns, dedicated boots/plastic boot covers, and gloves) should be used. Facilities should be disinfected frequently; steam-cleaning and phenol- or iodophor-based disinfectants (as well as many others) will kill EHV-1. Vaccination in the face of an outbreak is not known to be helpful although there are still questions surrounding the use of vaccination as well as frequency and type of vaccine.

Remember not every horse with a fever and ataxia has EHM! Other infectious diseases that can cause these signs are briefly described. Another important cause of neurologic disease is West Nile virus, which is a flavivirus transmitted by mosquitoes. Infected horses can be subclinical or clinical, with clinical signs including fever, muscle fasciculations or tremors, paraparesis, tetraparesis, ataxia, recumbency, and intracranial signs such as behavior changes. Cases usually occur from mid-summer to fall. Antemortem diagnosis is best accomplished with an IgM capture ELISA on serum (or CSF). Treatment is primarily supportive; hyperimmune plasma might be beneficial.

Two other viral causes of encephalomyelitis are Western and Eastern equine encephalomyelitis, like WNV, these are mosquito-borne virus that tends to cause more prominent signs of brain disease than spinal cord disease as well as fever. These diseases can be diagnosed antemortem using an IgM capture ELISA but more commonly is diagnosed postmortem due to rapid progression of signs and high mortality rate. No specific treatment exists. EEE is most often fatal while WEE has a > 50 % successful outcomes.

Rabies is a fatal zoonotic disease, which should always be considered when faced with an acutely neurologic, unvaccinated horse. Signs are inconsistent but most commonly include paraparesis and ataxia, lameness, recumbency, pharyngeal paralysis, and colic. Fever is present in some but not all cases (about 50%). No available validated antemortem test exists for horses; definitive diagnosis requires postmortem testing.

Coronavirus is an emerging infectious disease in horses that most commonly causes gastrointestinal disease, with signs including fever, inappetence, colic, and diarrhea. Leukopenia, neutropenia, and lymphopenia are common. In occasional horses the gastrointestinal pathology leads to hyperammonemia and encephalopathic signs. Diagnosis is usually achieved via fecal PCR. Treatment is supportive and non-specific.

*Anaplasma phagocytophilum* infection, previously known as equine granulocytic ehrlichiosis, is caused by a tick-borne rickettsial organism. Signs commonly include fever, lethargy, poor appetite, limb edema, icterus, and sometimes petechial hemorrhages. Hematology might reveal mild anemia, thrombocytopenia, leukopenia, lymphopenia, hyperbilirubinemia, and intracytoplasmic inclusions in neutrophils. Neurologic signs are rare but can be dramatic if they occur, including rapid development of recumbency. Whole-blood PCR can be performed for confirmation of infection. Oxytetracycline treatment is usually successful.

Infection of the nervous system with *B. burgdorferi*, known as Lyme neuroborreliosis, is an uncommon cause of fever and ataxia. The causative spirochete is transmitted to horses via ticks and most commonly causes subclinical infection. Exposure occurs when the horse is bitten by an *Ixodes* spp. tick carrying *B. burgdorferi*. Some horses develop chronic nervous system infection. Signs most commonly include weight loss and cranial nerve or brainstem involvement in addition to ataxia. Cerebrospinal fluid cytology is variable but most often shows a neutrophilic or lymphocytic pleocytosis. Antibody testing gives inconsistent results and post-mortem diagnosis is considered

most definitive. Horses may eliminate the infection, become chronically infected but asymptomatic, or chronically infected and symptomatic. Antemortem diagnosis of LNB is best achieved by these criteria, *B. burgdorferi* exposure, presence of neurologic signs, diagnostic testing to rule out other potential diseases, documentation of abnormal CSF, and identification of the organism or immunologic evidence of infection. Commercially available tests for *B. burgdorferi* include ELISA, IFAT, WB, C6 ELISA SNAP test, and fluorescent bead-based multiplex assay (Multiplex). A positive Lyme test usually indicates past or present infection with *B. burgdorferi*, although vaccination can also cause positive results for some tests. Some veterinarians use one of the three available Lyme vaccines for dogs. Antimicrobial treatment is not reliably successful but can lead to improvement.

Bacterial meningoencephalitis (aside from neuroborreliosis) is uncommonly seen in adult horses. If bacterial meningoencephalitis is suspected, predisposing factors such as immunodeficiency (such as common variable immunodeficiency), spread of local disease (such as severe sinusitis), or recent procedures (such as cerebrospinal fluid centesis or cervical injections) should be considered. Signs include fever, lethargy, severe neck pain or unwillingness to bend, ataxia, paresis, and tremors. Diagnosis is most commonly achieved via cerebrospinal fluid cytology and culture. Antimicrobial treatment can be curative.

## Brain and Spinal Cord Trauma:

Neurologic trauma is one of the most common causes of neurologic disease in horses accounting for up to 24% of presentations in one study Feige et al Equine Vet educ 2004. Three types and/or neuroanatomic location have been described. Traumatic brain injury accounts for 6 to 11% of the horses presented for neurologic trauma, while spinal cord injury is seen in 60 to 70 % of the affected horses and about 15 to 20 % of the horses have peripheral nerve injuries. Various clinical syndromes are seen following nervous system injury but range from coma, vestibular disease or syndrome to tetraparesis, paraparesis, tetraplegia and cauda equina syndrome. Head trauma often results from collision with an immovable object, falling down, flipping over backwards or being kicked by another horse. Another cause of head trauma is fracture of the petrous temporal bone associated with temporohyoid osteoarthropathy. Injury to the brain associated with skull fractures or closed head injuries occurs both at the site of the trauma or coup type injury as well as at the contrecoup or site opposite to the place of impact. The damage to the nervous tissue results from both direct trauma as well as secondary injury caused by a cascade of molecular, cellular and biochemical changes which occur over hours to days following the original trauma. The brain is enclosed in a nonexpandable case of bone with a nearly incompressible brain parenchyma and a constant flow of arterial blood in and an equal venous outflow of blood. The intracranial pressure and cerebral blood flow are controlled by a very specialized cerebral autoregulation. When this system is disrupted following traumatic brain injury an increase in intracranial pressure can rapidly cause damage to the brain parenchyma.

Clinical signs following traumatic brain injury vary from almost indistinguishable to recumbency secondary to unconsciousness and sometimes even death. In many horse's hemorrhage from the nose or ears can be seen. In some cases, this may be accompanied by presence of CSF in the ear canals along with these signs' horses may show respiratory distress, hypo or hypertension and sometimes cardiac arrhythmias. The level of consciousness is affected by damage to the cerebral cortex and the ascending reticular activating system in the brainstem.

Vertebral and spinal cord trauma: Trauma to the vertebral column and spinal cord are similarly caused by collision with an immovable object or falling down. Although vertebral trauma can occur at any site, cervical vertebral injuries appear most common and foals are more susceptible than adults, although again spinal cord injuries can occur at any age. With severe injuries damage occurs to the bony as well as the soft tissue supporting structures of the vertebral column. The types of injuries in young horses include luxation, subluxations, and epiphyseal separations. The latter are possible since the vertebral growth plates do not fully close until around five years of age. In both young and old horse's compression injuries occur when the horse runs head-long into an immovable object.

Primary injury to the spinal cord starts with the initial mechanical damage to the cord where blood vessels are broken, axons are disrupted and neuron and glial cell

membranes damaged. Following this there is a secondary insult involving both necrosis of the tissues and apoptosis or programmed cell death. This auto destructive cascade of events begins with ischemia and progresses as a result of inflammation, production of free radicals and release of excitatory neurotransmitters. Following acute injury hemorrhage occurs in the central grey matter and the lesions progresses in a centripetal fashion over the next few minutes to hours, eventually leading to white matter edema and necrosis. The ischemia and hypo-perfusion will lead to hypoxia followed by buildup of lactic acid and release of many vasoactive substances. Similar mechanisms occur following traumatic brain or spinal cord injury.

Traumatic brain or spinal cord injuries are currently incurable and treatment is limited to minimizing secondary complications and maximizing residual function by rehabilitation. Current treatments are focused on combating the secondary pathophysiologic processes which occur following the initial trauma. In the earliest stage of either brain or spinal cord injury the focus is on decompression of the damaged tissues either by surgical methods or by cooling or hypothermia. Following the use rescue therapies, the focus turns to helping axon pathways that remain intact or partially intact to heal and to allow reactivation of impulse transmission. Beyond this rehabilitation focuses on training other tracts to perform the job of lost pathways and/or helping the damaged axon pathways to heal and be retrained to perform their required tasks. In some patient's surgical intervention may be appropriate to help the patient.

## **SELECTED LAMENESS CONDITIONS IN THE STANDARDBRED AND THOROUGHBRED RACEHORSE**

**Patricia M Hogan, VMD, Diplomate ACVS**  
**Hogan Equine LLC, Cream Ridge, NJ USA**

There are a myriad of lameness problems affecting both the Standardbred and Thoroughbred racehorse – some very similar to both breeds and some are remarkably different in clinical presentation, diagnosis, treatment, and prognosis. A few talking points to consider for this case-based presentation -

### **THE LOST ART OF THE LAMENESS EXAMINATION**

Unfortunately, a real void exists in the racing world for a proper lameness examination. There are a variety of factors affecting this situation such as owner/trainer compliance, time constraints of the attending veterinarian, and financial factors associated with pursuing a lameness diagnosis. However I feel strongly that identifying the problem and focusing on a specific solution ends up ultimately being the most cost-effective and efficient method to achieving success. It is also important to differentiate that "injecting" a horse sound is not the same as "blocking" one sound.

### **STRESS REMODELING OF BONE**

In recent years, it has become obvious that the majority of lameness problems observed in both racing breeds have an underlying root cause related to stress remodeling of bone (i.e. "bone bruising"). This natural phenomenon is the skeleton's normal response to increased exercise, however many man-made factors influence that response to the point that it often becomes pathologic and problematic. The list of maladies is long and varied - from the low-key, somewhat manageable conditions of bucked shins, 3rd carpal bone sclerosis, distal cannon subchondral bone inflammation, etc. To the more serious and potentially catastrophic condylar fractures, dorsal cortical cannon bone fractures, carpal/tarsal slab fractures, and humeral/scapular/tibial/pelvis stress fractures.

Once bone has reached a pathologic state in this regard, there are some permanent changes that will occur that will often leave a lasting effect. This can make treatment options limited and frustrating as even extended rest and removal from training, may not result in a sound horse. As we have learned from previous research (Fisher and Nunamaker) detailing the stress remodeling that occurs in the shins of young Thoroughbreds undergoing race training, there are some management practices that can be implemented in the training program of both breeds that would help to minimize this issue. Pharmaceutical intervention (i.e. bisphosphonates) is highly controversial and in my opinion, a "Pandora's Box" that should never have been opened.

### **"NO FOOT, NO HORSE"**

Many lameness issues are secondary to sore feet. This is especially true in the Standardbred racehorse, which races weekly, at very high speeds, and over "concrete" like track surfaces.

Any lameness in the forefeet changes the symmetry of the entire gait of the horse, commonly leading to many secondary and tertiary problems.

I find hoof-testers to be an unreliable and crude diagnostic tool when we are talking about the more subtle but impactful causes of foot lameness in the racehorse. In these cases we need to rely heavily on clinical findings, a very specific blocking pattern, and/or sophisticated imaging modalities.

### A WORD ABOUT IMAGING

There has been remarkable strides made in recent years in affordable and portable imaging modalities in equine veterinary medicine. Digital radiography has allowed for instant high-quality stall-side imaging with the ability to email or text images instantly to a specialist anywhere in the world for immediate consultation. In addition to nuclear scintigraphy, standing MRI and CT are now becoming more widely available and affordable, which opens the door to enhanced diagnostic capabilities for the attending veterinarian to make the best decision for the patient.



*The pathology at the bottom of a racehorse's cannon bone is a whole new world - one that you will miss out on unless you make the FLEXED AP view a standard part of fetlock imaging routine.*

## MANAGING SYNOVIAL SEPSIS IN THE RACEHORSE

Patricia M Hogan, VMD, Diplomate ACVS  
Hogan Equine LLC, Cream Ridge, NJ USA

Synovial sepsis is a rare but inevitable event associated with intrasynovial injections in any species - the primary goal for the clinician is to recognize and minimize the risks associated with this procedure.

There are many racetrack practitioners who will inject thousands of intrasynovial structures in a year's time without incident. However if and when an issue arises, it is imperative that the problem is recognized (sometimes a difficult thing to do) and addressed immediately. Communication is KEY - and that includes with the ownership factions - not just the trainer. It is very important for you to realize that synovial sepsis is an accepted risk of intrasynovial injection - you are responsible for recognizing and treating the complication, but you should not feel that you are responsible for the costs.

An infected joint or tendon sheath used to be considered a life-threatening condition - that idea has become a rarity and currently it is expected that most joint infections will be resolved in a timely manner and that the affected horse will return to its previous level of competition with no untoward effects.

### ABSOLUTES TO LIVE BY

There are 5 key points that I abide by from start to finish when treating an infected synovial structure :

1. **Take baseline radiographs.** Whether you have known this horse for years or it is a new patient, things can change in the face of bacteria and any pre-existing pathology has the potential to worsen. There is also the distinct possibility that you are not the only veterinarian (or lay-person) who has tended to this horse. Additionally there is a good chance that this joint/sheath may have been injected without any prior imaging - a huge legal vulnerability for you. Always consider the possibility of a lawsuit.
2. **Culture and cytology.** More than 50% will be positive growth. Also many *Staphylococcus* are resistant or 'super-strains' - you need to know that! Cytology gives you immediate confirmation of *Staphylococcus* (most common) or the occasional *Streptococcus*.
3. **FLUSH, FLUSH, FLUSH!** If you can therapeutically inject this joint in a barn setting, you can certainly flush it. Most horses will tolerate this very well with sedation +/- local anesthesia. No additives necessary - just dilute the numbers of bacteria and give your antibiotics a chance to work. Flush daily until the WBC numbers are reduced by 50% twice, then taper to every other day, etc.
4. **LOCAL antibiotic therapy.** I stopped using systemic antibiotics for intrasynovial sepsis over 10-years ago and have never looked back. In my opinion, regional limb perfusion and aggressive intrasynovial antibiotics are far superior. Local therapy provides high concentrations of effective drugs, allows you to use expensive or limited-volume drugs that you might never be

able to use systemically, is not susceptible to absorption issues in a compromised synovial space, etc., and rids you of the potential health risks of systemic antibiotic therapy in the horse (i.e. colitis).

5. **'Less than 5000' Rule.** Aim for synovial WBC counts to remain below 5000 cells once all flushings have been discontinued. The protein level will lag behind but I feel confident that sepsis has been resolved if WBC counts remain below 5000 cells on at least 2 samples after all supportive treatments have been removed.



*Most joints can be flushed easily "in the field" using sedation +/- a local anesthetic. A concurrent regional perfusion (left) while performing a joint lavage/sampling increases treatment efficacy and efficiency.*

## UPPER AIRWAY CONDITIONS IN THE STANDARD BRED AND THOROUGHBRED RACEHORSE

Patricia M Hogan, VMD, Diplomate ACVS  
Hogan Equine LLC, Cream Ridge, NJ USA

Upper airway conditions of the racehorse are many and can either be obvious or subtle, silent or dynamic, and are often subject to a variety of interpretations. Diagnosis by resting endoscopic examination has been the gold standard for decades, however, the dynamic nature of many of these abnormalities has given rise to the tremendous popularity and value of the dynamic respiratory exam (DRE) or overground endoscopy.

### EPIGLOTTAL ABNORMALITIES

By far the most common epiglottal abnormality observed in the racehorse is entrapment by the aryepiglottal folds (AEF). This is almost always a surgical condition and uniformly these cases do extremely well following correction. There are a few key things to remember about making this diagnosis and recommending a course of action.

1. Entrapments come in all shapes and sizes and it is important for the practitioner to know the character of the tissue (i.e., fresh and elastic vs hard and fibrotic) when choosing the type of surgical approach (laser or hook).
2. Many entrapments are extremely swollen, or even infected at the time of diagnosis and preoperative medical therapy can be very beneficial to ensure a successful surgical outcome. Additionally it is critical to be able to differentiate an entrapped epiglottis from a case of epiglottitis or abscessation of the AEF.
3. The 10-14 days of postoperative care is critical to the success of surgical repair and the importance of the practitioner's oversight here cannot be overstated.



*It is critical to know the difference between and epiglottal entrapment (left) and epiglottitis (right). The case on the right is NOT surgical and a misdiagnosis could result in potential irreparable harm to the epiglottis.*

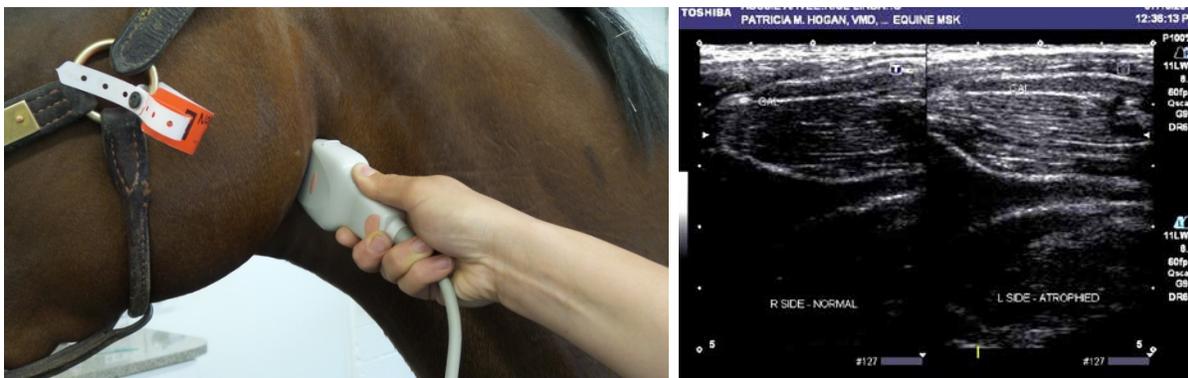
There are several other significant epiglottal abnormalities affecting the racehorse to be discussed including intermittent entrapment by the AEF, epiglottitis, epiglottal retroversion, chronic ulceration of the underside of AEF, axial deviation of the AEF, and subepiglottal cyst formation.

## DORSAL DISPLACEMENT OF THE SOFT PALATE (DDSP)

The more surgery I have done to address the complaint of intermittent dorsal displacement of the soft palate, the more I believe that this is primarily a medical disease and not a surgical one. It is my opinion that many horses displace their palate or experience other temporary upper airway obstructions when fatigued as a direct result of increased upper airway pressure due to lower inflammatory airway disease (IAD). I have found that by addressing the IAD, performance issues generally resolve and the clinical signs of upper airway obstruction dissipate.

## LEFT LARYNGEAL HEMIPLEGIA (LLHP)

Paralysis of the left arytenoid (and rarely the right) remains a relatively common airway abnormality, primarily in the Thoroughbred racehorse. Remarkably, the surgical procedure for correction of this condition, a laryngeal prosthesis or “tieback”, has remained relatively unchanged in technique since its development nearly 50 years ago. What has changed is our ability to detect the most subtle cases using either dynamic endoscopy or laryngeal ultrasound. Laryngeal ultrasound (*below*) has been a game-changer in my practice and has been a very valuable technique not only in detecting grade 2 or subclinical grade 3 paralyse, but also to differentiate and manage early cases of arytenoid chondritis.



In general, clients tend to have a very poor impression of tieback surgery. There is some substance to that impression due to the innate nature of the abnormality and the challenges associated with the surgical correction. However, there have been some small but very significant improvements in the technique and there is a definite learning curve for the surgeon as far as achieving success with this issue. Most surgeons with a concentrated upper airway caseload enjoy success rates appreciatively better than the reported 60-70%.

## WHAT CAN WE LEARN FROM LABORATORY EVALUATION OF EQUINE JOINT FLUID?

Janet Beeler-Marfisi

BA, DVM, DVSc, Diplomate ACVP (Clinical Pathology)

### SYNOVIAL FLUID COMPOSITION

Synovial fluid is an ultrafiltrate of plasma that contains electrolytes, glucose, and lactate as well as the lubricating proteins lubricin and hyaluronic acid. Capillaries within the connective tissue that surrounds the joint are fenestrated, and coupled with the hyaluronate and collagen mesh which supports synoviocytes, work to exclude large plasma proteins from synovial fluid. Synoviocytes are classified as either Type-A (macrophages) or Type-B (fibroblast-like synoviocytes) and sit directly on connective tissue as they have no basement membrane.<sup>1</sup>

Being phagocytic, Type-A cells likely remove particulate matter from joints. Type-B cells secrete hyaluronic acid and lubricin. The latter is also secreted by the menisci, chondrocytes, and tendon cells.<sup>1</sup>

The amount of joint fluid is sufficient to coat the articular surface but not to separate one surface from the other.<sup>1</sup> Lubricin, is adsorbed to the cartilage surface where it lubricates the cartilage surface, bringing the coefficient of friction at the cartilage surface to near zero under load.<sup>2</sup> It also helps prevent protein accumulation at the cartilage surface. The main role of hyaluronic acid within the joint is to hold water within synovial fluid.<sup>1</sup> Additionally, hyaluronic acid is the lubricant within the fluid phase that forms the non-compressible film that keeps cartilage surfaces separate during loading.<sup>1</sup>

In a task shared with subchondral blood vessels, synovial fluid is also responsible for delivering nutrition to the avascular articular cartilage, and is pumped in and out of this porous surface with each cycle of weight bearing and release.<sup>1</sup>

The volume, chemical and protein composition of joint fluid depends on synovial blood flow, capillary permeability, plasma protein concentration, and lymphatic removal as well as protein production within the joint.<sup>1</sup>

### LABORATORY SUBMISSION REQUIREMENTS FOR SYNOVIAL FLUID

**History:** Your notations about the characteristics of the joint fluid as it appeared at the time of sampling are very important to us, so please remember to include this in the history section on the submission form. This helps us interpret the submitted material, and more importantly, helps us make logical suggestions for next diagnostic steps in a case.

**Viscosity:** **Appropriately viscous joint fluid should hold an ~2 cm string** either as it exits the joint, e.g., through a needle inserted into the joint, or when manipulated with an applicator stick in the sample tube.<sup>3</sup> Viscosity relates to the adequacy and consistency of hyaluronic acid.<sup>3,4</sup> Viscosity will be qualitatively evaluated in the laboratory through the mucin clot test (see below).

**Fluid sample:** It is advisable to submit synovial fluid in both a serum tube (red top or “plain” tube) and in EDTA (lavender or purple top tube). Samples submitted in EDTA cannot be used for bacterial culture as it is bacteriostatic and EDTA also interferes with the mucin clot test. It

can help to use a pediatric or a 3 mL tube rather than a 10 mL tube, as the amount of EDTA in the larger tube can cause dilution of the sample. If bacterial culture will be requested, fluid from the plain tube can be used or fluid can be placed on a culturette swab and the culturette tube can be submitted. Apparently, culture results are not improved by submitting joint fluid in a blood culture bottle,<sup>3</sup> or from synovial biopsy samples.<sup>5</sup>

**Negative bacterial culture results:** Note that sometimes an infectious agent is present within the synovium, but not the fluid, may be compartmentalized in an area of the joint other than where sampled, may be present elsewhere in the body and cause immune complex deposition within the synovium, or may be present in a minute amount. All of these possibilities can result in suppurative arthritis and a negative bacterial culture result. Therefore, a negative culture result does not mean that an infectious agent is not present within the joint or elsewhere in the body.

**Bacterial isolates:** Because bacterial culture results can take 24 h or more to return it is important to know what the most likely culprits are. In one study including 95 cases of synovial sepsis, Gram-positive bacteria predominated at 75%, and 25% Gram-negative bacterial cultures.<sup>6</sup> Of Gram-positive isolates, over 50% were *Staphylococcus aureus*, including low numbers of multidrug-resistant forms.<sup>6</sup> Approximately 20% were other types of *Staphylococcus*.<sup>6</sup> Rare cases were affected by anaerobic clostridial species.<sup>6</sup>

**Direct smears:** It is always advisable to make two direct smears of joint fluid and to submit these along with the fluid. This is because the cells will degenerate with time and temperature during shipment, which can alter the cytopathologist's interpretation drastically. Therefore, we like to compare the field-made smear with our laboratory-made smear to determine what changes have taken place. To make the direct smear, place a small amount (~5 mm diameter drop) of joint fluid on a slide and make a push preparation (like a blood film). Pull-apart films can be made, but these don't tend to have as uniform a spread. Once made, **flap the slides very vigorously so they dry as rapidly as possible** to prevent drying artifact. Store these slides at room temperature in slide protectors, and when shipping, keep them away from freezer packs and any formalin-fixed samples!

Not that it occurs commonly with horse joint fluid, but if the volume of joint fluid is insufficient to send in sample tubes, **a meaningful cytologic evaluation can still be performed using a well-made push preparation.**

Note that normal joint fluid doesn't clot, but it is thixotropic, meaning it will become gelatinous on standing and return to a fluid state with agitation. If this occurs and you've not yet made smear preparations, gentle agitation will make the fluid less viscous allowing you to make good quality preparations.<sup>3,4</sup>

**At the laboratory:** Once the sample arrives at the laboratory, the technicians will note the physical characteristics of the sample – volume, colour, and clarity and will make smear preparations. They will also measure the protein concentration and nucleated cell count. It is also possible, and potentially useful to measure both serum amyloid A (SAA) in the joint fluid and in serum.<sup>7,8</sup> After this, the clinical pathologist will perform a differential cell count and the cytologic evaluation. **Normal equine joint fluid is pale yellow and clear, with no particulate material, and SAA is within the same reference interval as for serum SAA (0-20 mg/L).**

**Mucin clot test:** One to two drops of joint fluid from the plain tube are added to 8 drops of 2% acetic acid.<sup>3</sup> If the joint has good quality hyaluronic acid, a thick clot will form. If the hyaluronic

acid has been broken down or there has been decreased production in a diseased joint, then the clot is less well formed. **The clot will be described as good, fair, or poor.**

**Cell count:** Is usually performed using an automated cell counter but a hemocytometer may also be used. Hyaluronidase may be added to eliminate cell clumps. **Normal equine synovial fluid has a nucleated cell count < 3 x10<sup>9</sup>/L.**<sup>9</sup>

**Protein concentration:** Normal equine synovial fluid has a protein concentration < 25 g/L.

**Serum amyloid A (SAA):** Can be measured in synovial fluid, and in one study, were increased in horses with septic arthritis, but not in synovitis.<sup>10</sup> Serum SAA tends to increase more rapidly than joint fluid SAA in these cases. Repeated intraarticular administration of amikacin<sup>7</sup> or joint lavage<sup>8</sup> did not cause an increase in either synovial fluid or serum SAA concentrations in preliminary investigations.

**Cytologic evaluation:** The major goal is to determine whether the process affecting the joint is infectious or degenerative (i.e., osteoarthritis), and less commonly in horses, neoplastic. **In healthy joint fluid, mononuclear cells predominate and neutrophils account for <10% of cells.**<sup>4</sup>

**Microscopic analysis:** First, we note the density of the background (**normal joint fluid is very dense and may have protein crescents**). Then cellularity is estimated in an area of the smear where the background is thin and cells are well spread out (not at the origin where cellularity can appear high regardless of cell count). We look for windrowing of cells (cells arranged in straight lines following the direction of preparation) as this is an indicator of fluid viscosity (i.e., quality of hyaluronic acid content) but note that windrowing may be inapparent in low cellularity samples. We perform the differential cell count **only in thin areas where cells are well spread out** (otherwise all cells look like lymphocytes due rounding up caused by the highly proteinaceous background). If present, we assess neutrophils for degenerative changes and bacterial content, as well as the content of macrophages. If hemorrhage is present, we try to determine whether it was sampling-associated or a true hemarthrosis (see below). The final thing we look at is the background as it should have fine pink protein precipitates throughout (another indication of hyaluronic acid content, and a strong indicator that synovial fluid was sampled – this may have more relevance in samples submitted from dogs and cats).

**Infectious agents** can arrive in the joint by direct injury or hematogenous spread. Also, infection in one part of the body can cause immune-complex deposition in the synovium resulting in a neutrophil presence within the fluid (suppurative arthritis) in the absence of the infectious agent. Compartmentalization can occur in most equine joints, whereby an infectious agent can be confined to one part of the joint space, or indeed the synovium, but neutrophils will still be present in all parts of the joint.

Agents include bacteria or fungi and may not be seen by light microscopy. In these cases, our major clue to the presence of an infectious agent is a very high nucleated cell count composed predominantly of neutrophils showing various degrees of degenerative change. When tracked over time, and if treatment has been effective, the number of neutrophils should decrease and assume a normal phenotype.

In some cases of an infected joint, the nucleated cell count will be < 3 x10<sup>9</sup>/L, but will still be considered inflamed because the neutrophil percentage will be higher than 10%. This can happen if abundant fluid is present in the joint and dilutes the cells, but sometimes happens for

an unidentified reason.<sup>4</sup> This is why it is important not to rely solely on the cell count but to always look at a smear of the joint fluid to determine how many are neutrophils.

**Immune-mediated polyarthritis:** There are rare reports in the literature of steroid-responsive arthritis in horses<sup>11,12</sup> and of *Borrelia burgdorferi* causing polysynovitis.<sup>13,14</sup> I have seen cases of an unidentified causal agent being associated with steroid-responsive non-erosive polyarthritis in New Zealand. As the horses improved when moved to another farm, the cause was suspected to be an allergen present in one location but not the other.

**Lymphocytic synovitis:** In rare cases, joint fluid will have an increased nucleated cell count and a predominance of lymphocytes. This has two causes. The first is that the sample has been looked at in house and rounded up cells have been misidentified as lymphocytes. The second cause is synovitis with synovial infiltration by lymphocytes. This pattern has been seen in other species with mycoplasmal arthritis or rheumatoid arthritis, and in one case of *Borrelia burgdorferi* in the synovium of a pony.<sup>4</sup>

**Degenerative joint disease and traumatic injury:** Cases of degenerative joint disease (DJD) and traumatic injury can look very similar cytologically, and may even appear normal.<sup>4</sup> This point underscores the importance of obtaining radiographs in the investigation of suspected joint disease. In some cases, fragments of cartilage and/or osteoclasts may be seen. Osteoclasts imply that subchondral bone has been exposed due to erosion of articular cartilage and are a clear indicator that osteoarthritis is present. It is also important to note that early cases of joint trauma can look like joint infection,<sup>4</sup> so these cases will require multiple submissions over time as well as bacterial culture to help differentiate between the two possibilities.

**Hemarthrosis versus iatrogenic hemorrhage:** True hemarthrosis will often result in xanthochromic (orange-yellow tinged joint fluid) and in some instances may be uniformly red. Cytologically, we anticipate erythrophagy and hemosiderin within macrophages, and sometimes hematoidin crystals. This is another reason for preparing smears in house, as erythrophagy can occur during shipment (but not formation of hemosiderin). With iatrogenic hemorrhage we do not expect to see erythrophagy or hemosiderin and sometimes we may see platelet clumps, which are not expected with hemarthrosis.<sup>3,4</sup>

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# **PRACTICE MANAGEMENT PROGRAM**

**What is Your Communication Score?****Jayne Takahashi DVM MBA****OVMA January 2019**

## Asking open-ended questions

- statements or questions that requires more than a “yes” or “no” answer
- encourages clients to tell their story without your influence to shape the content
- funnel: start with open-ended and move to close-ended questions
- clients feel HUA’ed (Heard, Understood and Acknowledged)
- obtain an understanding of what is important to your client
- people like to “empty” before listening to new information

## Starting a conversation

- introduce yourself
- open ended question: people like to talk about themselves
- show an interest in the pet owner as well as the pet
- ask how things have been since last visit or with the medical condition

## Organizing your discussion

- setting an agenda
- setting out a ‘road map’
- signposting

## Giving clear instructions

- start from the client’s perspective and understanding
- use as many visuals as possible
- be attentive to your terminology
- presenting benefits from a client’s perspective

## Checking for understanding

- chunk and check
- open ended question versus closed ended questions
- watch non-verbal cues
- ask questions if you are unclear on any points

## Active listening

- reflect back words spoken as well as the context and/or emotion
- active process requires focus & patience – do not interrupt
- put aside your own thoughts and feelings to listen fully to other’s perspective
- “listen” to non-verbal cues

## Using empathic statements

- the most powerful communication skill to build trust
- ability to recognize and identify with the feelings that are being experienced by another person and communicating your understanding to the other person in a supportive manner
- add specific information to your statement to clearly indicate you can imagine the situation, feeling or perspective of the other person
- ensure your verbal and non-verbal cues are consistent

#### Responding to non-verbal cues

- non-verbal cues include tone of voice, volume, pauses, pace of speech, inflections, our position relative to the other person, and the setting in which the interaction is taking place
- non-verbal cues are involuntary and tend to reveal what we truly feel and believe
- watch for contradictions between your client's verbal and non-verbal messages
- watch for contradictions between your own verbal and non-verbal messages
- respond to what you see

#### Validating thoughts of others

- 'HUA' principle
- active listening, empathic statements, open-ended questions
- start from your client's perspective

#### Dealing with conflict

- be curious when differences arise
- many sources of differences
- power of the pause to avoid quick judgements/comments
- ask factual questions
- find common goal

#### Saying no

- you are saying no to the particular request – you are not rejecting the person
- empathy for other perspective
- link your rationale to the common objective

#### Clearly presenting your recommendation

- be clear on your message and intention
- express sincere empathy for the other perspective
- understand the client's definition of successful treatment
- clearly identify the objective of your recommendation and how it will address the specific concerns of the pet owner and the pet

#### Encouraging others to speak

- be mindful of the balance between the amount of time you are speaking and the time that the other person is speaking
- make it safe for pet owners to ask questions
- open ended inquiry
- use positive words

#### Using summaries in your discussion

- internal summaries
- final summary

#### Responding to the reaction you have caused

- watch non-verbal cues
- pause and check in with the person
- do you need to change your approach?

## “You Are The Client”

Jayne Takahashi DVM, MBA  
Communication Leads

Are we treating diseases or caring for pets and their people?

During this seminar, we will work through a hospital visit with from a client’s perspective with a review of a number of skills and techniques to enhance client communication. Concepts that will be reviewed in detail include but are not limited to the following:

### **Setting an agenda, Signposting, Summaries**

Clients are more likely to view the level of expertise and care for their pet as exceptional if you deliver your information in manageable amounts that are easy to follow. Providing this structure enables you to also organize your own thoughts and will create a more balanced discussion that will encourage your client to stay attentive and informed.

Before launching into your discussion, set an agenda to prepare your client for the information that will be covered. By letting your client know the purpose of your discussion and what it will cover, they can mentally prepare their own questions, relevant information or concerns. Invite the owner to provide additional information as they think of it. The use of open ended questions to probe for owner concerns will contribute to efficient information gathering and identification of key concerns at the beginning of your conversation. In this way, you can tailor your discussion to the information that is most relevant to your client.

As with any road map, you rely on signage along the way to check on your progress along your journey or to signal changes in direction. There are two communication skills that serve a similar purpose during your conversations with pet owners: signposting and summaries.

Signposting is a skill that alerts the client that you are moving the discussion to a new topic or simply draws attention to what you are about to say: “Now I am going to talk about the treatment for this condition.” Signposting can also be used to addressing a specific client concern “You mentioned that you are nervous about causing Benji pain during the ear cleaning. Let’s talk about that.” Signposting can also introduce the second communication skill of summarizing: “I’d like to review the follow up steps that we have just discussed.”

To check on the progress of the understanding between yourself and your client, it is useful to summarize or review what the client has told you or check on your client’s understanding of your information. This is referred to as an internal summary and is helpful in organizing sections of your discussion when several topics have been introduced. Internal summaries

can be inserted throughout your discussion to check on mutual understandings of a chunk of information that has just been presented.

A final summary at the conclusion of your interview captures the key points of the entire interaction. This allows the client and yourself to be clear on what has taken place and sets expectations for what is to happen next

### **Chunk and check**

How often do you check for understanding after you have shared a piece of information? Probably less often than you think. There is a tendency to dump your entire presentation and to check for understanding once at the end of your delivery. Your clients appreciate small breaks in your conversation that will allow them to process what you have presented and to address concerns about specific pieces of information.

Make it a habit to incorporate the “chunk and check” technique into your explanations. Deliver a chunk of information then pause and ask an open ended question to check for understanding such as “What questions do you have about this?” or “How are you feeling about what I have presented so far?”

### **Empathic statements**

Research indicates that there is significant room for improvement when it comes to the effective use of empathy in client interactions in veterinary practice. Shaw, Adams, Bonnet, et al<sup>1</sup> found that partnering with clients through the use of empathic statements were observed in only 7% of 300 client-patient-pet interactions. Similar to the medical appointments of physicians, I would expect that there are ample opportunities for expressing empathy during a typical consultation. Bylund and Makoul<sup>2</sup> found that 60% (100/168) of clinical encounters had a least one opportunity for physicians to express empathy.

It is important to differentiate between sympathy and empathy. Sympathy is feeling pity for or feeling sorry for the client from outside the patient’s position. Empathy, on the other hand, is viewing the situation from the other person’s position and being able to share, to some extent, what the other person is feeling.

Empathy is a two step process. The first step is to actively attempt to understand the client’s world from their point of view. In other words, put yourself in your client’s shoes and view the pet’s medical condition from their perspective without judgement or trying to make their opinion consistent with your own viewpoint. The second step is to communicate your understanding to your client in a supportive manner. A helpful way to remember this two-step process has been suggested by the Institute for Healthcare Communication. Think of empathy in terms of the inhalation and exhalation phases of the breathing cycle. Using open-ended inquiry and reflective listening as described in my previous articles, we ‘inhale’

the client's story in order to obtain the information that will help with our understanding of the client's world. Then we 'exhale' by communicating our understanding to the client so that they feel heard, understood and accepted.

Showing empathy does not necessarily mean that you are in agreement with the client's position. It simply means that you recognize every person will have an opinion, you are willing to listen to this viewpoint and you have an appreciation for your client's point of reference. You can legitimize a person's beliefs without agreeing with them.

### **Personalizing your communication approach**

Without personalizing your approach to meet the different social styles of others, you may come across as uncaring, lacking in compassion, disinterested and insincere. Behaviour or social style refers to the a person's preference for type of information, information processing and manner of decision-making.

It is important to view our interactions from our client's perspective so that we can tailor our discussions to match the preferred behaviour/social style of our client. The first parameter of behavioural style is the FOCUS of the information that the person prefers. Is there a preference for hard facts and data or for more personal discussions and the experiences that other people share with them?

The second parameter of behavior styles that can influence your interaction – the individual's preferred PACE. Pace refers to the time that a person prefers to take when processing information before making a decision. Some individuals are comfortable making intuitive or quick decisions, while others prefer to review all information and deliberate for a longer period of time. Neither preference is better or worse than the other however, having a sense of a person's preference allows you to recognize your client's comfort zone and to manage your conversations accordingly.

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## **KNOW WHO IS ON YOUR TEAM YOU ARE THE EMPLOYEE!**

Jayne Takahashi DVM MBA

Communication Leads

OVMA 2019

1. Know “WHO” is on your team
  - a. People have to come first
  - b. Recognize that people think and communicate differently
  - c. Be aware of each person’s social style preference and adapt your approach
    - i. Task focused or people focused
    - ii. Deliberate decision makers or quick decision maker
  - d. Be aware of different learning styles: visual, auditory, tactile
  - e. Be on the floor, regular check-ins or “walking meetings” in addition to annual review conversation
  
2. Create a safe environment that encourages open, supportive communication
  - a. Create awareness and acceptance of diversity within the team
  - b. When differences arise, encourage people to be curious rather than critical
  - c. Be transparent - don’t be afraid to talk about the challenges facing the business
  - d. Create opportunities for the team to express their thoughts
  - e. Make it safe to ask for help
  - f. Admit mistakes and limitations
  
3. Define clear roles, responsibilities and expectations
  - a. Job descriptions for every position
  - b. Provide and seek regular feedback
  - c. Assign tasks directly and clearly
  - d. Set deadlines
  - e. Establish cultural values = guiding principles that clearly define acceptable behavior and attitude
  
4. Learn how to provide and seek supportive feedback
  - a. Genuine, specific acknowledgement and corrective feedback
  - b. Use cultural values as the basis for all feedback
  - c. Train your team in the language of sharing – model behaviour
  - d. Focus on problems/issues vs personalities
  - e. Coach on how to graciously receive feedback
  
5. Avoid surprises!
  - a. Keep your team informed - gaps in information are often filled with misinformation
  - b. Remove uncertainty
  - c. Explain your “why” for all decisions and projects
  - d. Tell your team why decisions are important
  - e. Provide regular updates on projects
  - f. Staff meetings

6. Don't avoid "icky" conversations
  - a. Plan and structure your conversation and base it on fact
    - i. Refer to breaches of cultural values
    - ii. Use assessments of attitude and performance to guide your discussion (Topgrading by Bradford Smart)
  - b. Use "intention and impact" sentence structure: "Although it was not your intention to shake the confidence of a team member, your action did have this impact." The individual must own the impact of their actions.
7. Have more face time with your team
  - a. Email is a rational tool - great for sharing information
  - b. Team dynamics have a relational, emotional basis so face to face time is valuable and most effective
  - c. To obtain the emotional response or opinion of others – phone or speak directly to person
8. Involve team members
  - a. People with the most information can act on it so seek the thoughts of team members
  - b. Value their knowledge and skills
  - c. Taking away ownership of a role suggests that you don't have confidence or don't value the work
  - d. Encourage sharing, input and dialogue
  - e. Make your goals, objectives, progress public
9. Practice asking more questions than telling
  - a. Be curious – obtain all information and perspectives BEFORE making decision or judgment
  - b. "Learning questions" - to better understand an individual's perspective or work environment
  - c. "Coaching questions" - to encourage the person to solve their own issue or guide the thought process to find solutions
10. Lead by example
  - a. Model the behaviour you want the team to adopt
  - b. Own your mistakes – humility goes a long way
  - c. Know your own limits
  - d. Act according to your cultural values – be consistent
  - e. Consider all information before taking action; gather and confirm info before making decision
  - f. Establish vulnerability-based trust

### Additional Reading

The Five Dysfunctions of a Team, Patrick Lencioni; 2002

Topgrading, 3<sup>rd</sup> edition, Bradford D. Smart; 2012

Good Leaders Ask Great Questions: your foundation for successful leadership, John C. Maxwell; 2014



## Communication Skills Inventory

This is not a test. It is a tool designed to help you determine what your areas of strength might be as well as areas that require improvement. Feel good about the things you do well, and focus on the skills you don't do as well.

**Directions:** Read each item then circle the number to the right of each item that best describes how well you think you use a specific skill. After you have rated all the items, choose three that you will commit to improving.

	Ineffective /Unsure			Effective/Confident	
1. Starting a conversation	1	2	3	4	5
2. Asking open-ended questions	1	2	3	4	5
3. Organizing your discussion	1	2	3	4	5
4. Actively listening	1	2	3	4	5
5. Checking for understanding	1	2	3	4	5
6. Using empathic statements	1	2	3	4	5
7. Responding to non-verbal cues	1	2	3	4	5
8. Validating thoughts of others	1	2	3	4	5
9. Giving clear instructions	1	2	3	4	5
10. Encouraging others to speak	1	2	3	4	5
11. Using summaries in your discussions	1	2	3	4	5
12. Clearly presenting your recommendations	1	2	3	4	5
13. Responding to the reaction you have caused	1	2	3	4	5
14. Dealing with conflict	1	2	3	4	5
15. Saying "no"	1	2	3	4	5



## YOUR ACTION PLAN

### Which skill requires improvement?

Skill: \_\_\_\_\_

What might an improved form of this skill look or sound like?

What will be your trigger in a conversation to modify this skill?

### Which skill requires improvement?

Skill: \_\_\_\_\_

What might an improved form of this skill look or sound like?

What will be your trigger in a conversation to modify this skill?

### Which skill requires improvement?

Skill: \_\_\_\_\_

What might an improved form of this skill look or sound like?

What will be your trigger in a conversation to modify this skill?

## TOP DIGITAL MARKETING OPPORTUNITIES

Eric D. Garcia, IT & Digital Marketing Consultant  
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### THE KEYS TO MARKETING SUCCESS

Making sure your veterinary practice is running smoothly day-to-day is your priority, as it should be. But what about expansion, marketing, return on investment and all of the elements needed to truly ensure that your veterinary practice isn't just open for business, but is also **thriving like never before**?

A surprising amount of veterinary practices are operating with old software/infrastructure in place and are even avoiding the huge array of tools that can help them to truly maximize their marketing efforts across the board. On the other hand, some veterinary practices are achieving unprecedented growth and success by utilizing both new marketing techniques and software, while continuing to dedicate their efforts to delivering remarkable service, day in and day out.

**The difference is in the effort put forth and the dedication it takes up to keep up with evolving tools and trends when it comes to social media, marketing and digital infrastructure.**

While some of the advances and changes can be daunting at first, all it takes is a slight adjustment in thinking to realize that these new tools, whether we're talking about *Survey Responses* or *Social Media*, can help us to get our veterinary practices running in top-gear with a full roster of very happy clients.

Fortunately, for veterinary practices that may have yet to make the transition toward a new world of marketing, there are tried and true techniques that can enhance marketing efforts for all types of practices; whether emergency care, specialty, general practice or other.

When things are moving quickly within your veterinary practice, it's especially important to make sure that there are techniques already in place to ensure that your clients are satisfied, feeling appreciated and consistently leaving your veterinary practice in full confidence that both their time and money have been well spent. You might be surprised at how many veterinary practices handle a full client load, but forget to capitalize on strategies that could be enhancing their business from the inside out. This might mean that they are missing opportunities to gain new referrals and gain crucial feedback on what may or may not be working when it comes to their veterinary practice. This could also mean that they are being out-paced by their local competitors, without even knowing how to keep up...

**Here are a few proven techniques that can help you to successfully implement a marketing strategy that's built for the twenty-first century and beyond.** Using these

techniques, while making adjustments that allow you to customize the strategies to benefit your specific veterinary practice, will allow you to retain your current clients, expand your outreach in order to gain new ones, spend your marketing dollars far more effectively and enhance the way that your veterinary practice operates each day. By following and implementing these simple rules, you can set your veterinary practice up for total success, with the tools needed to keep improving each day:

### **MEASURE CLIENT SATISFACTION**

- 1.) The importance of measuring client satisfaction cannot be overemphasized. **Every client that leaves your practice without giving feedback on services and their overall experience is a lost opportunity to improve and gain invaluable information from the most important person to your veterinary practice: your client.**

In order to measure client satisfaction successfully, you may want to focus most intently on clients that have recently visited your practice, preferably within the last one or two days. Their visit with you is still in recent memory, and any feedback and/or critique can easily be garnered at this stage. You can complete this step by sending out surveys via email, or even handing out a final form once the client is making payment and scheduling a future appointment.

Techniques for the client survey can vary, but should effectively monitor satisfaction and ensure that you've provided good service that will render future visits and references. You may try asking questions with a "One to Ten" level of response to most effectively gauge the services that can be improved by your veterinary practice. Use this strategy to measure the promptness of the visit, friendliness of the staff, knowledge of the primary veterinarian, etc. You can design surveys to be anonymous or to even enter the client into a contest as incentive for completion. For example, "Tell us what you think and you can win a \$100.00 gift card!" Different clients have different ways of rendering their experience in total candor, so you may want to experiment with different incentives and techniques here.

You can also measure which techniques of giving a survey yields the most results, I.E. sending out an online survey the same day or the next day after a client appointment and/or giving out a physical survey which can elicit an on-the-spot response.

The technique you choose to use is of course up to your veterinary practice, but should allow for honest feedback to be gauged, analyzed and recorded by your veterinary practice, resulting in room to improve service and consequently ratings, for future appointments. Even the most successful veterinary practices have room to improve and measuring client satisfaction is one of the best possible ways to do this.

## **TRACK YOUR RETURN ON INVESTMENT**

- 2.) It can be tempting to start plugging money into marketing techniques that are polished, newly released and seem exciting. Why not spend \$100.00 to promote a cute Facebook post or \$50.00 on a new Facebook advertisement? Well, the truth of the matter is that **you should not spend money on marketing without putting the necessary tools in place to track your return.**

**ROI** is a term that you should learn inside and out. Simply put, it means **Return On Investment**. Unless you know that a specific advertisement is bringing new patients directly through your front door, there is no way to ensure that you're effectively advertising. Ineffective advertisements can actually do more harm than good, causing your veterinary practice to spend money and direct attention to a specific medium, without actually garnering results. Practices that do not implement a proper infrastructure to measure their return can blindly spend money, without ever achieving the results that they desire. This can actually be trickier than you think. For example, if you do spend \$100.00 on Facebook and get plenty of replies and shares, this may seem like a successful campaign! The post may even lead more people to visit your website or to follow your Facebook account. Still, if the visitor is not contacting your veterinary practice directly to schedule an appointment, you may be spending more money on web-traffic and your online promotion, than you are actually securing a new client! There are however, ways to distill your marketing efforts and ensure that dollars spent, result in dollars earned:

One great place to start is with **CallRail.com**; a tool that records incoming calls and determines where the client came from before calling you directly. For example, if a client finds you on Facebook (a very common example) and proceeds to your website before calling you, CallRail will allow you to gain valuable insight on the process, noting which lead resulted in the call (in this case, Facebook) and other important data about their process. You can then distinguish if the call came from a targeted Google search, Google AdWords, Facebook, Twitter, etc.

One of the largest advantages of our digital age, is the ability to leverage new tools and analytics to derive more data than ever. While the process of caring for your clients and delivering impeccable service may stay more or less the same, the process of tracking leads, traffic and growth has changed more than ever before. By making sure that your veterinary practice is adjusting to these changes and implementing the right software, you'll be poised to compliment your great service with great tools and marketing as well. You'd be amazed at what can be achieved when great veterinarians and staff deliver outstanding care, while implementing the right tools to measure their success.

### **APPRECIATE YOUR LOYAL CLIENTS**

- 3.) While showing appreciation to your local clients may seem like it goes without saying, it's actually something that a surprising amount of veterinary practices forget to do on a daily basis. When you've got a packed calendar, it can be easier to focus on the next client coming in, than it is to thank the one that's walking out the door after spending money on your services. Instead, show your appreciation by saying thank you, leaving a note, calling the next day to follow-up, or building in a client loyalty program to your practice. One way to do this is to **create a referral program**, which is an all-in-one way to reward existing clients, attract new ones and spread a feeling of generosity to your existing clientele. This can be small but significant, allowing the person who refers the practice to receive \$50.00 (or another appropriate amount) toward a future service or an additional purchase.

Many times, a client has a built-in network of friends and family that they could easily recommend to your veterinary practice, but this additional step is often overlooked. Simply by appreciating your loyal clients and adding in this extra layer of incentive, you can extend your outreach to a new level and bring in new clients who already may be right around the corner. If your client has had a positive experience, the best thing you can do is parlay this positivity outward. By showing your appreciation for the client and offering a small monetary incentive, you can do just that!

Companies like Uber do this same thing, utilizing referral codes directly through their app. This occurs to the tune of tremendous success, as those who are already utilizing the app can actually offset the cost of the service by recommending it to another. For a rider who is in transit, this may just be the most effective use of their time. Similarly, if you offer these incentive based strategies while your client is in the waiting room or while they are making a final payment, you may just find that they are eager to use the time to offset existing costs. Little things like gift cards, discounted purchases or free pet treats can make the difference between a new referral and a shrug.

### **CUSTOMIZE FOR YOUR PRACTICE**

These strategies allow room for you to make changes and implement the techniques that will work best for you and your veterinary practice. This can also read: there is no single way to go about any one of these specific marketing techniques and the strategies will require adjustments and fine-tuning to fully optimize them toward your individual practice. A large part of successful implementation will rely on your understanding of your individual veterinary practice and your individual clients, including how to customize your client survey/satisfaction techniques, marketing strategies and reward/incentive programs directly toward your clients. You may find that certain strategies work with incredible consistency, while others need to be scrapped or fine-

tuned along the way. The key is to take the larger themes, and work within them to enhance what's already working and fill in the voids where you may be lacking a strategy at all.

While technology as a whole continues to accelerate, it's more important than ever to tune into the increasing needs, wants and expectations of clients and consumers alike. From text-message updates to newsletters and online pharmacy prescription refills, we rely on the digital world more than ever to get things done every day. Your clients expect the same, wanting to experience an exceptional veterinary experience while ensuring the health of their pet is totally optimized. Sure, expectations are high. But so are the opportunities to deliver and amaze.

With all of the tools at our disposal and new strategies that can be used to enhance marketing and customer experience, the only thing left to do is implement accordingly and reap the rewards.

## SOCIAL MEDIA UPDATES PART I AND PART II

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Most veterinary practices around the globe utilize social media to engage pet owners. However, most veterinary practices are making major mistakes when communicating. Here are some of the top mistakes and how you can fix them.

### NOT TELLING 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.

### NOT GETTING 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:

- **Dermatology:** Before and After Skin Cases
- **Dental:** Before and After Dental Care (Photo)
- **Surgical Case Examples**
- **Laser Therapy Cases** (Pets can often improve a limp in a matter of weeks after laser therapy)

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.

### COPYRIGHT AND STOCK IMAGES

The unique beauty in your veterinary practice can be best shown with the authentic moments that are true to your specific practice. While displaying stock photos on your website may have helped you to launch your website initially, the influence of stock photos is something you should work to decrease over time. Personalized images that are taken within your practice that show compassionate moments (say for example, a technician restraining a cat using a cat-friendly technique or a receptionist with a warm smile in your front lobby) helps to shed light on what your practice is really like on a day-to-day basis!

Stock images might work in a pinch, but they can also give off a cold impression if they seem too generic and especially if a pet owner has seen them previously elsewhere.

Still, I wouldn’t recommend deleting all of your current images right away. You can gradually transition toward more personalized images over time *or* you can even decide to hire a photographer to help you get new images of the whole practice all at once. The main takeaway here is that every chance you get to distinguish your veterinary practice and personalize it, is a chance you should take. Your practice has its own recommendations, staff and nuance and you should be proud to tell your story and share those unique characteristics!

### INSTAGRAM

Instagram is a platform that may already extensively showcase your veterinary practice... *even if you’ve never set up an account.*

In fact, there is a very good chance that your practice is already on Instagram (also known as *Insta* for short) because of geolocation tagging that exists as a major function of the platform. This *location* feature within Instagram, allows people to share and post their location, wherever they are. Check it out for yourself, visit [Instagram.com](https://www.instagram.com) or download the Instagram app. To easily look up your location page, simply type in the name of your practice. You'll see a listing come up, like this one:



This listing at the top with the little pin and your address is the location that Instagram has already created for you.

Very few practices know that this location listing exists. It's like both Google and Yelp, where the listing was created *for you* by using a database of businesses. The biggest differentiating factor is that, with Instagram location tagging, you can't leave reviews and you can't claim your listing (at this time).

People are social by nature, so when pet owners are sitting in the waiting room, right after the veterinarian leaves the exam room, they might just take out their phone and start posting!

*In fact, they probably are.*

This is how the conversations about your practice on Instagram starts in the first place. After all, *who can resist a selfie with their beloved pet?*

**What this ultimately means for you is that if you haven't started using Instagram to interact and engage with pet owners who are already posting about your veterinary practice, you're missing a major opportunity!**

Your veterinary practice can immediately tap into its existing user base by liking and commenting on photos, and by posting your own. This can serve as the perfect opportunity to offer pet care tips, send a quick thank you, offer condolences for a pet whose final moments are posted to Instagram by the pet owner (it happens often) or anything else that truly lets the client know that they are appreciated. If you haven't set up your account yet, it's certainly not too late. Currently, there are close to 6 million posts that use the hashtag #dogsofinsta and almost **55 million** using the hashtag #catsofinsta (yes, let the rivalry continue).

A hashtag, by the way, is just a way to tag your photos. Hashtags categorize or describe the picture or details at hand. So, when I post a picture with Elvis and Penny, tagging #dogsofinsta is an easy way to reach more viewers and gain more likes.

To get started, you'll need a practice owned smartphone (or iPod Touch). Then you'll need to download the Instagram app, even though you can browse photos through the Instagram website, you'll need to have the app downloaded and logged in to post photos or short videos to your own account.

Now, without further ado (and if you promise not to use too many hashtags), let's start using Instagram to interact with pet owners in fun and meaningful ways.

*Get started by taking these simple steps:*

- (1) Download the latest version of the app to avoid bugs and ensure you have the best version possible at your disposal. Make sure you use a practice owned device, and pick a user handle. Your handle is your social media short name and should contain the keywords of your veterinary practice name. My handle is *@EricGarciaFL* and I use it consistently across social media channels to make sure veterinary practices and pet owners can find me easily.
- (2) Switch your account to a business profile by following these simple steps (<https://www.facebook.com/business/help/502981923235522>). This is important because it will unlock a full range of advertising and analytics options that are otherwise unavailable. Don't forget to include a bio, your website, your practice phone number and a nice, high-resolution logo.
- (3) Now, download the free Perch App ([perchapp.com](http://perchapp.com)), which will automatically notify you via email and/or push notification when a pet owner or client tags your location on Instagram. Since you can't control whether your veterinary practice is listed or what people post with *their* location tag, the Perch App keeps you a step ahead by notifying you instantly when a post is made.
- (4) Finally, it's time to start posting! Look at Instagram as your opportunity to share exclusive behind the scenes access to your veterinary practice. Post compelling pictures of your hard-working team and the occasional selfie to let pet owners know who's behind the camera (as if I needed to convince you to take another selfie!) As long as they sign a photo-release form, you can even post pictures of pet owners and their pets. In general, have fun with it and don't look too surprised when you see new likes flooding in almost instantly after posting! One of the most fun parts of Instagram is the immediate feedback you'll get after a great post.
- (5) Finishing touches on a great post can include hashtags that appropriately describe your local area, theme or mood. For example, #Happy #TampaBay #DogsofInsta (with emojis optional) might be the perfect addition to a post with you and your dog's outside on a sunny day.

By creating an Instagram account, converting to a business profile and using Instagram just like pet owners do, you'll tap into something very special. People share photos of their pets and themselves not because they must, but because of the incredible joy that these pets bring and the fun of it all!

The more you tap into this feeling and share this bond with those coming to your practice, tagging pictures and commenting along the way, the more you'll see that your pet family is even bigger than you first thought it was.

Now that's something worth posting about.

Follow me on Instagram at @EricGarciaFL!

## TOP DIGITAL COMMUNICATION TIPS: PART ONE AND TWO

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### A DIFFERENT GENERATION OF PET OWNERS

When the doors of your veterinary practice swing open in the morning, you want pet owners of all types to come inside to welcome arms. Regardless of their age, background, location, vocation or any other type of classification, pet owners want personalized and attentive care that won't break the bank, no matter who they are.

We now see that two generations consist of a large volume of pet owners and foot traffic at your veterinary practice. As of 2016, Millennials (those whose age range between 18 – 34) have surpassed the Baby Boomer generation (whose age range between 51 – 69) for the very first time. With approximately 75.4 million Millennials and 74.9 million Baby Boomers, these two demographics make up a huge swath of America's current population.

However, these two demographics can differ greatly with the way that they operate on a daily basis and what they consider to be effective methods of both conducting business and going about daily communication.

**This makes it all that much more important to understand and respect both demographics, while learning to run your veterinary practice in a way that will benefit them both!**

This approach is truly the only way to ensure you won't alienate one demographic over the other, and that your business won't suffer as a result. I've combed through *Forbes*, *Business Insider*, and *The Wall Street Journal* to learn more about ways that these two demographics relate and ways that they differ. I've also spoken to dozens of veterinary practices about what they experience on a daily basis, and have come away with a few primary takeaways that you can integrate to ensure your veterinary practice remains a destination of choice for both demographics, and of course, for those in-between.

First, let's take a look at some of our overarching themes. Baby Boomers typically have a more traditional approach and tend to be later adopters of technology. The Internet revolution simply came along later in their lives, so this wasn't an integral part of doing business or communicating for the majority of their careers. As a result, Baby Boomers tend to enjoy in-person communication, reviewing paperwork with hard copies and other nuances that align more with their habits, traditions and background.

Millennials, on the other hand, have been raised with the advent of the Internet and typically use technology as an absolutely integral part of their lives. They do research online, they Skype with friends, and they may be much more comfortable with online bookings and open to remote consults instead of in-office ones.

However, this certainly doesn't make any particular preference more valid than any other. This only shows us that different demographics may view the same procedure or

offering in a very different way. For example, if you offer appointment bookings through an app, this may absolutely delight a young millennial who uses their iPhone for everything. After all, they book hotels with their Expedia app and they sign paperwork with DocuSign, all without printing a single piece of paper! This same app however, might not thrill a Baby Boomer who uses an iPhone, but mainly for calls, and otherwise prefers hard copies of appointment reminders and telephone calls.

So, let's take a look at different techniques your veterinary practice can take to ensure that you *delight both populations*, without making anybody feel left out!

***“Communicate with each client in a way that honors the individual and their specific preference!”***

- (1) Technology is an incredible tool. But it still doesn't provide the peace of mind that a solid handshake can, especially to a more traditional demographic. If your next pet owner is a Baby Boomer, you may want to consider designating a bit of extra time to in-person communication and offering to answer any questions while they're with you during a visit.
- (2) If you're going to enhance your veterinary practice with new offerings like an app or online portal for prescription refills or online bookings through your practice website, make sure everything is presented in a clear and methodical way.

Don't assume that pet owners already know how to use the technology at hand, but instead, make sure that everything that's integrated is clear and easy to use for everyone.

- (3) Don't assume that Millennials don't want to chat with you in-person about their pet. They may have booked their appointment through the app, and they may be flipping through Instagram in the waiting room, but the feedback you can get from them during their visit is still invaluable, and they crave information about the care of their pet. In fact, in a recent DVM360 study called “Pet Owner 2.0”, it was reported that “63% of millennials say staying current on pet health topics is important, compared with 54% of boomers.”. It's easy to argue more than half of both generations want information on how to best care for their pet(s).
- (4) Remember, every pet owner and every person is different! By training your team staff to listen well, accommodate feedback and respond directly to client reviews, you'll be taking steps to get ahead of the curve and honor every client relationship in a meaningful way.

You can take notes in a client's file about their preferred methods of communication and provide tips to make their next appointment even smoother than the one before!

Demographics will always shift and it will always be up to veterinarians, staff and practice owners to adapt to the change at hand. Still, adapting is much easier with an open mind, open heart and open ears!

Stay tuned to the trends and do your best to offer new technology and convenient solutions to pet owners. But be sure to also offer more traditional methods of client engagement which are tried and true, and are likely to remain important fixtures of the industry long into the future. This includes in-office visits, hard copies of paperwork and other established methods of doing business.

When you customize your approach to benefit each client individually, you make sure that everyone feels welcome at your veterinary practice and avoid the pitfalls that can come when you make an assumption about preferences.

No one particular demographic is more important than the other. Valuing them both in the ways that you choose to communicate and conduct business is the only surefire way to make sure that every pet owner who comes to your veterinary practice leaves delighted...and comes back again.

### **USE STORY TELLING TECHNIQUES TO COMMUNICATE**

It's proven true time and time again that people gravitate toward a good story more so than just numbers or facts. We look for narrative when attributing meaning, simply because it resonates more deeply with us than an isolated statistic. Let's look at some examples below:

#### *Approach #1 – Not Recommended*

Simply Done Veterinary Clinic is a full service animal hospital. We offer state-of-the-art-care and advanced diagnostics.

#### *Approach #2 - Recommended*

Dr. Garcia founded our veterinary practice on the core belief that by enriching the lives of pets, we enrich the world around us. The staff and veterinarians at Simply Done Tech Clinic take immense pride in this philosophy, bringing this belief to action by implementing passionate, compassionate veterinary care.

*While Approach #1 is technically accurate, it won't compel a pet owner to visit your hospital, and it won't help to gain interest and trust like Approach #2 does.*

*Let's look at another example:*

#### *Approach #1 – Not Recommended*

Dr. Garcia was born in Tampa, Florida. He graduated from the University of Florida in 2000. He has 2 dogs by the name of Elvis and Penny. Dr. Garcia is excited to meet both you and your pet!

#### *Approach #2 - Recommended*

Dr. Garcia knew from a young age that pets were his passion. The joy and wonder of a happy pet immediately inspired Eric to pursue a career in veterinary medicine after completing his undergraduate degree. Now, as the founder of a successful veterinary

practice, Dr. Garcia does what he loves each and everyday. Stop by soon, because Dr. Garcia can't wait to meet you and your pet!

*As people, we crave a good story! Make sure that your veterinary practice is telling your tale, and you'll be amazed at the results that can come from a more narrative-driven approach to marketing, social media and more.*

## SENDING TEXT MESSAGES

I recommend utilizing one-way text messages with pet owners, because this approach has some distinct advantages over two-way texting that is more commonly used when engaging with friends and casual colleagues. There are a variety of reasons that go into this recommendation, but the predominant reason is because two-way text messages open you up to non-stop communication, a whole host of follow-up expectations and perhaps most importantly, potential legal repercussions. Instead, one-way text messages allow you to dictate the content and tone of communication, insulating you from additional liability while still giving you the benefits of instantaneous communication.

If you're interested in getting started with a text message service for your veterinary practice, I recommend using Zip Whip or purchasing a cell phone line (which typically will run you less than \$12 per month), effectively skipping out on the extra phone but using the extra number. Once using the extra line, you can download the texting app for that specific carrier that work specifically with your mobile device (whether it's iOS, Android or other) and program effective auto-reply statements such as the following:

*This is an unmonitored number;  
Please call our practice directly at (xxx) xxx-xxxx*

This allows you to send a particular pet owner in the right direction, without assuming the liability of the two-way text message. You can also easily set auto-reply messages and keep track of any messages that you've sent out.

I recommend sending pet owners simple and concisely constructed text messages whenever a pet is dropped off for surgery, a procedure or appointment, as well as boarding or grooming services. Even just a simple text message can help you to alleviate any potential worry from a pet owner, and demonstrates your accountability and care in one fell swoop. Something simple, yet compassionate will do:

*Hi Mr. Garcia! Elvis is doing great and is recovering from his procedure. We will call you shortly!*

Thanks to rapidly accelerating forms of technology, text messages can be sent to any type of mobile device, and even the new Apple Watch (or any wearable technology)!



Even a brief text message can go a surprisingly long way when it comes to building a steadfast bond with a pet owner. Not only are you adapting to new technology, but you are going above and beyond to stay in touch and alleviate any possible concerns that could crop up along the way. This is a significant way to differentiate yourself from competitors, cut down on extra calls from pet owners wanting updates, and show that you're truly committed to every step of the client experience at your practice.

Yes, new technology does open up new avenues of liability, most of which are covered by opting into the one-way text message feature instead of the two-way. Still, you will need a consent form from your clients before sending them text messages. You can easily capture this consent by adding a brief, additional section to any new client registration form, or a drop off procedure/authorization form. While the adjustments are simple, they're important steps of creating a technologically equipped infrastructure for your veterinary practice.

The resources at your disposal don't stop there. There are also third-party companies such as AllyDVM, Vetstreet, Petly or ePetHealth that allow you to send pet owners text messages regarding due and upcoming appointments. These services seamlessly integrate with your existing practice management software, and will send text messages to clients automatically. Automation is absolutely imperative for this level of communication, and some services like Vetstreet and Petly will offer two-way texting for confirmations. A simple, "Y" or "N", will allow clients to confirm or dissolve appointments, and a list will automatically generate to display who has confirmed their appointments and who has yet to do so. You can then make follow-ups as necessary, leveraging the convenience and quick communication that these services have to offer.

The bottom line for your veterinary practice? These features are an outstanding way to increase compliance and reduce missed appointments that cost your veterinary practice a significant amount of money over time. It's proven that pet owners are more likely to respond to a text than a phone call, and by adapting to text-message technology off the bat, you'll be quick to notice increased compliance and efficiency within your veterinary practice. Still, it's important to remember that this form of technology isn't meant to

replace more traditional methods of communication such as routine phone calls, but is instead meant to supplement traditional communication. Calling your clients regularly remains important, but is in fact complimented by the extra effort of text-message communication.

It's never too late to get started. In fact, the sooner you adapt to these recommended features, the sooner you'll start to notice results.

### ***Tips to Get Started Sending One-on-One Text Messages***

- (1) Find the platform that works best for you and your veterinary practice. I really like ZipWhip and is popular in veterinary practices or perhaps adding an additional cell-phone line? Depending on your existing structure, you may find that some approaches work better than others. Find the platform that's best for you, and sign up!
- (2) Find a champion (or two) and try out the service! Assign these team members to begin sending out test text-messages before rolling out the service to pet owners in real-time. This extra measure will allow you to work out any bugs, and ensure that by the time it's ready for public use, the service is completely ready for primetime.
- (3) Gear up and get going; start rolling out your new text message service and have fun!

I know that your practice will benefit tremendously from the increased communication and accountability that come with text messaging and confirmations. You'll gain an advantage over the competition and let pet owners know that you're thinking of them with a single gesture. The best of tech and contented pet owners; now what could be better than that?

## LEADERSHIP LESSONS THAT WILL CHANGE YOUR LIFE

**Dr Dave Nicol**  
**BVMS Cert Mgmt MRCVS**  
**VetX Founder**

Leadership is one of those topics with an almost bottomless pit of resources describing it. Despite this, a useful working definition can sometimes feel a little hard to pin down in practical terms. This morning, as I was doing my daily journaling exercise to start the day the following flowed forth. I thought it might be worth sharing as I travel on my own messy journey as a leader. Let me know what you think.

### **Leadership is:**

The daily ritual of positively influencing those around you such that they can achieve their latent potential over the course of each day, week, month, year and lifetime.

### **You achieve this by:**

1. Mastering your emotions - particularly fear and anger.
2. Applying good judgement - having the serenity to know what you can fix, the courage to know what you cannot and the wisdom to know one from the other.
3. Starting now and seeing every occurrence as a chance to develop better outcomes by practicing the first two things.

### **This will take:**

A lifetime of practice and dedication - kings are born, managers are appointed, good leaders work at it every second.

### **Your reward:**

The fulfilment and serenity that comes from seeing your world change positively as result of reciprocity when you positively change the world of others.

### **A Revealing Conversation About Leadership**

I like questions. And good questions deserve answers and often lead to fascinating conversations. So when I saw this on the Vets: Stay, Go or Diversify Facebook group I simply could not help but jump in.

One of the group members asked the following question.

*“Question for practice owners, managers, directors: what do you want from vets? What makes you want to keep a vet? I expect high turnover is a big one...? I am curious about how we can be better vets so that we increase our value to the practice?”*

And I could not help but jump in and answer... [Profanity warning].

*“I want you to give a major F\*@K about the following:*

- *"us" - the team.*
- *Your clients - if you can do clients then you can do vet med. If you can't then you are in trouble.*
- *Your patients (obvs).*
- *You.*
- *Really, YOU in quite a big way.*
- *Money and not be embarrassed or ashamed about it. It'll never come first, but it is there and we all need to work on this.*
- *I want you to be willing to learn and grow.*
- *I want you to help others learn and grow.*
- *I want you to be willing to be amazing.*
- *I want you to leave your ego at the door.*
- *I want to laugh and cry with you at the shit we have to deal with.*
- *I want to be able to have a beer with you.*
- *I want you to understand that I care very deeply about helping you get where you want to go and helping you become what you can be.*
- *I want you to accept that you are not perfect, that none of us is and that's OK. Failure is OK.*
- *I want you to be happy.*

*If you can do that, then you'd be a lot better than I was when I started out, and I'd be very, very proud to call you a teammate.*

*(And if you can be that, or be willing to work towards it.... could you also never leave?)*

*Now let me ask you back.... what do YOU want/need me to be as a leader? Because I think that probably matters more...”*

The vet who asked the question then responded with this:

*“David, for me, off the top of my head it is similar to many of the things you said you want from a vet.*

*Plus either be a good role model or be there to support and facilitate us vets to achieve the desired outcome.*

*To understand what it is really like to do the job and care about whether or not I have had my lunch today.*

*Have good systems in place to make everything run smoothly and efficiently and listen when we give feedback.*

*I want you to care that I get away on time and that I have had to deal with a really obnoxious client and notice that I dealt with it well.*

*Call me needy, but I do like acknowledgement when I work hard. I want you to empathise when shit happens and take time to understand the whole story before you criticise.*

*I think it would be great if somehow bosses and/or colleagues could positively feedback regularly on what we appreciate, value and admire in one another.*

*Sometimes I think it's lack of communication, but generally, I want you to show that I am valued every so often.*

*I want you to provide an environment and facilities conducive to practising good medicine.*

*I also want to be inspired by you and be able to respect you.*

*And I want you to totally trust me and have my back if something goes wrong. Just a few things off the top of my head..."*

And when we were done, it felt like maybe, unwittingly, we just had a really honest conversation that helped to clarify what we have to do as team members and leaders to improve relations and be happy working together.

## **Nine Leadership Lessons**

My notes on becoming an awesome leader/human. Make them real.

### **1. No Ego - Humility**

Ego puts I before others. Ego is your view of yourself. Ego is a trickster. Ego will hold you back. The antidote to ego is to put others first. To ask others for their feedback. To not believe your hype. Be prepared to change if your plan isn't working. Be open and humble to the feedback of others, though it may sting.

### **2. Listening Skills**

Seek first to understand in all situations that confront you. Open questions are the way forward in this regard.

### 3. **Clear Plan**

At all times one should know which way to travel. Make a plan - write it down. Tell everyone what the plan is and what their part in it is. Then go make it real.

### 4. **Be Honest**

You are not Wonder Woman or Superman. You will not be able to do everything everyone wants you to do. Be honest about what you can and cannot do. People will respect you more. Be honest with others as well, be they colleagues, suppliers or clients.

### 5. **Build Good People Up (hire well, empower, train, coach, mentor, delegate, respect, recognise)**

If you want to reach for the sky then you are going to have to stand on the shoulders of giants. People need certain things in order to grow:

1. Nourishment (training, coaching and feedback).
2. Sunlight (respect and a happy environment to work in).
3. Space to grow (projects and tasks to work on where they have responsibility).
4. Freedom to do the work (do not take responsibility away).

When people get these things they will grow faster and further than anyone thought possible.

If you have people who get these things and fail to grow then move these people on with kindness and compassion.

### 6. **Execute The Plan - Be Effective - Focus and hold everyone accountable for their part.**

Getting stuff done means having a plan and then focussing on your bit of that plan until it is done. Take tasks, break them up then work on nothing but these tasks in order to get a massive amount done.

Hold others accountable to do the same.

### 7. **Praise & Recognise**

When you people grow then praise them often, praise them proudly/loudly and praise them publicly. When they stumble, catch them quietly and privately.

### 8. **Have a Growth Mindset**

If you think you can't do it, then you won't. If you think you can, then you might. A growth mindset is the ability to see failure as an experience and an opportunity to learn what we can do better. Curiosity did not kill the cat. It allowed it to run up fences as if gravity doesn't exist.

### 9. **Become Resilient/Able to Function**

Resilience to stress and life's challenges is a battle of the mind and body. Beware FEAR. Beware of anything that saps your resolve and actively seek out that which boosts your resilience.

Actions that will help:

Meditation/mindfulness

Journaling

Exercise

Stoicism

Gratitude

Sleep

## THE TRANSFORMATIONAL MAGIC OF OBJECTIVES

Dr Dave Nicol  
BVMS Cert Mgmt MRCVS  
VetX Founder

### Part 1 – What are cascading objectives and how will they help your practice.

Many veterinary practices are run on a rather adhoc basis. Without clear leadership and clear guidance in the form of job descriptions and job objectives is hard for a team to function as a unit and equally difficult for individuals and managers to know what is expected on them.

This situation is what I describe as 'Jazz Management'. It is an unhealthy situation for a practice to be in, but is all too common.

Objectives help to remove this problem and area great way of providing clarity of purpose to your team.

So what is an objective?

### Part 2 – Writing effective objectives

As with many written corporate documents, management objectives are often wooly, non-specific and bloated with meaningless management- jargon. In other words they are completely useless!

Objectives have certain characteristics:

SMART – Specific, Measurable, Achievable? (Or perhaps not), Realistic (I have changed this to Relevant!), Timed.

Now however we have a move away from this model into the BHAG style.

BHAG stands for Big, Hairy, Audacious, Goals.

The British cycling team, team sky is an awesome example of this at work. It now dominates world cycling winning almost every conceivable gold medal on the track and on the road, including the Tour de France.

### Part 3 – Objectives: measurement, feedback and reward

An objective by definition has to be measurable. With today's practice management IT systems is it possible to measure just about everything, so what things matter? How do you measure them and how do you get the best reaction from your team?

Dr Nicol will discuss how to measure objectives that matter. Why feedback is so important and a different way to reward.

#### Part 4 – Objectives in action

I'll present 2 case studies will be presented where clearly written objectives made a remarkable difference. One from outside where objectives allowed the business to double profits each year for five years running. The other is from the veterinary industry and shows how a clearly written objective helped one clinic's stock levels drop from 40% of sales down to 21% and stay there! Feel free to take some notes from these.

**Final Destination:** You'll be inspired to write good cascading objectives that will take the guess-work out of practice performance management.

**MEASURE FOR SUCCESS: GROW YOUR BOTTOM LINE NOW**

Dr Dave Nicol  
 BVMS Cert Mgmt MRCVS  
 VetX Founder

**The Holy Trinity of Delivering a Successful Veterinary Service**

For a veterinary clinic to deliver a successful and sustainable service certain things have to be in balance: People, Profit and Practice

**Part 1- Profit – The Financials- turnover and profit.**

- What should we be measuring? List three things that you think will be important to begin measuring in your clinic tomorrow. How will you get this information reliably?

1.....  
 2.....  
 3.....

- Beware analysis paralysis – there is so much information now available that it can be difficult to know what to look at. Each business will be different in what matters most.
- The obvious – turnover, profit, average spend, client visits.
- The not so obvious – lost charges, ratio of first to second consults, percentage breakdowns of veterinary turnover and what you can learn about your vets.
- The Four Cs – Four deadly sins that will sink your business and how to combat them. The Four Cs are not just Dave Nicol’s contribution to veterinary management theory but one of the most important business management tools you will have. If you only stay awake for one 15 minute period the entire day, then this is it!

**The Four Cs**

Confusion – we’re not sure what we should be charging because no-one trained us.

Clipping – We provide clients with verbal guesstimates rather than printed estimates, so when we get the price wrong we massage the invoice total to reflect the guesstimate, not the reality.

Carelessness – we missed the charge because we were sloppy or working under time pressure.

Charity – we decided the little old lady could not afford the treatment, so we did not invoice for it.

How can we use this information strategically and tactically to build and run better clinics?

## Part 2 – Practice – clinical quality

Clinical audit is one of the pillars of good practice – what is it and how can we use it?

- Clinical protocols
- Invoicing protocols
- Reviews
- Feedback.

Generating the information – choose three to five cases per vet and look closely at what they did. How did it stack up against your protocols? Do you have protocols? Was the standard of care at the required level? Was the billing accuracy at the required level?

This information is easy to get your hands on, though it is expensive in terms of time as really only a vet can do it.

But the info is really helpful for managing quality thereafter. Again the Four Cs referred to above come in very handy in generating data for our clinical audit.

## Part 3 – People – customer and staff care

If we aren't pleasing our clients then the rest is completely irrelevant.

How do we know what clients want? The answer is to ask them. Or to give this another phrase, to do some market research.

How do we know if we are meeting their needs? Do you solicit feedback? Surveys, new client sign ups, complaints, one to one interviews/focus groups, mystery shopping s a vastly underused resource that actually generates a great amount of data and turns staff meetings into awesome learning sessions, not mudslinging matches.

A cool tool you can use to gauge our performance with clients:

Survey monkey – [www.surveymonkey.com](http://www.surveymonkey.com) - a free online survey tool.

Don't forget your staff! A good measure of your quality both as a management group and a clinical institution is how well you retain staff, how engaged they are, morale, the buzz in your clinic.

Reps can be a great source of this info.

**Final Destination:** Getting you hands on useful information is critical to managing a high performing profitable team.

## RECRUIT IN HASTE, REPENT FOREVER

Dr Dave Nicol  
BVMS Cert Mgmt MRCVS  
VetX Founder

The single most important decision you will be responsible for making will be when to hire and who to hire. I believe there is no more important differentiator of success between good businesses and poor businesses than the people who work in it.

Yet this is one of the most chronically under performing area of management in almost every industry. There is a frankly gob-smacking and self-defeating lack of skill or evidence of any learning/ development when it comes to recruitment. And it is costing clinics hundreds of thousands of dollars per vet each year.

In this lecture we are going to learn more about the recruitment process and I'll uncover the top five errors that vets make in recruitment.

### Step 1: Planning for a new hire

- Organisational assessment
  - Do you need to hire? This is a recession.
  - If you don't hire how will you grow?
  - What staff are really going to help the most?
- What are the organisational objectives?
- Why did your last vet leave? Can you conduct an honest and objective review? Most people leave organizations because of their manager, not because of the job.
- This is an important opportunity to revisit the plan/vision. Refocus.
- Assess current team mix. Do you know the strengths and weaknesses of your team and of yourself?
- What traits would best meet the clients, practice and the team needs.
- Involve your staff.

I have been researching as part of a new business I've launched here in Australia, a recruitment tool which will help you to understand what specific personality traits are desirable in a vet, nurse and reception team member.

You have to get this right because the costs of recruitment and induction are huge. Many people think about the cost of the advert and perhaps the time taken to interview as the real cost of recruitment. But that is simply not true.

The real cost is in the impact of that new hires actions on everyone else in your business.

1. **On you** – if you hired someone who's values do not match you own or those of your team then prepare for a draining emotional fight.
2. **On those around you** – if you hired someone with the emotional intelligence of a rhino, then prepare for a lot of upset team members and disharmony as a toxic blame culture springs up around you.
3. **On your clients** – if you hired someone who cannot or will not sell your product and services or lacks the skills to bond wit people then performance will plummet.
4. **On the animals** – this is a profession in which competency is assumed. But my experience says this is a mistake and when clinical actions go wring it gets expensive to fix them.

Overall, I estimate (and every business adviser that I know agrees) that the difference in performance for a Good vet Vs a Poor vet is measured in the tens and most likely the hundreds of thousands of dollars. You can either have this money, or lose it. Your choice!

## Part 2 How to do recruitment and selection well

As they say, proper preparation prevents p\*\*s poor performance!

So let's get started with helping you out.

### Step 2: Writing your job advert

We write a laser targeted advert based on our analysis of our business needs. You are not trying to attract as many people as possible. You are trying to attract only the people who match you values and have the skills to do this job. This represents a fundamental shift in approach for virtually everyone I know doing recruitment.

We are going to infuse your advert with personality...not play buzz word bingo!

It will look uncomfortably different – that's the point!

### Step 3: Designing tests to rule poor candidates out at a low cost.

Most people jump straight into an interview at this stage which makes no sense for two reasons:

1. Interviews take up a lot of your time and are hence expensive.
2. You probably aren't very good at interviews anyway!

The solution to this is to make sure that you have a series of selection tests created and ready to send to prospective candidates that whittle them down objectively for you.

These tests can be delivered online, take no additional work once you've created them the first time, make novel use of technologies like video, are testing for characteristics we know we need based on our personal specification.

I use written scenarios with multiple layers to the problem. I use video, I use spreadsheet tasks. What can you use.

If you felt this is too much work, or that you perhaps don't have the skills to create video, or just the time to write good, useful tests then please again visit [www.recruitrightforvets.com](http://www.recruitrightforvets.com) to find a range of these tests available for download.

#### **Step 4: The interview**

##### Interview skills

- Like, trust, respect.
- Clinical skills
- Values based techniques
- Personality profiling
- Going on gut plus weird and wonderful.

A simple way to get to the information you need is to ask the following question series:

1. have you ever experience situation when [insert thing you are interested I learning about or is relevant to the role] happened?
2. Tell me what you did? – this open question allows the candidate to talk about the experience in their own words, without any leading language. You will get a good view of how they perceive the world by doing so.
3. Tell me why you did it like that? This is a super important question because why people do things is a reflection of their core values.

Repeat this over and over to find out more and more.

#### **Following up – Is an opportunity to look good or bad.**

I always email people to let them know I received their CV and of I don't want to hire them also. It just leaves a good impression and is polite. Both are helpful things in our small world!

I'm also happy to give feedback to people who don't make it. The process I'm describing uses a select set of criteria to test people, making it very easy to explain why you didn't give someone a job.

### Part 3 Induction – the first month

Unbelievably, most practices do not have an induction process. But if you want your new recruit to get on their feet and perform fast you have to have a structured onboarding program that serves to allow them to quickly perform the job as required.

**Final Destination:** Take recruitment and induction seriously if you want a top performing practice.

# PLENARY SESSIONS

## MEDICAL USE AND TOXICOSIS OF MARIJUANA IN VETERINARY MEDICINE

Carolina Duque, DVM, MSc, DVSc, DACVIM (Neurology)

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### Introduction

The use of the *Cannabis* plant and its derivatives in veterinary medicine is a rapidly evolving field. The two major cannabinoids are delta-9-tetrahydrocannabinol (THC), the primary psychoactive component, and cannabidiol (CBD), the primary nonpsychoactive component and the component purported to convey medicinal effects.

CBD has limited oral bioavailability (6% in people, 0-13% in dogs) due to high first pass metabolism in the liver. *Cannabis* plants have, on average, a 1.2% THC concentration and only 0.2% CBD concentration, indicating that the use of medicinal products directly derived from the unaltered *Cannabis* plant is unlikely to achieve serum concentrations of CBD with therapeutic effect, while producing undesirable psychotropic effects associated with the THC content. Thus there is a need for synthetic or purified medication containing solely CBD in order to achieve the medicinal potential. Interestingly warning letters were issued by the FDA in the year 2015 to Canna Companion and Canna-Pet indicating that the products were unapproved animal drugs and their marketing was violating the FD&C act.

There are many purported uses of CBD, such as seizure control, cancer therapy including anti-neoplastic effects, treatment of inflammatory bowel disease and reversion of diabetes mellitus. *Cannabis* products have been shown to have neuroprotective ability, cause an improvement in pain, have anti-inflammatory, anti-oxidant, anti-pruritic and immunomodulatory effects, anti-emetic ability, an ability to stimulate bone formation, and anxiolytic properties.

The recently legalized use of the *Cannabis* plant in Canada leads to a lot of confusion as to the ability to prescribe marijuana to our patients. In addition, legalization may result in an increase in the number of cases of *Cannabis* toxicosis in Canadian pets.

### Neurologic disease

The exact mechanism of seizure suppression by marijuana is unknown but it is likely related to depletion of calcium that typically is excitatory to the cells. Among neurologists there is controversy on whether marijuana products obtained by owners have helped to control seizures in animals. Seizures occur through infinite pathways. If the events are directly linked to endocannabinoid neurotransmitters or fatty acids amid hydrolase issues, the use of marijuana may have a positive effect but this needs to be proven in controlled studies. Because cannabinoids are metabolized by the P450 system and the P450 system tends to concentrate solely on metabolizing CBD when presented with it, it may inhibit or slow down the metabolism or clearance of other medications processed by the P450, thus increasing their blood levels. In humans (and likely in pets), it has been documented that people required less diazepam and diazepam derivatives as well as phenobarbital when taking hemp oils.

The controlled substances Act in 1970 designated marijuana as a Scheduled I drug, a categorization for drugs with high potential for abuse and no currently accepted medical use. There is currently no evidence-based support of the benefit of medical marijuana in veterinary medicine. However, it will likely come with time. Interestingly the drug Epilodex, which

is 99% CBD, has had successful stage 3 FDA trials in severe forms of childhood epilepsy. Therefore it is reasonable to believe that CBD may have the potential to control seizures in dogs.

### **Cannabis in veterinary cancer patients**

Study of the use of *Cannabis* products in veterinary oncology is limited at this time. In human oncology, cannabinoids are used to support patients undergoing treatment by reducing vomiting, nausea, and pain, and stimulating appetite. Although some studies have shown anti-neoplastic effects with *Cannabis* products, there is evidence that there may be negative effects, such as a reduction in host anti-tumour immunity.

### **Cannabis in veterinary internal medicine**

Study of the use of *Cannabis* products in veterinary internal medicine is virtually non-existent at this time. However, if you search for CBD uses in fields of human internal medicine, there are an increasing number of reports. Of greatest note are studies involving diabetes mellitus, and inflammatory conditions such as inflammatory bowel disease. Often, studies are carried out in mice, which may be translatable to some areas of veterinary medicine. Studies have shown an increase in diabetic remission, and a reduction in diabetic side effects, in mice with diabetes mellitus treated with CBD. These improvements, and also those seen in inflammatory conditions, appear to be primarily related to anti-inflammatory effects.

### **The toxicology of *Cannabis* products**

While there may not be much information available on the therapeutic use of *Cannabis* products in veterinary medicine, there is a large amount available on the toxicology in dogs and cats. In the United States, where an increasing number of states are legalizing the use of medicinal marijuana, the Pet Poison Hotline reported a 448% increase of reports in the past 6 years. After Colorado legalized the use of medicinal marijuana, the frequency of marijuana toxicosis in dogs at two veterinary hospitals increased 4-fold over a 5-year period.

Most cases of *Cannabis* product toxicosis in pets are due to accidental ingestion of laced products, although toxicosis secondary to marijuana smoke inhalation is possible. Owners may be reluctant or unwilling to admit that their pet had exposure. In addition, many pets ingest additional toxic substances along with the *Cannabis* product, such as chocolate, which will complicate toxicoses. Some forms of *Cannabis* products may contain additives of which the owner is unaware.

In some cases, treatment may be pursued with an assumption of exposure, and tailored to clinical signs at presentation. Clinical signs can be seen within minutes to hours of exposure. Clinical signs of poisoning include lethargy, incoordination, disorientation, a dazed expression, slow response times, ataxia, mydriasis, hyperesthesia, tremors, twitching, vomiting, ptialism, dribbling urine, bradycardia or tachycardia, vocalization, hyperactivity, and coma.

Treatment of pets with *Cannabis* intoxication varies with presentation, and also with exposure to additional toxic substances. Many cases can be treated on an out-patient basis; a 2012 study in the *Journal of Veterinary Emergency and Critical Care* indicated that 58% of cases were treated this way. Activated charcoal can be administered if the *Cannabis* product was ingested within 4-6 hours. Benzodiazepines can be administered if tachycardia and/or excessive nervous system stimulation are noted. Oxygen therapy may be required, and safety provided so that the pet

does not injure itself or aspirate vomit. Vital parameters should be monitored. If hospitalization is required, it is rarely needed for more than 24 hours. Although intra-lipid emulsion therapy can be used due to the very high lipophilicity of cannabis, it is rarely needed. Since there is near absence of cannabinoid receptors in the brain stem, marijuana does not stimulate the autonomic nervous system. Mortality is low; the previously mentioned study in the Journal of Veterinary Emergency and Critical Care showed a mortality rate of 1.6%, and the deaths could not be definitely attributed to marijuana exposure.

### **Client education**

Although the legalization of marijuana happened a few months ago, this legalization does not immediately extend to pets. Veterinarians commonly prescribe off-label medications, however marijuana is not currently a substance that has enough research regarding its medicinal use in companion animals to provide backing for off label use. However, it is likely that veterinarians will have a significant increase in requests from pet owners to prescribe marijuana and its products for their pets. Until more research is available, the safest approach is to not prescribe marijuana products for pets.

If owners are still interested to use medical marijuana for their pets against FDA/regulatory advice, they should look for:

Water-soluble hemp (increases absorption by 80%)

Look for USDA certified organic (it has to meet strict criteria of not been contaminated with harmful ingredients to achieve this status)

Look for 0.0% THC- the maximum allowed is 0.3% THC by law in medicinal marijuana products

References available from the authors on request.

## TICKS AND LYME DISEASE SURVEILLANCE IN ONTARIO

Katie M. Clow, DVM, PhD

### Ticks in Ontario

Ontario is home to numerous species of ticks. Some have long been inhabitants of our natural environments, such as the American dog tick, *Dermacentor variabilis*, and the groundhog tick, *Ixodes cookei* [1]. Aside from being pests, these species currently pose minimal risk to our companion animals [1,2]. On the other hand, we have also seen the invasion of other tick species into the province, which are vectors for several pathogens of animal and human health significance.

The blacklegged tick, *I. scapularis*, is the most notable example of northward range expansion. In the early 1990's, one longstanding population existed at Long Point [3]. Now, established populations of blacklegged ticks are present in many areas of southern, eastern and northern Ontario [4], and its range continues to expand [5]. The blacklegged tick poses a risk because it is the vector for *Borrelia burgdorferi*, the causative agent of Lyme disease in humans, dogs and horses, and *Anaplasma phagocytophilum*, the causative agent of anaplasmosis in humans, dogs and horses [6-8].

The lone star tick, *Amblyomma americanum*, is not believed to be established in the province, but is routinely introduced on migratory birds [9,10]. In the southern United States, the lone star tick can transmit *Ehrlichia chaffeensis* and *E. ewingii*, which causes Ehrlichiosis in humans, dogs and cats, and *Cytauxzoon felis*, the causative agent of feline Cytauxzoonosis [6]. Little data currently exists on the infection prevalence of these pathogens in adventitious lone star ticks.

Pets can also be bitten by ticks if they travel to more southern areas (e.g., United States, Mexico), thus exposing them to other tick-borne pathogens. In these cases, tick species such as the brown dog tick, *Rhipicephalus sanguineus*, and the Gulf coast tick, *A. maculatum*, become important considerations.

### Tick and Tick-borne Disease Surveillance

Public Health Ontario (PHO) conducts several types of surveillance for ticks and tick-borne diseases. Passive surveillance is present in many public health regions. Tick submissions are only accepted from people (and no longer from pets), and in regions with high tick density (e.g., Leeds and Grenville, Kingston and surrounding area), the program has been discontinued [11]. Active surveillance, typically conducted via tick dragging, is routinely employed in many areas. If one blacklegged tick is found at a site during both spring and fall drags, the area is deemed a risk area and added to PHO's Lyme disease map [4]. This map is published annually and provides an up-to-date resource for risk assessment and client communication. Finally, human Lyme disease is a reportable disease in Canada, and incidence is assessed routinely [12].

The Pet Tick Tracker is a citizen science tool launched by Dr. Scott Weese at the Ontario Veterinary College. It now resides at [www.petsandticks.com](http://www.petsandticks.com). Pet owners and veterinarians from across Canada are encouraged to submit information on any tick found on a pet. The option to submit a photo or the actual tick sample for identification also exist. Maps are routinely produced showing the location of tick submissions [13].

eTick is an online surveillance tool that produces a map of tick distribution using submissions of high-quality tick photos [14]. It is currently only present in Quebec, but there are plans to expand to other provinces in the near future, including Ontario.

Several researchers in Ontario also collect ticks through a variety of methods and this data contributes to surveillance for ticks and tick-borne diseases [15,16].

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# 2019 OVMA CONFERENCE SPEAKER BIOS

## SMALL ANIMAL PROGRAM:

**J. Brad Case, DVM, DACVS**

**Associate Professor, University of Florida**

Dr. J. Case is a small animal surgeon and associate professor at the University of Florida. His primary clinical interests are in minimally invasive surgery and interventional radiology procedures to minimize pain in his patients and to improve clinical outcomes. His major research is in the development of biodegradable, nanocomposite, arterial stents which inhibit neointimal hyperplasia following coronary artery placement. He is an advocate of non-terminal, translational research using naturally occurring disease models in place of purpose bred animals. He is also actively involved in developing high-fidelity models for surgical training of veterinarians.

**Douglas J. DeBoer, DVM**

**Professor of Dermatology, University of Wisconsin**

Dr. Douglas DeBoer is a graduate of the School of Veterinary Medicine, University of California-Davis, and completed postgraduate training at Michigan State University and at UC Davis. In 1986, he joined the faculty of the School of Veterinary Medicine, University of Wisconsin-Madison, where he is currently Professor of Dermatology. His research and clinical interests center on the immunology of recurrent and chronic skin diseases, with a focus on canine allergic skin disease. He is a diplomate of the American College of Veterinary Dermatology and has served on the scientific editorial boards of the American Journal of Veterinary Research and Veterinary Dermatology, and is former chair of the International Committee on Allergic Diseases of Animals. Dr. DeBoer is the author of more than 200 clinical and research publications in veterinary dermatology.

**Jennifer Devey, DVM, DACVECC**

**Consultant and Emergency and Critical Care Specialist**

Jennifer is a graduate of the Ontario Veterinary College, University of Guelph. She completed an internship and an emergency and critical care residency at the Animal Emergency Center in Milwaukee, Wisconsin and became a Diplomate of the American College of Veterinary Emergency and Critical Care in 1996. She completed a surgical residency in 2004 studying at several private practices in the US. Jennifer has been director of emergency and intensive care services at a number of large private referral practices in Canada and the United States. She is currently working as an independent consultant and emergency and critical care specialist. She has published over 50 articles and book chapters, is actively involved in research, and is a member of seven professional veterinary organizations around the world. Jennifer enjoys teaching and is actively involved in training residents as well as lecturing and teaching workshops to nurses and doctors.

**Maria Carolina Duque, DVM, MSc, DVSc**

**Head of Neurology Department, Mississauga-Oakville Veterinary Emergency Hospital**

Dr. Maria Carolina Duque completed her training in veterinary neurology at the Ontario Veterinary College (OVC), University of Guelph. She obtained a Masters degree in veterinary neurology in 2000 and also completed a DVSc program (neurology) in 2003. Dr. Duque has fulfilled the strict guidelines set by the American College of Veterinary Internal Medicine in order to receive her board certification in neurology. In 2004, Dr. Duque joined the Veterinary Teaching Hospital of the OVC as a Clinical Neurologist and provided training to graduate and undergraduate students. In 2006, Dr. Duque founded the neurology department at the Mississauga Oakville Veterinary Emergency Hospital and has been working in the service for the last 12 Years. Dr. Duque is involved in continuing education programs locally and internationally.

**Barden A. Greenfield, DVM, FAVD, DAVDC**

**Owner, MidSouth Veterinary Dental Referrals**

Barden Greenfield is a graduate of Mississippi State University College of Veterinary Medicine in 1985. He practiced small animal medicine for 24 years before entering a non-conforming dental residency. He became a Diplomate of the AVDC in 2011. Dr. Greenfield is currently the Past-President of the AVDC and also serves on the Board of Directors of the Foundation for Veterinary Dentistry, which is the educational and fundraising branch of veterinary dentistry in the USA. He has a dental specialty practice in Memphis and Little Rock. In addition to his specialty practice, Dr. Greenfield founded and runs the Mississippi Valley Veterinary Dental Educational Center in Memphis, TN. This center trains DVM's and technicians in veterinary dental techniques and procedures. Dr. Greenfield has published in peer-review journals and has spoken on the national and international level.

**Eric Herrgesell, DVM, DACVR**

**Chief Radiologist, Veterinary Diagnostic Imaging**

Dr. Herrgesell received his Bachelor of Science, and Doctor of Veterinary Medicine degrees, and completed a radiology residency at the University of California, Davis where Dr. William Hornof served as one of his mentors. After completing his residency, he was named a Diplomate of the American College of Veterinary Radiology and joined the faculty of the University of California Davis that same year. He served on the faculty for 7 years bringing curricular innovation in ultrasound instruction receiving the Norden Award for excellence in teaching. Throughout his career he has been actively involved in continuing education. He left the university to help start Veterinary Diagnostic Imaging where he currently serves as Chief Radiologist.

**William Hornof, DVM, MS, DACVR**

**Professor Emeritus, UC Davis and Antech Imagine Services**

Dr. Hornof received his Bachelor of Science and Doctor of Veterinary Medicine degrees, and completed a radiology residency from the University of California, Davis. He was then named a Diplomate of the American College of Veterinary Radiology and joined the faculty of the University of California Davis that same year. He had a distinguished academic career with over a hundred publications in the peer reviewed literature in imaging and informatics. In 2004 Professor Hornof retired as head of Radiology and Hospital Computing at UC Davis to join Elkin Medical Systems as Chief Medical Officer. When Elkin was acquired by VCA he continued to serve as Chief Medical Officer for Sound Technologies and then Antech Imaging Services (both VCA companies). Dr. Hornof has recently retired but continues to serve as a consultant for Antech and Sound. Throughout his career, he has been actively involved in teaching and curricular innovation. He is the current Past President of the American College of Veterinary Radiology, and is serving as Co-Chair of DICOM Workgroup 25.

**Jennifer Kyes, DVM, DACVECC**

**Criticalist, Mississauga Oakville Veterinary Emergency Hospital and Referral Group**

Dr. Kyes received her veterinary degree in 2004 from the Ontario Veterinary College followed by a rotating small animal medicine and surgery internship at the VCA Veterinary Referral & Emergency Center in Norwalk, Connecticut. In 2009, she completed her residency in Emergency and Critical Care at Ocean State Veterinary Specialists in Rhode Island and was board certified that same year.

Dr. Kyes joined the VCA – Mississauga-Oakville Veterinary Emergency Hospital and Referral Group in 2009.

**Jonathan Lichtenberger, DVM, MSc, DACVIM**

**Specialist in Cardiology, Animal Health Partners, North York, Ontario**

Dr. Jonathan Lichtenberger became Diplomate of the American College of Internal Medicine (Cardiology) in July 2017. He practices both medical and interventional cardiology at the Toronto Emergency Hospital.

Dr. Lichtenberger originates from France, where he received his Doctor in Veterinary Medicine in 2012. He then pursued his education at the Oregon State University with a Small Animal Internship, followed by a 4-year combined Residency and Master in Science program in Veterinary Cardiology at the Atlantic Veterinary College, PEI, Canada.

**Richard F. Quinn, DVM, DVSc, DACVO**

***Veterinary Ophthalmologist, Veterinary Eye Specialists***

Rick Quinn received his DVM degree from the Ontario Veterinary College in Guelph, Ontario, Canada in 1981. Following several years of general practice, he returned for a residency and graduate work in Ophthalmology at the University of Guelph. He completed a Doctorate of Veterinary Science in Ophthalmology and obtained Board Certification by the American College of Veterinary Ophthalmologists in 1996. Following 5 years of academia that included teaching undergraduate veterinary students, residents, and graduate students, he returned to private practice establishing the Veterinary Eye Specialists in 2002. Dr. Quinn is an adjunct Professor in the Department of Ophthalmology at Western University in London, Ontario, Canada. He currently serves on the Appeals Committee of the American Board of Veterinary Ophthalmologists. Dr. Quinn has lectured across Canada and internationally. He is the Founding Director of Docs4GreatApes. He serves on both the Canadian and Global Boards of the Jane Goodall Institute. An avid wildlife photographer, he has trekked the rainforests of Africa and Indonesia photographing Great Apes. He and his wife Diane, A family Doctor, have four adult children.

**Lisa Radosta, DVM, DACVB**

***Owner, Florida Veterinary Behavior Service***

Lisa Radosta is a board certified veterinary behaviorist and owner of Florida Veterinary Behavior Service in Southeast Florida. Dr. Radosta lectures nationally and internationally for veterinarians, their staff and lay people. She has written book chapters for textbooks including Handbook of Behavior Problems of the Dog and Cat; Blackwell's Five Minute Veterinary Consult and Canine and Feline and Small Animal Pediatrics. She is the co-author of From Fearful to Fear Free, the ultimate guide for the fearful dog.

Dr. Radosta has published scientific research articles in Journal of Applied Animal Behavior Science and The Veterinary Journal and written review articles for Advances in Small Animal Medicine and Surgery, Compendium, NAVC Clinician's Brief and AAHA News Stat. She is the section editor for Advances in Small Animal Medicine and Surgery. She serves on the Fear Free Executive Council and the AAHA Behavior Management Task Force.

Dr. Radosta has been interviewed for many publications including Cat Fancy, Dog Fancy, Palm Beach Post, NAVC Clinicians's Brief, Sun Sentinel, WebMD, AAHA Trends, Real Simple and AAHA News Stat. She has appeared on Lifetime television, Laurie Live, News Chanel 25 (West Palm Beach, WPBF), Mitch Wilder's Amazing Pet Discoveries, Nat Geo Wild, Animal Planet, News Channel 10 (Miami, ABC), and Steve Dale's Pet Talk. Dr. Radosta also podcasts for VetGirl.

**Nicole Rolfe, BScH, DVM, DACVAA**

***Anesthesiologist, Veterinary Emergency Clinic, Toronto, Ontario***

Dr. Nicole Rolfe is the anesthesiologist at the Veterinary Emergency Clinic (VEC) in Toronto. She completed both her DVM and her residency in anesthesia at Ontario Veterinary College at the University of Guelph. She is a diplomate of the ACVAA.

**Jinelle A. Webb, DVM, MSc, DVSc, DACVIM (Small Animal Internal Medicine), Adjunct Professor, OVC**

***Associate, Small Animal Internal Medicine Department, Mississauga-Oakville Veterinary Emergency Hospital***

Dr. Jinelle Webb received her DVM in 2001 from the Ontario Veterinary College. An interest in small animal internal medicine led to a residency and board certification at the OVC, which she completed along with a DVSc in 2005. In 2006, Dr. Webb started the internal medicine and oncology service at the Mississauga-Oakville Veterinary Emergency Hospital, where she remains today, seeing clinical cases and performing small research projects. She is an Adjunct Professor at the OVC. Dr. Webb is a published author and speaker.

**Joe Wolfer, DVM, DACVD**

***Ophthalmologist, Toronto Animal Eye Clinic***

Joe Wolfer graduated from the OVC in 1988 with his DVM. This was followed by an internship at the WCVI and then a residency in ophthalmology at the OVC. Dr. Wolfer has been in private practice since 1992. He became a Diplomate of the ACVO in 1994 and member of the American Society of Retina Specialists in 2004. He currently works at the Toronto Eye Clinic in Toronto Ontario.

Dr. Wolfer's main area of interest is retinal reattachment surgery and this is his current area of research. He speaks internationally several times per year on this topic and has taught retina surgery to aspiring retinal surgeons in Sydney Australia, Zurich Switzerland and Hong Kong.

Dr. Wolfer's past times include road biking, horseback riding, reading and walking his field spaniel Charlie.

## FOCUS ON INFECTION:

**Maureen Anderson, DVM, DVSc, PhD, DACVIM**

**Lead Veterinarian, Animal Health & Welfare, OMAFRA**

Maureen Anderson is a 2003 graduate of the Ontario Veterinary College and is ACVIM Board-certified in large animal internal medicine. Her graduate research focused on MRSA in horses and equine personnel, as well as hand hygiene and infection control measures used in small animal clinics. She is currently Lead Veterinarian, Animal Health and Welfare at the Ontario Ministry of Agriculture, Food and Rural Affairs, where she continues to work in areas bridging animal and public health, including infection control.

**Katie M. Clow, DVM, PhD**

**Postdoctoral Fellow, Department of Pathobiology, Ontario Veterinary College, University of Guelph**

**Vice Chair, Veterinarians Without Borders Canada**

Dr. Katie Clow is a veterinarian and postdoctoral fellow in the Weese Laboratory at the Ontario Veterinary College (OVC). She began her DVM at OVC in 2007 and during this time, Katie completed internships at Canadian Food Inspection Agency (CFIA), the World Health Organization and the Centres for Disease Control and Prevention. Following graduation in 2011, she spent a rewarding year in practice at a rural small animal clinic, and then worked as a regulatory veterinarian at the CFIA developing proactive biosecurity guidance documents for goat and dairy producers. Katie returned to OVC in 2013 to complete her PhD, which focused on the ecology and epidemiology of the blacklegged tick and the risk of Lyme disease in Ontario, Canada. She has tremendously enjoyed research, teaching and service and hopes to continue her career in academia, with a strong focus on the ecology and epidemiology of vector-borne zoonoses using One Health and EcoHealth approaches to research. Dr. Clow is also the Vice Chair of Veterinarians without Borders Canada. In her spare time, she is an avid long distance runner, cook and gardener and loves spending time with her husband, two dogs and cat.

**Les Eccles, BSc.**

**Apiary Specialist; Lead Specialist**

Les Eccles is the Ontario Beekeeper's Association Technology-Transfer Program Lead. Les started his agricultural career on his family's dairy and beef operation, co-managing 125 head dairy and beef herd. He has now integrated 250 honey bee colonies to this family business, which focuses on honey bee genetics and breeding. Les's educational background includes both a Diploma in Agriculture from the Ontario Agricultural College and a Bachelor of Science in Agriculture and research at the University of Guelph Apiculture Research Centre from the University of Guelph. Les has been instrumental in various research projects, workshops as well as provincial and national strategies including his role as Vice President of the Canadian Association of Professional Apiculturists.

**Paul Kozak**

**Provincial Apiarist, the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA)**

Paul Kozak is the Provincial Apiarist of Ontario and Apiary Specialist at OMAFRA. Paul works with beekeepers across Ontario in a regulatory (apiary inspection and provincial registration of beekeepers) and advisory capacity. Paul develops strategies and collaborates with other apiculture programs across Canada and the United States.

**Andrew Morris, MD, SM(Epi), FRCPC, Professor**

***Medical Director, Antimicrobial Stewardship Program, Sinai Health System & UHN***

Dr. Morris is a Professor of Medicine at the University of Toronto and the Director of the Sinai Health System – University Health Network Antimicrobial Stewardship Program. He is currently Chair of the Antimicrobial Stewardship and Resistance Committee for the Association of Medical Microbiology and Infectious Disease Canada (AMMI Canada) and is a member of the parallel committee with the Society for Hospital Epidemiology of America (SHEA). He was appointed to the Canadian Government's Expert Advisory Group on Antimicrobial Resistance (EAGAR) in 2015, serves as a member of the Technical Expert Panel on Antimicrobial Stewardship for the Agency for Health, Research and Quality in the United States, and was the Principal Investigator of two peer-reviewed grants from the Council of Academic Hospitals of Ontario, responsible for implementing antimicrobial stewardship programs in academic and subsequently community hospitals.

Dr. Morris has worked closely with regional, provincial, and federal governments and interprovincial organizations to help develop and coordinate antimicrobial stewardship efforts. Dr. Morris recently stepped down as Chair of the Specialty Committee of Infectious Diseases with the Royal College of Physicians and Surgeons of Canada but is an advisor to the Royal College on Resource Stewardship.

Dr. Morris obtained his medical degree from the University of Toronto, where he subsequently completed sub specialty training in Infectious Diseases. He went on to complete a Master of Science degree in Epidemiology from the Harvard School of Public Health, while completing a Canadian Infectious Diseases Society (now AMMI Canada) research fellowship. He often says that his primary job is coaching basketball, which he started doing 30 years ago.

**Scott Weese, DVM, DVSc, DACVIM**

***Professor, Ontario Veterinary College***

Dr. Weese is a veterinary internist and microbiologist, and a Diplomate of the American College of Veterinary Internal Medicine. He is a Professor at the Ontario Veterinary College, University of Guelph. He is also Chief of Infection Control at the Ontario Veterinary College Teaching Hospital and holds a Canada Research Chair in zoonotic diseases. He has authored or co-authored over 300 papers in peer reviewed journals, edited two books and speaks extensively on infectious disease topics.

## EQUINE:

**Janet Christine Beeler-Marfisi, DVM, DVSc, DACVP (Clinical Pathology)**

*Assistant Professor, University of Guelph, Ontario Veterinary College, Department of Pathobiology*

Janet Beeler-Marfisi, a Diplomate of the American College of Veterinary Pathologists, teaches clinical pathology and shares diagnostic service for the OVC's Large and Small Animal Health Sciences Centre. Her current research is focused on mild and severe asthma (inflammatory airway disease and heaves) to determine whether, similar to people, there is a cause and effect relationship between air pollution and asthma. Ultimately this research will help improve athletic and earnings potential of equine athletes, as trainers could limit high-intensity training on days when air pollution is high, to help their horses avoid developing asthma.

In her spare time, Janet enjoys developing and sharing clinically-relevant continuing professional development materials with her colleagues out in the field.

**Patty Hogan, VMD, DACVS**

*Owner, Hogan Equine*

Dr. Hogan has turned a lifetime love for horses into a successful career as an equine surgeon attending to some of the most valuable Standardbred and Thoroughbred racehorses in the country. Dr. Hogan is originally from New Jersey and obtained her veterinary education at the University of Pennsylvania, graduating in 1992. She is also a diplomate of the American College of Veterinary surgeons. After an internship at the Rood & Riddle Equine Hospital and a 3-year surgical residency at Texas A&M University, Dr Hogan returned home to New Jersey to practice. Dr. Hogan is the rare type of equine surgeon who is equally proficient in both orthopedic and soft tissue surgery disciplines. The majority of her patients are referred for fracture repair, arthroscopy, or upper airway surgery. Although the bulk of her caseload consists of Thoroughbred and Standardbred racehorses, some of her most memorable patients have never set foot on a racetrack and are just as valuable. Dr. Hogan's background may be in racing, but her dedication to her patients is universal.

**Steve Reed, DVM, DACVIM**

*Internal Medicine Specialist, Rood and Riddle Equine Hospital, Lexington, Kentucky*

Stephen M. Reed, earned his degree at The Ohio State University before completing a residency at Michigan State University. After a period at Washington State University, he returned to Ohio State University, retiring from there as a professor emeritus in 2007. At the same time, Dr. Reed chose a new career path away from academia, and is now a shareholder and member of the Internal Medicine Service at Rood and Riddle Equine Hospital in Lexington, Ky.

Dr. Reed is widely recognized for his commitment to the horse, equine veterinarians and the equine industry. A diplomate of the American College of Veterinary Internal Medicine, he developed an interest in the specialty of equine neurology and became recognized for his work in this area. In addition, Dr. Reed has authored or co-authored more than 150 peer reviewed publications.

Dr. Reed considers the opportunity to assist in the mentoring of thirty internal medicine residents, more than twenty interns and hundreds of veterinary students as his most significant contribution to the veterinary profession. At this time, the most enjoyable part of his life is spent with family, including three grandchildren.

## PRACTICE MANAGEMENT:

**Sarah Bernardi, MA**

*Registered Social Worker, Veterinary Emergency Clinic and Referral Centre*

Sarah is part of Toronto's Veterinary Emergency Clinic and Referral Centre's (VEC) professional team. As their first Social Worker on staff, she strives to connect her knowledge of the Social Work profession with the specialty services offered at VEC. Sarah is originally from Northern Ontario where she has grown up with a variety of animals, including several rescue dogs. She is passionate about helping all diverse populations facing adversity and is currently volunteering with Link Coalition Toronto, a Not-for-Profit addressing the link between domestic violence, child abuse, animal abuse and elder abuse in the GTA. Sarah holds her Masters of Social Work Degree, with Distinction in Gerontology from the Factor-Inwentash Faculty of Social Work, University of Toronto.

**Eric Garcia**, Information Management

**CEO, *Simply Done Tech Solutions***

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. Eric is an active advisory board member with the American Association of Feline Practitioners and marketing columnist for Today's Veterinary Business, an NAVC publication. He speaks regularly at conferences throughout the world.

**Dave Nicol**, BVMS Cert Mgmt MRCVS

**Director, *The VetX Graduate Community***

Dr. Dave Nicol is a marketing, performance management and leadership expert. He has written three books, most recently "So You're a Vet...Now What?", has authored hundreds of articles and hosts the Blunt Dissection Podcast, a monthly conversation about success with the rock starts of veterinary medicine (which receives more than 60,000 downloads per year). Dr. Dave is also the founder of VetX Graduate, an independent teaching, mentoring and networking community helping young veterinarians around the world thrive in practice, not just survive by teaching essential non-clinical skills.

In his spare time, he helps to run Roundwood Vets, a boutique, independent veterinary practice in London. You can access all of his content (the majority of which is free) and get in touch at [www.drhavenicol.com](http://www.drhavenicol.com) or follow his life in photos and video on facebook or Instagram @drhavenicol.

**Jayne Takahashi**, DVM, MBA

**Owner, *Communication Leads***

Dr. Jayne Takahashi loves all things communication! Throughout her diverse career, professional communication skills have remained her focus and her passion. Jayne has held a number of positions including companion animal veterinarian, pet nutrition educator, national marketing manager in the pet food industry, Vice-President of Communications for a network of veterinary practices and currently, communication consultant through her company, Communication Leads. In each of these roles, her focus is educating, coaching and training individuals and teams on the power of communication skills with clients and colleagues.

Jayne received her Doctor of Veterinary Medicine degree from the Western College of Veterinary Medicine at the University of Saskatchewan and a Master of Business Administration degree from the University of Calgary. She has written a number of published columns on communication within the veterinary practice and speaks nationally at conferences and training events.