

## **Up & Away – The Only Way to Schmear**

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### **How to Prep – Our Patients**

While we're here to focus on the blood smear, we can't make them without our patient, so we do need to spend a bit of time on getting them - and us - prepped.

**Signalment** – this is your species/breed/sex/and age. This is simple, but important. Because as you know cats are not small dogs, and Chihuahuas are certainly not Great Danes! Having this bit of information will allow you to be prepared on how to approach that patient and you can communicate that to the rest of your team.. Not only that, but there are several disease processes that are breed specific, So if we know a bulldog is coming in, we can change our restraint approach. So, it's always a good idea get your "nerd books" out and jot some notes prior to these appointments.

**Approach/Restraint** – We'll keep this short and sweet; the least stressful for our patients is best, but we also must keep ourselves and our teammates safe. Proper restraint techniques and tools – blankets, muzzles, pre-meds & sedatives. This aids in the process. , Recognizing signs of fear and stress in our patients as well as when our teammates are tapping out must be communicated. Yes we need our samples, but at what cost? It's always good to have a calm, cool & collected approach. Not only will this help you , but it will help your patients and they should follow your lead.

**Presenting Complaint** – is the pet limping? Are they lethargic? V+/D+? Routine exam? Chronic illness? Acute onset? Pre-op? Knowing what our patients are coming in for is not as concrete as you may think. From the time the owner schedules the appointment, till they get the confirmation call – things may have altered. On the day of the appointment , what they tell the CSR is different from what they tell us as nurses/techs, and it's rare that the stories match from what's relayed to our docs at the end. It's sometimes like playing the worst round of "Telephone"/"Whisper Down the Lane" and it's critical that we document EVERYTHING. Because if you don't write it down, it didn't happen. Taking what an owner is saying into account versus what we physically see with the patient will aid in getting the full picture, and who doesn't like a nice clean SOAP?

**History** – All joking aside our next point is another vital piece of the puzzle. If the patient is currently in remission from LSA, a lifelong diabetic, a hyperthyroid kitty, a bouncing pre-op TPLO pup, or a Cushing's canine – all of this must be taken into consideration. Blood smears may/may

not be a part of every practice's screening protocols, whether pre-op or routine testing, but then again, they may be. Understanding these conditions should get us thinking and could alter what diagnostics we need for that patient.

### **How to Prep – Ourselves**

As you can see, this list is fairly comprehensive, but if you don't see something included that is utilized in your practice don't stress, this is simply a framework of recommendations. Let's go through it point by point.

Gloves:

- Love `em or hate `em, ya gotta wear `em
- Try nitrile, no residue, no latex & decent fit
- Keep yourself & your patient safe; zoonosis is not a laughing matter

Needles:

- Nothing smaller than a 22g, and nothing larger than an 18g – for small animals.
- Reason? Too big and we can cause unnecessary trauma to the vessel and too small we risk lysing our RBC's and skewing the results

Syringes:

- Depends on our patient, vessel & sample size
- 1-12mL again in SA practice
- Why? Don't be greedy, but don't shortchange yourself either
  - A geriatric feline patient's veins will collapse if a larger volume syringe is utilized, and if we're concerned with conserving hemostasis, don't take more than is necessary

Butterfly catheter:

- These are wonderful for saphenous sticks, particularly the distal medial aspect on a fractious cat
- Practice using these as muscle memory takes a while to master
  - Tip: allow blood to flow the length of the tube until ~0.5" before hub before attaching syringe to ensure adequate pressure & ability to withdraw

Clippers:

- May/may not need these, and always make sure to confirm with the owner prior to doing so. We can be the best phlebotomist, at a top-notch practice and if we shave a show poodle incorrectly – GAME OVER.
- However, if the patient is less than hygienic, matted, or there is only one shot at this and you need visualization that vessel, clip away: with permission

#### Alcohol:

- Spray bottle, squeeze bottle, pre-soaked gauze, or cotton balls – any/all are viable options.
- This is not to disinfect or even clean the patient, although it helps a bit. However, it must be allowed to fully evaporate/dry to enable disinfection and our window for sampling from a less than cooperative patient is usually much slimmer than this
- Alcohol is applied for 2 main reasons:
  - 1) Wet fur & improve visibility
  - 2) Contraction of superficial vasculature to allow easier visualization/location of the vessel – makes it 'pop'

#### 4x4's:

- To dry the patient, but typically to create a compression band-aid with the VetWrap
- If you send your patient home with these Band-Aids, it's important communicated to the owner to remove in 1-2 hours

#### LTT:

- Lavender Top Tubes are our go-to for any samples that a smear may need to be performed
- EDTA is the anti-coagulant here and it's been shown not to interact with heparin that so many of our HCT tubes are coated with
  - Also, it's the most stable and does not need to be refrigerated for up to 4 hours; this is NOT the case for analysis of other values, but the RBC, WBC & platelet counts will remain unaffected at room temp

#### HCT tubes:

- At least three please & thank you; two for spinning and an extra in case it breaks
- Further along we'll discuss why utilizing HCT for making the schmear is preferable to a needle
- If utilizing for PCV/TS place the sample DIRECTLY into the HCT tube
- If utilizing for smears, can place directly into the LTT or the HCTs

#### Slides:

- You'll need at least 6 – 2 slides per schmear, and it's always better to have more than you think you'll need
- Preferably with a frosted edge AND that the slides are faceup
  - Upside down slides will absolutely impact the quality of not only the schmear but our ability to analyze and interpret the cellularity

## Sampling

OK, our patient is ready and so's our team. Now, do we have everything we need? This may be 2<sup>nd</sup> nature for some of us, but there is nothing worse than going for that juicy vein only to realize you're missing something. And let's be honest, we're a superstitious lot, so having an extra of everything is essential!

Before you poke it, you've gotta locate it. These vessels are housed in a live patient. They don't sit & stay for said poking. Knowing what to poke, where it is, and how to access it is what we'll cover here.

2H's to avoid – think of yourself as the lifeguard, protecting the sample & your patient.

Ready? Let's dive in!

Vasculature- most common places for venipuncture are the jug, saphenous, and cephalic veins.

Jugular:

- Unless our patient is extremely anemic, coagulopathic and/or uncooperative this should be the go-to vessel for sampling. You should always communicate with the attending vet if there are any concerns about using the jug for venipuncture. Saphenous vein should be your second choice. These vessels are easily accessible and can be less stressful for the patient. Cephalic veins should be your last choice (especially if in ER, specialty, or preop patient) because this is the vessel of choice for an IV catheter.
- Techniques will differ from practice to practice as well as on an individual basis
- Take these recommendations into consideration:
  - Sternal or lateral, recumbency
  - Assistant/teammate to hold off the vessel (jugular hold off in the thoracic inlet)
  - Gently palpate the vessel prior to venipuncture. Some vessels are hard to visualize due to fur or if your patient is overweight. Brachycephalic patients are usually hard to visualize.
  - Apply alcohol
    - Avoid spray/squeeze bottles with feline patients
  - Insert needle, bevel up, at a 45° angle in one, smooth, fluid motion until a flash of blood appears in the needle hub
    - Do NOT insert the needle entirely
    - Only 1-2 mm at a time
    - Redirect only if necessary and when doing so make micro-adjustments, 1-2 mm/time
    - Do NOT fully remove the needle & reinsert
    - Change the needle and/or needle & syringe if needed
    - No more than 2 attempts per team member

- Maintain stability of the barrel of the syringe with your middle and index fingers
- In a steady motion, draw back on the plunger of the syringe with either your ring or pinky finger, not too hard can collapse the vein
- Once sample is obtained, quickly & smoothly remove the needle & syringe in one motion
- Maintain hemostasis with digital pressure and a 4x4 gauze square
- Transfer sample to teammate or directly into LTT or HCT

Moving on to the finer details of sampling technique - the two H's – **Hemolysis** is up first, and it's exactly what we're looking to prevent. Good ol' vet med, taking a simple concept and making go all Greek!

This is the breaking or rupturing of blood cells and it can happen in one of two ways – in our patients (in vivo) or by us either intra- or post-sampling (in vitro). We've all seen this happen when we spin a RTT and the serum is red tinged, yes we broke the cells.

How can we prevent this? We can't do anything about the in vivo component, but we can absolutely control our part:

Good stick:

- Clean, smooth, fluid motion with no re-directs.
- Avoids unnecessary trauma to the vessel

Proper needles:

- Remember, nothing smaller than 22g, and if you have a larger patient with a larger vessel, use a larger gauge needle
- Keep in mind though, this isn't a time for 'go big or go home,' as the larger the bore, the larger the hole it leaves
- We typically stick with 22-20g needles for a happy medium

Tidy Transfer:

- Directly into the LTT via a vacutainer butterfly – ensure it's a 22/20g
- Change the sampling needle to a new sterile 20g and allow the vacuum to draw the sample in when puncturing the LTT top
- You can take the LTT lid off, but there is a potential for:
  - Contamination
  - Spilling the sample

The 2<sup>nd</sup> H is **hemostasis** – maintaining vascular stability – and this is equally important, especially for those patients that are getting repeated samples drawn. This is your hospitalized patient – consider a sampling line (different webinar, but I digress) – or one that is coming for routine checks i.e., the oncology patient for its weekly treatments and pre-chemo screenings. Again, stay safe & stay small – don't go big or go home here. Keep the needle sizes down & rotate sampling vessels, document this so everyone knows which vein was sampled, when, & by

whom. Another factor here is the prevention of hematomas. Immediate & consistent digital pressure followed by a hemostatic pressure band-aid – 1 gauze square with vet wrap – triggers the body to start the clotting cascade. Excellent phlebotomy technique will aid in this. We have to remember good sampling keeps the vasculature stable & less trauma leads to less bruising, and a happier patient = happy owners, everybody wins!

### **It's All in the Wrist**

- Place sample onto flat slide
  - Using an HCT:
    - Small drop of blood
    - Close to the frosted edge
- Second slide
  - "Spreader" slide
  - Non-frosted end
  - 45° angle
  - Draw the slide top à down
  - Allow sample to spread
    - Capillary action
- Push!
  - Rapidly, smoothly
  - Up & away
  - One fluid motion
- Create feathered edge

### **How to Prep – The Slide**

It's all about the airtime! After successfully making the smear, follow these steps:

- Allow slide to fully dry
  - ~ 5 minutes
  - Hair dryer on COOL
- Staining
  - Using Diff-Quick stain:
    - Dip sequentially
      - Blue, pink, purple
    - 10 times in EACH
      - For 1 second
    - Tap off excess
  - Water rinse –after your final stain
    - Distilled or tap
    - Steady, gentle stream
    - AFTER final stain
- Air-dry Part II
  - Allow stained slide to dry

- ~5 minutes
- Hair dryer on COOL

## Interpretation

What are we interpreting when we look at a smear?

- Cell count
  - WBC, platelets, RBC
- Diseases
  - Blood borne
- Coloration
  - Polychromasia
- Artifact
  - Stain precipitate
  - Poor smear technique
- Morphology
  - Schistocytes, Rouleaux
  - Adds great value
  - Detects unsuspected disorders
    - Ex: *Babesia*

## Why Do Good Schmears Matter?

To aid in the diagnosis of emergent disease processes, confirm the presence of microorganisms, determine the course of interventional therapy, and/or prevent unnecessary procedures from occurring.

Examples:

- ER – SIRS, sepsis, hemoabdomen
- In-House equipment failure – CBC analyzer, flags
- Chronic – IMHA, ITP, Renal
- Onco – Cell counts, sick LSA
- SX – confirmation, intervention

## Case(s) in Point

**Immune Mediated Hemolytic Anemia** – when the immune system reacts against its own RBCs just like it would to any other invader (bacteria, foreign substance). When this process is occurring, the RBCs are destroyed, the patient becomes anemia, and oxygen delivery is drastically reduced.

As you can see the RBCs are markedly decreased. This patient is going to need hospitalization and a pRBC (or whole blood if Packed RBCs are not available) transfusion with potential oxygen supplementation, stat!

A microscopic parasite transmitted by multiple tick species, infects RBC's causing **babesiosis**, and often occurs simultaneously with Lyme disease. Patients tend to present with general malaise, intermittent & shifting limb lameness, fever, and/or GI symptoms.

It's preferred to utilize either Wright or Giemsa stain, but DiffQuick will also show on a blood smear the distinct pleomorphic rings (vacuolated & do not produce pigment) noted by the black arrows and occasional classic tetrad-forms known as the Maltese Cross, circled here in blue. Extracellular rings may also be present (red arrows), and it's worth noting that infected RBCs tend not to be enlarged but will have an increased central pallor.

Take a moment and think about what medication therapy would be indicated for these patients...that's right, doxycycline. Ensure that your patient is not sensitive to it and address any concurrent symptoms. Sometimes these patients can experience nausea.

Heartworm is a serious disease that results in severe lung disease, heart failure, other organ damage, and death in pets, dogs, cats, and ferrets. It is caused by a parasitic worm called *Dirofilaria immitis*. The worms are spread through the bite of a mosquito. As you can see *Microfilariae* are visible in a blood smear.

**Idiopathic thrombocytopenic purpura** or immune thrombocytopenic purpura. This condition makes it even more clear as to why acronyms are so helpful in our field!

ITP occurs for various unknown reasons. This happens when a patient's immune system randomly mistakes its own platelets as foreign. This can be either a chronic or acute condition, and is impacted by species, breed, and age. ITP may occur following a viral infection, ingestion of a toxin, and symptoms typically include petechia (pin prick) and/or ecchymosis (sm.-lg. bruising) along the extremities, MM's, or ventral abdomen.

In the bottom slide you can see, there are no platelets.- most platelets are smaller (pin head), more purplish cells as seen in this top slide – the normal range are 10-15 platelets/HPF. This example has 0 platelets/HPF and as this patient's nurse I am looking for hematochezia, melena, petechia, or ecchymosis.

When ITP is suspected, hemostasis MUST be a top priority. This doesn't mean you cannot utilize the jugular vein, but it shouldn't be your go-to vessel, especially if an accompanying chemistry screen shows elevated LE's and/or your patient is also presenting with icterus! Use smaller peripheral vessels. Try to avoid the cephalic, save it for your IVC's! – and a good pressure band-aid post sampling are recommended here.

## **Spinal Localization Parts 1 and 2:**

**By Carrie Journey DVM DACVIM (Neurology)**

### **Introduction**

The goal of this lecture is to take a deep dive into spinal localization, including relevant exam findings and neuroanatomy.

There are many reasons that patients present for AWR (“Ain’t Walkin Right”). A general physical, orthopedic exam and neurologic exam are critical parts of any consult for AWR. A variety of conditions can be discovered in the course of this examination including: neurologic disease, orthopedic conditions (i.e. cruciate ligament disease, bone tumors), metabolic conditions (i.e. hemoabdomens, shock, hypoxia), pain (i.e. anal gland disease, candy stuck between toes) and behavioral conditions (these more likely to present as a brain disease than a spinal disease). In this lecture, we’ll be focusing on the neurologic, and specifically spinal, causes of AWR, but it’s important that the practitioner keep this full differential list in mind when presented with a patient with AWR.

### **Relevant Neuroanatomy and Pathology**

#### **Spinal Segments vs. Vertebral Segments**

It is important for the clinician to recognize that when we are discussing segments of the nervous system, we are naming them by their spinal segments. There are 8 cervical spinal segments and nerves, but only 7 cervical vertebrae. These spinal segments are of variable length and are named for the spinal nerve they create. These segments sit close to but not exactly near the vertebrae of the same name. This is important clinically when, for instance, assessing if clinical signs match radiographic findings. For example, a L3 vertebral fracture can injure the L4 segment. There for an injury in the “T3-L3” vertebral segments, may present as an L4-S1 spinal segment injury.

#### **Motor Neurons**

Upper Motor Neurons (UMN) are neurons located in the brain that are responsible for the generation of voluntary movement and maintenance of normal resting tone. Clinically, we see disturbance of this system manifest in loss of voluntary movements, as well as, hypertonia/reflexia due to loss of descending inhibition. While in day to day localization, one need not focus on specific UMN system, it’s interesting to note that the Corticospinal and Rubrospinal tracts are facilitatory to flexors and the Reticulospinal and Vestibulospinal tracts are facilitatory to extensors.

Lower Motor Neurons (LMN) are neurons with cell bodies in the ventral horn of the spinal cord. The LMN for the limbs are located in the intumescences (C6-T2 & L4-S1 spinal segments)

#### **Function Concepts: Motor systems**

To achieve voluntary movement the UMN sends signals to the LMN who in turn causes muscle contraction. You need both a functional UMN and LMN system for voluntary motor to work. In general, a LMN lesion will show more weakness than a UMN lesion. A UMN lesion will show relatively more ataxia.

Important terms to know include paresis, which is a partial loss of voluntary motor, and plegia which denotes a complete loss of voluntary motor. The deficit is further characterized by which limbs are involved:

- 1) Mono: one limb
- 2) Para: the hind limbs
- 3) Hemi: limbs on one side of the body
- 4) Tetra: all four limbs

So for instance a dog that has a complete loss of motor in the hindlimbs is “paraplegic”, versus a dog who has a partial loss of voluntary motor in the right fore and hindlimb is “hemiparetic.” For paretic animals, I find it also helpful to specify if an animal is “ambulatory” or “non-ambulatory”.

Another important concept in motor systems is resting tone. The UMN sends signals, the majority of which are inhibitory, to the LMN to maintain a normal resting tone (called descending inhibition). When this connection is lost, the overall activity of the LMN is increased, which results in hyperreflexia and hypertonia.

### **Functional Concepts: Proprioception**

Proprioception is the sensory system that informs the brain of the limb/joint position. This helps maintain limbs under the center of gravity. These pathways send projections to both the cerebellar and cortical parts of the brain, which results in the terms “unconscious” and “conscious” proprioception, respectively. However, it is difficult clinically to differentiate between “unconscious” (aka cerebellar) and “conscious” (aka cortical) proprioception. While people commonly refer to the testing of this system as “CPs” for conscious proprioception, this is a bit of a misnomer as it reflects both the conscious and unconscious systems.

These nerves are big floppy fibers that are affected early in compression. With lateral compression, pelvic limbs are affected first. This might fool you on an exam of a lateralized cervical lesion- can look T3-L3 early on!

There are many ways to test proprioception. If deficits are subtle, test the system with multiple modalities! Knuckling, single leg hopping, wheelbarrowing, extensor postural thrust (,) as well as (,) tactile and visual placing can all be used to test proprioception.

### **Functional Concepts: Nociception**

Nociception, or the system that allows us to feel pain, is a very important and robust system. In domestic animals, this is a bilateral, highly redundant system. It takes a lot of damage to a spinal cord for this not to work.

The order of disease progression in compressive spinal disease is: pain, ataxia, loss of motor, loss of nociception. Initially, patients will appear painful. However, due to a variety of pain tolerances and other factors, not all patients show pain. With further compression, ataxia then starts. Most often this will be proprioceptive ataxia, although we will discuss notable exceptions later in the lecture. As the compression continues, patients will lose voluntary motor function. This is a sliding scale that starts with ambulatory paresis down to paraplegia as things become more severe. Finally, with severe compression, patients will lose sensation (aka no deep pain).

Testing sensation is the single most important test to do to determine a dog's functional prognosis. However, it is the test that is most often done incorrectly. In your average spinal patient, testing for deep pain should only be performed when a patient has **no voluntary motor**. To test for the absence of nociception start with a light pinch with your fingers. If they respond (look at you/the foot, growl, cry) then stop here. They have sensation. If they do not respond to that, pinch harder- get a hemostat, pinch across the toenail bed. It is critical that you are absolutely sure of this result. This may require you to be mean, but you need to be sure. If you are concerned the animal is just on pain meds or overly stoic- pinch something that you know is working (like a front foot on a T3-L3 dog).

A withdrawal of the foot is **NEVER** a positive response on this foot as it only indicates the reflex. The dog needs to be obviously conscious that a response has occurred, and frankly they should be fairly upset with you. If there is no deep pain, pinch along the dog, moving one centimeter up the leg and then up the back until a "line of analgesia" is reached (i.e. where they get mad at you). Draw on the dog with a sharpie to indicate this line.

### **Function Concept: Sensory systems**

Stimulation from the periphery of the body is carried in to the nervous system via the nerves in the dorsal root ganglion. Fibers either synapse locally to cause reflexes, or ascend to inform the brain about what's happening.

### **Functional Concept: Reflex Arcs**

Reflex arcs are a functional concept that put together the clinical concepts of sensory and motor systems, and more importantly they give us important clinical information.

Pathway of a reflex arc:

Stimulus -> Sensory Nerve to Spinal Cord -> Synapse on the LMN in the Spinal Cord -> Motor Nerve to Muscle -> Muscle Contraction

When we lose descending inhibition (the UMN telling the LMN to calm down), the reflexes become exaggerated. Conversely, hyporeflexia occurs when we lose the resting tone and effect of the LMN, then the reflexes become weaker.

### **Functional Concepts: Gait Analysis**

When analyzing a neurologic gait there are three components: Level of Paresis, Stride Quality & Length and Ataxia.

)When assessing level of paresis ask yourself: which legs are affected and can the animal walk?

Then look at the stride. When the UMN connection is the primary disruption, the strides are long. Conversely, when the LMN are affected the strides are often short and choppy. Then finally pay attention to the pattern of the foot placement. A disruption in pattern is ataxia. There are three types of ataxia: Proprioceptive, Cerebellar and Vestibular. Proprioceptive is the most common in spinal lesions. In this form of ataxia the feet cross midline, knuckle/scuff, and limbs are held in positions not under the center of

gravity. Cerebellar ataxia results in dysmetria/hypermtria, truncal sway, as well as tremors. Finally, there is vestibular ataxia in which patients exhibit leaning, falling, rolling and circling.

Putting it all together you should be able to come up with an excellent description of how your patient is walking such as: "Ambulatory paraparesis with proprioceptive ataxia and short strides in rear."

### **Functional Concepts: Cutaneous Trunci**

The cutaneous trunci reflex can be helpful for further narrowing your localization. Much like any reflex, it has an afferent sensory pathway and an efferent motor pathway. The sensory stimulation on the skin travels 1 to 3 spaces cranial to enter the spinal cord. From here there is cross over and the signal ascends to the level of the LMN for the cutaneous trunci reflex (specifically, the lateral thoracic nerve, which arises from C7-T1). The signal synapses here and stimulates a reflexive motor movement by the LMN which results in a twitch of the muscle. This reflex can be difficult to consistently get, but can help greatly with localization if you find a cut off or asymmetric response. In many animals, the reflex cannot be elicited from skin stimulation caudal to L4.

### **Functional Concepts: Respiration**

Neurologically speaking, there are two important motor systems involved in breathing: the diaphragm and the intercostals. Each of these systems has an upper motor neuron in the brain that drives the LMN. The LMN for the diaphragm is the phrenic nerve, which arises from the C3-6 spinal segments. The LMN for the intercostals arise from thoracic spinal segments. Disruptions in either the UMN tract or to either of these LMN systems cause loss of voluntary motor to the muscles of respiration, resulting in respiratory paralysis and neurogenic hypoventilation. Clinically this will be recognized by paradoxical respiration (diaphragm paralysis) in which the abdominal contents appear to contract on inspiration (normally they expand), or by abdominal breathing in which the chest wall does not move but the abdomen does. Animals will be in respiratory distress (often appear to be "fish breathing"), with elevated respiratory rates and increase blood CO<sub>2</sub> levels. Mechanical ventilation can be required in severe cases.

### **Functional Concepts: Bladder Anatomy**

Bladder control is achieved with a beautiful symphony of sensory systems, autonomic control (sympathetic and parasympathetic) as well as conscious control via the pudendal nerve to external urethral sphincter. It's a complicated system, however the practicalities of clinical bladders are that neurologic incontinence is defined by the action on the pudendal nerve- the LMN for the external urethral sphincter. An UMN bladder has a hypertonic sphincter and we have a full bladder that is difficult to express.

A LMN bladder has a hypotonic sphincter, the bladder dribbles urine constantly, and the bladder is easy to express.

It's interesting and frustrating to note that nearly all existing medications to deal with incontinence deal with the autonomic system, which is why these medications provide less than ideal results.

### **Neurolocalization**

In the final part of lecture 1 and for all of lecture 2, we will be focusing on localization of each spinal segment.

The cornerstone of any good neurologic differential is correct localization. And much like any physical exam, it's important to do a complete exam the same way every time to maximize your diagnostic potential. Clinically you may not recognize every single feature of a localization that we discuss here, but it's important to pick the one that fits best.

A full neurologic exam consists of the following:

- **Mentation:** This is a subjective finding. I find it best to limit responses here to a small list of words. Normal, Dull, Obtunded, Stupor and Coma are my preferred terms.
- **Posture:** This is where we look for things like head tilts and turns, as well as tremors.
- **Gait:** This is where we put all of that hard work we just did on learning about paresis/plegia, ataxia and stride length to work. Also the place to denote circling/leaning/falling or non-neurologic gait disturbances (like orthopedic lameness). Be descriptive!
- **Cranial Nerves:** A full cranial nerve exam goes here.
- **Proprioception:** List how you tested and which, if any, limbs are abnormal.
- **Spinal Reflexes and Tone:** Is the patient hyperreflexive? Hyporeflexive? Hypertonic? Hypotonic? List here any disturbances in reflexes. Be sure to list the affected limb as well as the finding.

## C1-C5

Due to its proximity to the caudal brainstem, it's possible to see some disturbances in the vestibular system with this segment.

- **Mentation:** Normal
- **Posture:**
  - Often Head and Neck held in a neutral position
  - Rarely head tilt with vestibular tract damage
  - Rarely curvature of the neck, especially with sensory damage (Caudal Occipital Malformation Syndrome dogs will have this due to the syringomyelia in the cervical spine)
- **Gait:**
  - Long strided "floating" gait in all four limbs
  - Ataxia: Proprioceptive ataxia (rarely vestibular or cerebellar with cranial lesions)
  - Weakness: Tetraparesis/plegia
- **Cranial Nerves:** Can have Horner's Syndrome, can be vestibular (rare)
- **Proprioception:** Deficits x 4
- **Spinal Reflexes and Tone:** Hyperreflexia, Hypertonia in all four limbs
- **Breathing:** Hypoventilation Possible
- **Cutaneous Trunci Reflex:** Can be absent if a caudal lesion
- **Urination/Rectal tone:** UMN bladder (clinically rare). Rectal tone is normal (technically increased, but this is not something we clinically realize)

## C6-T2

- **Mentation:** Normal

- Posture: Often Head and Neck held in a neutral position
- Gait:
  - Stride Length: “Two Engine Gait” Short strides in fore, long strided in back.
  - Ataxia: Proprioceptive ataxia
  - Weakness: Tetraparesis. Should be more weak than ataxic in forelimbs.
- Cranial Nerves: Can have Horner’s
- Proprioception: Deficits x 4
- Spinal Reflexes and Tone: Hyporeflexia, Hypotonia
- Breathing: Hypoventilation Possible
- Cutaneous Trunci Reflex: Can be absent if a caudal lesion
- Urination/Rectal Tone: UMN bladder (clinically rare), Normal rectal tone

### **Cervical Tips & Tricks**

Patients with cervical pain often present for screaming with no known reason. They also occasionally present as seizures, inappetence or ear problems.

It is challenging to localize correctly between C1-5 and C6-T2. Forterre, et al showed that two boarded neurologists got this localization right only 65.8% of the time!<sup>1</sup>

To find cervical pain, I start with a gentle, then firmer, palpation laterally of the bones of the spine. Remember that the spine is actually under quite a bit of muscles, so you are going to be palpating the middle of the neck. After this, perform a full range of motion. Bring the nose to each shoulder, then nose to chest (push in and up slightly) and finally pull the nose back so it is pointed in the air. I call this “dog yoga” to make my clients more comfortable. Use caution with ventroflexion in toy breeds or juvenile patients- if they have an AA lux it can dramatically worsen their symptoms. If you still can’t find it feel for the transverse process of C6 and rock it gently to isolate the caudal neck. You can also do the same for the wing of C2 in the cranial cervical spine.

### **T3-L3**

- Mentation: Normal
- Posture: Maybe hunched through mid-back
- Gait:
  - Long strided in the rear
  - Ataxia: Proprioceptive ataxia
  - Weakness: Paraparesis/plegia. Should be more ataxic than weak.
- Cranial Nerves: Normal
- Proprioception: Deficits in rear
- Spinal Reflexes and Tone: Forelimbs normal, Hyperreflexia/Hypertonia in rear
- Breathing:
  - Normal
  - With diffuse lesions may see paradoxical respiration (breathing only with diaphragm).
- Cutaneous Trunci Reflex: Can be absent if a caudal lesion
- Urination, Rectal Tone:

- UMN bladder (clinically common)
- Normal rectal tone
- Can see delayed notification incontinence

### **T3-L3 Tips & Tricks**

In acute spinal trauma reflexes can be transiently be hyporeflexive, this is referred to as spinal shock. It is due to a loss of descending facilitation from the UMN. We talk a lot about descending inhibition, but remember from our discussion of UMNs, that there is a balance of things that facilitate and things that inhibit. In the acute stage, sometimes we clinically note the loss of descending facilitation more than the loss of descending inhibition. Eventually the LMN resets itself to its “natural” unfettered state, which is hyperreflexive with respect to a normal animal. This process takes about 24 to 48 hours.<sup>2</sup>

Another severe injury presentation is Schiff Sherrington posture. This is a posture in severe T3-L3 disease where the front limbs are hypertonic. This can be confusing as it looks like C1-5 disease. However, when tested the proprioception in the forelimbs is normal. The presentation is due to a loss of ascending inhibition from border cells. These cells live from L1-5 and normally are part of gait pattern generators in the spine. This posture is in and of itself not prognostic. However, practically it is often in animal with severe lesions. The absence of nociception (loss of deep pain) is often found on exam of these animals- but the finding of loss of nociception and not the Schiff Sherrington posture is what affords prognostic information (generally guarded to poor).

There is a less common form of fecal incontinence that is due to a sensory problem in T3-L3 disease. This presents as a delayed notification incontinence- the animal is aware that it needs to defecate, and often attempts to go to the normal location. However, they often do not make it in time (i.e. ask to go outside, then defecate by the door). They will posture to defecate normally and have normal anal tone.<sup>3</sup>

T3-L3 animals can present for abdominal pain (without secondary GI signs). Support under the chest and push on the dorsal spinal processes, then palpate laterally to differential TL pain from cranial abdominal pain.

### **L4-S1**

- Mentation: Normal
  - Posture: hunched through lower spine (tail tucked under)
- Gait:
  - Short strided in rear
  - Ataxia: Proprioceptive ataxia in rear
  - Weakness: Paraparesis. Should be more weak than ataxic.
- Cranial Nerves: Normal
- Proprioception: Deficits in rear limbs
- Spinal Reflexes and Tone: Hyporeflexia, hypotonia
- Breathing: Normal
- Cutaneous Trunci Reflex: Normal
- Urination: LMN bladder
- Decreased anal tone

## **L4-S1 Tips & Tricks**

Make sure to test the patellars AND the withdrawal. If the patellars seem hyper, but there is decreased withdrawal (especially through the hock) this indicates pseudo-hyperreflexia. In this finding, the effector muscles of the patellar reflex (the quads), no longer have balancing tone from the caudal aspects of the limb due to neurologic weakness. This makes the reflex seem bigger.

Differentiating LS pain from hip pain can be challenging. Start by palpating dorsally as you would in any other location. Then move the tail in all directions, including over the back ("tail jack"). If you are still confused, you can palpate the ventral aspect of the sacrum via the rectum. Some dogs object to the rectal more than that palpation, so be sure to palpate the brim of the pelvic canal to test the dog's general reactivity, and then compare to pushing up on the sacrum. If it is still not apparent. You can try lordosis postures (taking both legs and stretching them backwards while you push down on the lower back)- just be aware that this also puts pressure on the coxofemoral joints. And finally, always make sure to palpate the hips with the dog in lateral recumbency to differentiate hip pain.

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## **A Whole Lot of Shaking Part 1: Emergency Seizure Management**

By Carrie Journey DVM DACVIM (Neurology)

### **Definitions**

There are several definitions of seizure- some are electrochemical and discuss the hypersynchronous electrical firing of the brain that happens in seizures, some are more clinically oriented. The definition from the International Veterinary Epilepsy Task Force (IVETF) is a combination of both. It reads:

“Manifestation(s) of excessive synchronous, usually self-limiting epileptic activity of neurons in the brain. This results in a transient occurrence of signs which may be characterized by short episodes with convulsions or focal motor, autonomic or behavioral features and due to abnormal excessive and/or synchronous epileptic neuronal activity in the brain.”<sup>1</sup>

I think it's important to note that the manifestation of those uncontrolled activities can look like almost anything. As one of my mentors always said the brain ultimately controls everything, so when it goes haywire, all symptoms are possible. When I am trying to determine if something is a seizure, I focus on the clinical part of the definition- seizures are transient short episodes with convulsions or focal motor, autonomic or behavioral features.

### **Seizure Classification:**

There are many schemes for classification of seizures. Personally, I have used the terms Partial/Focal or Generalized, and these are classifications that IVETF keeps up in their new definitions. Partial seizures are seizure that arise from focal electrical activity in the brain, and they often have motor activity that does not involve the whole body. For instance, facial twitching, or a single limb twitching can be seen in a partial seizure. The IVETF has done away with the subclassifications of simple and complex- which were calls we made based on consciousness. They did this because impaired consciousness is a subjective call on the part of the clinician, and the task force is trying to get us more objective categories so we can do better research.

Generalized seizures involve electrical activity from the whole brain and therefore the abnormal movements are much more diffuse. There are further classifications of seizures by their clinical phenotype- ie what the animal does- does it move, is it stiff, thrashing, just vacant etc. However, the IVETF has recommended a simplification to just Generalized Seizure.

### **Reactive Seizures vs. Epilepsy**

When most vets say “epilepsy” they mean idiopathic genetic epilepsy. However, what epilepsy really means is just two seizures that happen more than 24 hours apart. So, by the correct definition, a brain tumor can cause epilepsy- specifically a form of epilepsy called structural epilepsy. When we categorize epileptics, meaning animals that have seizures more than 24 hours apart, we have:

- 1) Idiopathic: suspected genetic cause or no known cause.
- 2) Structural: identifiable cerebral pathology

- 3) Cryptogenic: Epilepsy where a cause is strongly suggested but none is found. This is the 13y golden retriever who also has behavior change and a menace deficits but you can't find its brain tumor no matter how hard you look.

We contrast epilepsy to reactive seizure. Reactive seizures are a reaction to something in the body, and that correct once that metabolic problem is corrected. The brain is a rather delicate, picky organ- it wants its environment just so. If it gets deprived of something, like glucose, or if a toxin is introduced, it will seizure. There is nothing wrong with that brain though, just the environment the brain is in. Correct that environment and the seizures should stop.

## **Treatment of Seizures**

Broadly speaking, treatment of seizures is broken up in to three categories: emergent, rescue and maintenance. This lecture will focus on emergent seizures, and in part 2 we'll tackle rescue and maintenance.

Emergent therapy is aimed at the seizures that are happening today right in front of you. Often these are severe seizures- it usually takes a cluster seizure or status epilepticus (SE, a continuous seizure longer than five minutes or multiple seizures without full recovery in between) for an owner to present to their veterinarian with an actively seizing pet. It's very important to stop the seizure. And equally important to remember that even partial seizures are still seizures. All too often I see veterinarians under react in status epilepticus. They'll stop when seizures are reduced to partial seizure twitching, and this is very dangerous. Mortality of status epilepticus increases dramatically as time goes on. As the brain depletes its resources by seizing, a patient can convert from convulsive status to non-convulsive status epilepticus.<sup>2</sup> Non-convulsive status is very difficult to recognize and treat in the veterinary environment and carries a poor prognosis.

In general, when treating emergent seizures, I start treatment with benzodiazepines- first rectal/nasal, then IV and then with a CRI as necessary. I concurrently gather the information I need to make a diagnosis by getting some lab work and taking a history. I treat any underlying causes based on that information. I then add a long acting anticonvulsant like phenobarbital and/or Keppra. If the patient is still seizing, we start to think about other therapies like propofol or general anesthesia. Finally, along the course of this treatment, it's important that we consider the rest of the body and the patient's intracranial pressure. There are several major morbidities that can result from status epilepticus that must be tended to if present and avoided if possible.

### **Rectal Benzodiazepines**

Benzodiazepines are the cornerstone of emergency seizure therapy. Blood levels of 300ng/ml generally considered anticonvulsant. Diazepam is rapidly broken down in to nordiazepam and oxyazepam by the liver, which are active metabolites and responsible for much of the anticonvulsant activity. Mealey et al showed that 2mg/kg of the intravenous preparation of diazepam rectally achieved acceptable anticonvulsant levels in the blood stream within 15 minutes, and these levels stayed over the anticonvulsant threshold for over 8 hours.<sup>3</sup> Similarly Papich has shown rectal doses of 0.5mg/kg reach 150ng/ml in 5 minutes and exceed 500ng/ml in 10 minutes, where as 2mg/kg rectally exceeds >500ng/mL

in 5 minutes.<sup>4</sup>

It's important to note that all of these studies were performed by using the intravenous preparation of diazepam rectally. Studies with compounded rectal suppositories in dogs have shown that, unfortunately, the drugs in this form are not absorbed rapidly enough to be clinically useful.<sup>5</sup>

### **Nasal Benzodiazepines**

Nasal administration can be an attractive substitute to rectal administration. Musulin et al showed that 0.5mg/kg of diazepam nasally both with and without a mucosal atomization device reached acceptable blood levels within five minutes of administration.<sup>6</sup>

Nasal midazolam (0.2mg/kg) has been shown to be equivalent to IV diazepam in its ability to control status epilepticus in children and in fact the time to seizure control was shorter in the intranasal midazolam group.<sup>7</sup> I find that midazolam is a better choice for nasal use than diazepam because it is generally a smaller volume. I'm a big fan of nasal administration overall. It works quickly and effectively. The only caveat is that you need to be careful to not get bit by the thrashing, chomping seizure patient.

### **IV Access and Basic Laboratory Analysis**

I think it's a good general policy to place an IVC in all patient that present for SE or cluster seizures. When you hit that vein, get a BG at a minimum, and if owner finances allow, a general CBC and Chemistry including electrolytes is recommended.

### **Benzodiazepine IV and Start a CRI**

Once IV access is obtained, if the patient is still seizing, give another bolus of benzodiazepines. If it doesn't stop in a few minutes, give it again. In general, I will repeat the same dose of benzodiazepines three times. Don't be shy! It's hard to kill a patient with benzodiazepines.

The dose for diazepam is 0.5mg/kg and the dose for midazolam is 0.2mg/kg. If one benzodiazepine doesn't stop the seizure, I'll switch to the other one and give that three times as well. Once you stop or pause the seizure, start a CRI. My general rule is whatever cumulative dose it took to stop the seizure is what I start in mg/kg/hr. So for example if it took two 0.5mg/kg doses of valium to stop the seizure, then start a 1mg/kg/hr CRI.

If seizures continue:

Diazepam CRI: bolus 0.25mg/kg and increase CRI by 0.1mg/kg/hr until 2.0mg/kg/hr

Midazolam CRI: bolus 0.1mg/kg and increase CRI by 0.05mg/kg/hr until 0.5mg/kg/hr

Generally, midazolam is more recommended for CRI use. It has an aqueous base and is a bit friendlier to the veins. However, I find in the clinical setting there is rarely a large difference between diazepam or midazolam as far as catheter survival and patient morbidity- so don't be shy and use what you have.

## **The Quick and Dirty Seizure History**

We will cover a more detailed history in the next lecture. This history taking should be done later once the situation is under control. While you are working on getting the dog stabilized, send a staff member to get the following data.

1. Is this the first seizure the pet has ever had?
2. If not, have they had a severe seizure like this before?
3. Any chance they got in to something today?
4. Any other neurologic symptoms (behavior change, ataxia, circling, weakness) in the last month?
5. Any signs of systemic disease (V/D/C/S, changes in appetite, PU/PD/PP)
6. What medications are they on if any?

That information will get you far, and generally can be collected by a well-trained assistant.

## **Treating the Underlying Cause:**

If your history, physical exam and lab work have given you a good working diagnosis, then it's important to treat that immediately. Remember, if these are reactive seizures we need to return the brain back to homeostasis to stop the seizures. Some common conditions that can be address quickly include hypoglycemia, hepatic encephalopathy and toxin ingestion. Hypoglycemia of <40 is generally severe enough to result in convulsion and is treated with IV dextrose, first by bolus, then followed by titrated drip. While the underlying cause of hepatic encephalopathy may require high level and long term care, the immediate problem can be addressed with lactulose enemas and antibiotics. Finally, toxin treatment will of course be dictated by what toxin was ingested, but generally decontamination, supportive care, and, in many instances, lipid emulsion therapy may be useful. In this therapy, patients are infused with the lipid base for TPN with the hopes of creating a lipid sync.<sup>8</sup> This lipid sync will bind lipophilic drugs pulling them from affected organs. In theory, toxins that are lipophilic enough to cross the blood brain barrier should respond well to this therapy.

## **Start a Maintenance Anticonvulsant**

Whether or not the patient is still actively seizing, I will generally at this point load a maintenance anticonvulsant. The use of phenobarbital and keppra can be effective in management of these acute emergent seizures, as well help stave off any seizures that may be coming in the days to come.<sup>9,10,11</sup>

If you are using phenobarbital, generally you want to give a loading dose. On maintenance therapy alone, phenobarbital takes 2 weeks to reach steady state, and we don't have that kind of time right now. I generally give four or five doses of 4mg/kg as this will bring a pet to a therapeutic phenobarbital blood level. The time interval for these doses depends on the patient's seizures. They can be given in rapid succession if a patient is not responding to other therapies. Twelve hours after the last loading dose, I start the patient on 2-4mg/kg twice daily until a long-term anticonvulsant plan is made.

Keppra is a very safe drug, and is my drug of choice when lab work is not immediately available or if I have concerns about the patient's liver. I typically give 60mg/kg diluted 1:1 with saline and given slowly over a few minutes. This dose should be repeated every 8 hours until a long-term anticonvulsant plan can be made.

Speaking of long term plans, once you have successfully controlled this acute event, the patient need not necessarily stay on these drugs long term. This is especially true if the seizures seem reactive in nature. We'll discuss choice of long term maintenance anticonvulsants in the second part of this lecture.

If your patient is breaking through one maintenance add on, try adding the other. Should phenobarbital and keppra not be available or sufficient for your patient you can also load some maintenance anticonvulsants rectally. You can load Kbr rectally by giving 400-600mg/kg divided over several doses. Use caution, as this can cause a rather significant osmotic diarrhea that I can assure you that it does not endear you to your nursing staff.

Brewer et al looked at zonisamide 10mg/kg given rectally in PEG and in water. PEG carrier had a higher bioavailability, and both rectal administration bioavailabilities were lower than oral. The authors recommend higher than 10mg/kg if rectal route is to be used (no specific dose given. Try 15-20mg/kg?). Peak plasma concentrations were 5 to 6 hours post administration. So, this is a nice way to get started on medium term seizure control in a patient that can't take oral meds, but not helpful immediately.<sup>12</sup>

### **Still Seizing? Try Ketamine.**

In a recent retrospective analysis ketamine use terminated 100% of episodes of status epilepticus and 29% of cluster seizure events.<sup>16</sup> Personally, I have seen dramatic benefit in using ketamine for these patients, and now will try a 5mg/kg bolus of ketamine or telazol prior to starting benzodiazepine CRIs. I have also had this an effective treatment given nasally at home for a limited number of patients with severe uncontrolled cluster seizures.

### **Still Seizing? Try Propofol**

I must confess that I am not a big fan of propofol for this use. I feel this way for a couple of reasons. First of all, multiple sources tell us that propofol has both pro and anti-convulsant properties. That means that in some patients, propofol can actually cause seizures, and those seizures may in fact be non-convulsant in nature. This means your patient can look quite still, but can still be seizing inside its brain. To overcome this limitation in human medicine, propofol is often titrated to effect using continuous EEG monitoring.<sup>13, 14</sup> Unfortunately, this modality is rarely available in veterinary practice, so propofol must be approached with caution. Additionally, propofol anesthesia is occasionally accompanied by myoclonic twitches, which can complicate monitoring of your patient.<sup>15</sup> Finally, while all emergency anticonvulsants, save Keppra, have a respiratory depressive effect propofol is particularly potent in this regard.

I only use propofol in dogs as a tool of last resort before going to general anesthesia. Occasionally it does become necessary. If you find yourself in this dire place, start a propofol CRI. Give a 4mg/kg bolus Then start CRI at 0.3mg/kg/hr. Increase by 0.1mg/kg/hr until 0.6mg/kg/hr. Remember to monitor respiratory rate and character very carefully.

### **Still Seizing? Time for a Sanity Check**

Is your patient still seizing? If your patient is still seizing after benzodiazepines, phenobarbital, keppra and propofol, we need to check in. Is this really a seizure? I've been fooled a few times by a severe tremor. If you think tremors are a possibility, try a dose of robaxin at 30-50mg/kg IV. Once again, as we stack these drugs, respiratory compromise becomes more and more likely- so monitor carefully.

### **Still Seizing? General Anesthesia**

If nothing else works, you need to place this patient under general anesthesia. Induce with alfaxalone or propofol, intubate and maintain on isoflurane. You want these patients to reach a good surgical plane of anesthesia. It's important to maintain normal systemic blood pressure, as well as a normal to slightly low ETCO<sub>2</sub> (30-35).

### **The Rest of the Body**

Seizing is a very energetic event and can result in significant morbidity for your patient when prolonged or severe. Monitor TPR frequently, at least every hour, in the early phases of treatment. Patients may come in hyperthermic secondary to seizures and may become hypothermic as the seizure stops. A diseased brain does not necessarily regulate body temperature as you would expect. Be conservative with attempts to correct abnormal temperatures.

Monitoring of heart rate can alert you to changes in the intracranial pressure (See Cushings reflex below). In a busy ICU, I find it convenient to put these patients on a continuous EKG, so staff can keep a close eye. Respiratory rate and character can be slowed by drug therapy, and patients with prolonged seizures are prone to both noncardiogenic edema and aspiration pneumonia. I find it prudent to treat with antiemetics to try as well as elevate the head above the heart to try to prevent aspiration pneumonia. Monitor systemic blood pressure frequently, at least every four hours, as it can alert you to changes in your intracranial pressure.

### **Intracranial Pressure**

Patients with prolonged seizure may have elevations in intracranial pressure. The Cushing's Reflex/Triad can be a helpful clinical tool to identify these patients. The Cushing's reflex was first described by Dr. Harvey Cushing during his physiologic experiments on dogs in 1901. It is still considered an excellent predictor of increased intracranial pressure today. Dr. Cushing noted that when the pressure in the skull was increased, patients got a constellation of signs called the Cushing's triad which include: bradycardia, hypertension and irregular breathing. As pressure in the brain increases, the brain receives less blood (Cerebral Perfusion Pressure or CPP decreases). The brain is an autoregulating organ though, so it responds to this loss of CPP by increasing systemic vascular resistance, resulting in systemic hypertension. There is a reflex response by the aortic baroreceptors that drops the heart rate.

Should you recognize this dangerous state in your patient, immediate action is warranted. Osmotic Diuretics (Mannitol 1g/kg IV slow or Hypertonic Saline 4 – 6 ml/kg IV slow) are recommended, with or without the additional use of Lasix.

The head should be elevated to 30 degrees above the heart, taking care not to place pressure on the jugular veins (a slant board is ideal for this). If your patient is intubated, ensure a normal or low normal

ETCO<sub>2</sub> (30-35). If a structural cause of epilepsy is suspected, therapy with corticosteroids may also be warranted (0.1-0.2mg/kg Dexamethasone SP).

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## **A Whole Lot of Shaking Part 2: Maintenance Seizure Management**

**By Carrie Journey DVM DACVIM (Neurology)**

### **INTRODUCTION**

Seizures are a very common presenting complaint in small animal practice, with an estimate 0.7% of all dogs having a seizure at some point in their lives. That's one in every 130 dogs seen in a veterinary practice! Within certain breeds, the incidence can be much higher. Did you know that 3% of Labrador Retrievers have seizures?<sup>1,2</sup>

This lecture will aim to demystify seizures, their diagnosis and treatment. We will also be reviewing the most current guidelines from the International Veterinary Epilepsy Task Force (IVETF)

### **SEIZURE DEFINITIONS**

Seizure definitions and classifications were covered in part 1 of this lecture series. Patients that present for reactive seizures will only need emergency therapy. Once their brain is returned to homeostasis, they no longer require anticonvulsants. In this lecture, we will be dealing with epileptic patients; that is, patients who have had at least two seizures more than 24 hours apart.<sup>3</sup>

It's very important to realize that owners will call a variety of things seizure, when in fact it is not a seizure at all. The following list can present as seizures, but aren't seizures:

- Vestibular disease
- Narcolepsy
- Neuromuscular disease (particularly those brought on by exercise)
- Severe neck pain
- Cerebellar disease
- Tremors
- Syncope
- Intense pruritus
- Mouth pain
- Behavioral problems

### **TAKING AN EXCELLENT SEIZURE HISTORY**

In Lecture 1 we covered the quick and dirty history. Those five questions can be asked rapidly by an assistant in a patient who is actively seizing. For this appointment, we need to dive deeper. This is not a quick appointment. We need to get a lot of information, and to do a lot of education. So, I start this appointment with some open-ended questions like, "Tell me what's happening". After the clients tell me their story, I make sure we've got the event well classified.

Questions to ask to classify an event:

- What was the animal doing before the event?
- How long did the event last?
  - Realize that this is often exaggerated.

- Was there drool? Urine? Feces? Vomit?
- Define the motor activity. Did they shake, stretch or paddle? Which legs? Did they chew or have other facial movements?
- Were they conscious? ("all there?")
  - Realize this is subjective. Try to differentiate an actual response: eyes are often open, did they look at you, change behavior at all when you talked to them?
- What did they do right after?
- Do all the events look similar?
- How often do the events happen?
- Do they happen at a certain time of day?
- How often are you with your pet?
- Does anything seem to trigger events?
- Do you know any genetic relatives? Are they normal?
  - In ten years of practice I have never had a breeder admit to having seizures in their line. Perhaps this is because people are not following up with their breeders? This communication is important!
- Anything else going on?
  - Changes in behavior- going to weird rooms, change in relationships to people or other pets
  - Going "deaf or blind"
  - "Getting old"
  - Loss of house training
  - Coughing, Sneezing, Vomiting, Diarrhea
  - Trouble walking?
  - Trouble getting up?
  - Dizzy? Off balance? Drunk?
  - PU/PD
  - Appetite changes?

## **DIFFERENTIALS AND DIAGNOSES**

There are three categories of processes that causes of seizures: Metabolic seizures, structural seizures and idiopathic epilepsy. Most clients, armed with Dr. Google, seem quite sure that their pet's seizures are caused by anti-parasite treatments (possible, but rare) or epilepsy. It helps my clients understand when I tell them that seizures are just a symptom, like a cough of the brain. We can cough because we have asthma, or a cold or lung cancer- but all we see from the outside is a cough. Seizures are the same. We can seizure because of low blood sugar or a brain tumor, and sometimes the only symptom is a seizure.

### **Metabolic/Reactive Seizures**

Metabolic Seizures (IVETF would call these reactive seizures) are seizures due to a disturbance of the brains homeostasis. Often these patients have other symptoms of metabolic illness, like weight loss, vomiting/diarrhea, etc. Common causes of metabolic seizures include hypoglycemia, liver failure and toxins. Common toxins include: bromethalin, chocolate, ivermectin, tremorgenic mycotoxins\*, Insecticides: pyrethrins\*, organophosphates, metaldehyde\* (\*more often present with tremors than seizures, but in severe cases can seizure). Rarely uremia or electrolyte disturbances can cause seizures. We diagnose these seizures with lab work and history.

### **Structural Epilepsy**

Structural disease of the brain means there is something physically wrong with the brain. Often, but not always, these patients will have other neurologic abnormalities. In one study, 23% of dogs with structural epilepsy had a normal interictal exam, so we cannot depend on finding other neurologic abnormalities to make this diagnosis.<sup>4</sup>

The list of specific causes is long, but most often we see neoplasia, vascular, inflammatory and anomalous/developmental (i.e. hydrocephalus) seizures. Less commonly we see infectious etiologies (geographically dependent) and nutritional causes (thymine in cats). Diagnosis usually requires advanced imaging (preferable MRI), spinal tap, infectious disease testing and/or biopsy.

### **Idiopathic Epilepsy**

Idiopathic/Genetic epilepsy is the most common cause of seizures in dogs, and is fairly common in cats with 22% of all cat seizures being attributed to idiopathic epilepsy.<sup>5</sup> They should have a normal interictal neurologic exam. However, it's important to note that not all dogs with a normal exam have epilepsy.

Patients presenting with idiopathic epilepsy tend to be between six months and six years for dogs. Median age of cats with idiopathic epilepsy is 3.8y (range 0.4-14.4), while structural epilepsy has a median age of 7.8y (range 0.3-19). This age difference between these causes is not statistically significant in cats.<sup>5</sup>

While both partial and generalized seizures can be seen in epilepsy in dogs and cats, cats with epilepsy more often experience partial seizures<sup>5</sup>, while dogs are more likely to have generalized seizures(77%).<sup>4</sup> No familial/genetic cause currently known.

When the brain is closely examined by MRI, CSF and even on post mortem, no structural cause can be found in idiopathic epilepsy. I often tell my clients it's "faulty wiring". It is often suspected (and in some cases proven) to be genetic in origin.

Diagnosis of idiopathic epilepsy is a diagnosis of exclusion. In practice, not all dogs will receive full diagnostics due to expense and availability. When diagnosing this condition, the IVETF gives us a handy classification scheme for confidence levels in dogs.<sup>6</sup>

1. IVETF Tier I confidence level: Normal CBC/Chem/UA/Pre and Post prandial bile acids, other biochemical and systemic evaluations as indicated by clinical picture
2. IVETF Tier 2 confidence level: Normal MRI & CSF. Negative on regionally appropriate infectious disease screenings. There are some MRI and CSF changes that can be seen secondary to seizure. These changes should at least partially resolve within 16 weeks.
3. IVETF Tier III confidence level includes EEG confirming seizure, however even the IVETF admits this level of testing is infrequently available and not yet standardized.

I am often asked, when should we MRI a patient? MRIs are expensive, and have limited availability, but the good news is, not all epileptics need an MRI. We should recommend MRIs for the following patients.<sup>6,7</sup>

1. Patient is a cat
2. Is a dog less than 6m, greater than 6y old
3. Inter-ictal neurologic abnormalities
4. Status-epilepticus or cluster seizure (non-reactive in nature)
5. Presumptive idiopathic epilepsy with drug resistance to a single AED (antiepileptic drug) titrated to the highest tolerable dose.

## TREATMENT

As previously discussed, the best treatment of a seizure is to correct the underlying cause of a seizure, so do that whenever possible (i.e. dextrose, and not Valium, is the most important drug to give to a patient experiencing hypoglycemic seizures). However, the seizures often also need treatment. There are three types of seizure treatments: emergent, rescue and maintenance. We covered emergent treatment of seizures in part 1 of this lecture series.

I would give the general caveat before we dive in that seizure therapy is a little bit like cooking. Everyone who makes marinara sauce makes it a little different and too many cooks in the kitchen screws it up. So, this is merely my recipe. If you ask another neurologist, they will likely give you a slightly different one.

### Rescue Treatment

Rescue treatment is targeted for seizures occurring in the next couple of days, especially in regards to cluster seizures or sharp changes in frequency (usually in the at-home setting). We have two categories of treatments here: benzodiazepines and burst therapy with maintenance drugs.

You can use the intravenous formula of benzodiazepines at home as both a rectal and intranasal drug. A complete discussion of the pharmacokinetics of this route of administration is available in Part 1 of this lecture seizures. IV diazepam binds to plastic and is light sensitive,<sup>8</sup> so we are left with two unattractive scenarios for its use at home: either pre-draw syringes and try to protect them from light, knowing that we will lose drug potency over time, or script a bottle of diazepam and syringes with needles to stressed-out pet parents to use in the middle of an emergency. Neither is ideal, and unfortunately compounded suppositories have shown to be ineffective.<sup>9</sup> There is also a rectal diazepam gel available by prescription. This gel has the benefit of being more shelf stable, but can be quite expensive, especially in a larger dog.

Before recommending nasal administration, I review safety with my clients. There is a chance of getting very seriously bit by a seizing animal when you are trying to hold its face and get something in its nose. If they seem unsure, or if the pet has particularly violent jaw behavior during the seizure, or post-ictal aggression I will preferentially recommend rectal administration purely for safety reasons.

Dose for rectal diazepam is 1-2mg/kg. When using the IV prep, I use the higher end of the dose, as we are likely to lose some drug potency due to handling. When using the commercially available gel, we will often use the lower end of the dose, due to cost and increased stability of the product.

Dose for intranasal diazepam is 0.5mg/kg, and intranasal midazolam is 0.2mg/kg

If a dog is more prone to cluster seizures, rather than SE, I preferentially choose burst therapy with clorazepate. Clorazepate is an oral benzodiazepine. I dose clorazepate at 1mg/kg q8h until seizure free for 48 hours. Some dogs will be overly sedate at this dose and require a dose reduction (trial and error is common- I reduce to BID, but if they break through that, will try 0.5-0.75mg/kg q8h). This drug should ideally not be used long term. Dogs develop a tolerance to benzodiazepines with long term use, which means this drug will be less effective over time and could potentially result in a lack of response to valium IV for emergency seizure control at a later date.

When utilizing burst therapy, I prefer to use benzodiazepines. However, not all patients respond well to them. In these cases, we can utilize some of our maintenance drugs. My preference is to use Keppra for burst therapy. It has a short half-life, high safety factor and low side effect profile, which makes it ideal for this use. I will start Keppra after the first seizure in a cluster. In a patient who is not on routine maintenance, I give 60mg/kg (route? I am assuming IV but wasn't sure if you wanted to be clear) once, followed by 30mg/kg q8h until seizure-free for 48 hours. If the patient is already on Keppra, I will double their Keppra dose for 48 hours after the first seizure in a cluster. I see a variety of other drugs used as burst therapies (pheno, zonisamide, etc), but I personally try not to use them in this capacity. These drugs have more side effects, so higher doses are not as well tolerated, and temporarily increasing a drug that is given for routine maintenance can interfere with a veterinarian's ability to collect meaningful blood levels in an urgent situation.

## **Maintenance**

There is not one perfect maintenance drug, or we would only have one drug. IVETF Recommends therapy be started:<sup>10</sup>

1. Two or more seizures in a 6-month period
2. Status epilepticus or cluster seizures
3. Severe postictal signs
4. The seizure frequency or duration is increasing and/or seizure severity is worsening over 3 interictal periods

Which drug you choose is based on many factors: your patients and their underlying personalities, comorbidities as their owners' personalities, schedules and financial limitation. Very often veterinarians ask me "what is your favorite anticonvulsant?", and the honest answer is, it depends. There isn't one perfect drug, or we would only have one drug. We have nine that are used in veterinary medicine. Very often more than one drug will be appropriate for a given patient's situation, and often the first drug and dose you try isn't going to work perfectly. So rather than give you a formula, my goal is to give you the tools you need to make rational decisions about anticonvulsants in your patients.

## **Patient Factors**

The most important part of choosing an anticonvulsant is tailoring it to your patient. It's important to understand their underlying disease process and any comorbidities they have and preferentially pick drugs that suit them. For instance, for patients with structural disease I try to select drugs with minimal sedation and ataxia as a side effect (i.e. zonisamide or Keppra), as monitoring for behavior change or ataxia can be an important part of monitoring their underlying disease. I try to avoid phenobarbital in patients with pre-existing thyroid disease as it can complicate monitoring.<sup>11</sup> Similarly, I try to avoid phenobarbital and KBr in patients who already over-eat, or have PU/PD as the side effects of these drugs

can worsen these pre-existing conditions. Zonisamide can cause a renal tubular acidosis, which can result in urinary stone formation, so it's a bad choice in patients who already have a tendency to form urinary stones.

Cats are hard. Evidence-based choices of anticonvulsants are more limited, and consistently medicating most cats is challenging. Both Keppra and phenobarbital are published as anticonvulsants for cats.<sup>12</sup> Pilling a cat three times a day is rough. So Keppra, while studied and effective, is not my first choice.

Zonisamide, gabapentin and topiramate have pharmacokinetic and non-epilepsy based studies in cats in the literature, but no specific clinical trials for this purpose.<sup>13</sup> Anecdotally they are used and reported to be intermittently effective. Zonisamide may cause inappetence/vomiting frequently in cats, especially at higher doses.<sup>13, 14</sup> Oral diazepam has also been historically used as an anticonvulsant, but a rare fatal hepatic necrosis has made this drug choice less popular.<sup>13</sup>

### **Owner Factors**

The best anticonvulsant in the world doesn't do our patient any good if the owner won't or can't give it.

When choosing a drug, we need to be mindful of things like the owner's schedule. Unless someone is consistently home midday, TID drugs are not very practical.

Owners have a wide variance in tolerance to side effects. While good communication in advance can mitigate some of this, some owners are philosophically unwilling to tolerate certain potential side effects, which will influence our drug choice. Finally, financial limitations are a reality for every veterinarian. Remember to factor not only the drug cost, but also the cost of the associated recommended monitoring when choosing a drug. This can make phenobarbital relatively more expensive than say Keppra or zonisamide.

### **Treatment Progress**

The key to excellent seizure management is organization. This is a team effort between you and your client. Their job is to give the drugs on time and record seizure frequency. Your job is to be a level-headed captain of the ship, and a good partner to your client in what can be a stressful disease.

Completely and totally exhaust one drug before adding a second drug. This means titrating to the max safe dosage, blood level, or to intolerable side effects. Do not add a second drug until you have completely crossed out the first one.

### **Dietary therapy**

Purina has a new diet therapy out based on ketogenic medium chain triglyceride fats (MCG).<sup>16</sup> Ketogenic diets are a common therapy in children with epilepsy, but were previously thought to be ineffective in dogs.

The study followed 21 dogs in a six month prospective, randomized, double-blinded, placebo-controlled, cross-over trial (three months in with MCG therapy, then three months on a placebo diet). They measured serum ketone levels, AED blood concentrations, seizure frequency and seizure days. Dogs on the MCG diet showed good reduction in seizure frequency:

- 71% of dogs showed a reduction in seizure frequency
- 48% of dogs showed a 50% reduction or greater
- 14% of dogs became completely seizure free
- No change to AED concentrations

As with many veterinary studies, more subjects would have been nice, but I think this was a well designed study. The overall mean reduction in seizures seen was not dramatic (2.6 to 2.3). However, there was a dramatic and rather rapid response in some individuals. This therapy has little negative consequence, so seems a reasonable thing to try to me. In practice, for me, clients have seemed resistant to this so far, which surprises me. Prescription food is always a bit annoying/expensive. I think honestly there is a brand stigma problem here. Clients who want a more holistic approach are often resistant to large national brands. I have yet to see any dramatic improvements in my patient population, however, frankly - my patient population tend towards generally pharmaco-resistant and severe cases, so this is not necessarily a comment on the food.

### **Cannabidiol (CBD)**

Please see my notes on Medical Marijuana in Veterinary Medicine for a full discussion of this topic. There is a lot of interesting and promising research about CBD for seizure control with children with intractable epilepsy.<sup>17, 18</sup> However, there are no veterinary studies completed yet. One study is ongoing at CSU. (<http://csu-cvmb.colostate.edu/vth/veterinarians/clinical-trials/Pages/efficacy-of-cannabidiol-for-the-treatment-of-epilepsy-in-dogs.aspx>). There is a lot of data about marijuana toxicity in dogs<sup>19, 20, 21</sup>, and some preliminary data about supplement tolerability and client perceptions in veterinary patients.<sup>22</sup>

However, the FDA, AVMA and State Vet Medical Boards have all said that there isn't enough data on safety and efficacy, and prescribing marijuana still remains illegal for veterinarians. There are rational arguments that CBD supplements do not fall under the legal definition of "marijuana", but legality of recommending cannabis based products is still very shaky. Please be careful.

### **Acupuncture**

The veterinary literature has two studies that support the use of acupuncture in patients with epilepsy, specifically gold wire implants. Goiz-Marquez et al showed 9/15 dogs had at least a 50% reduction in seizures after a 15-week treatment with gold wire implants.<sup>23</sup> Klide et al showed 5/5 dogs had a reduction in seizure frequency with gold implants, though the decrease was transient in two dogs.<sup>24</sup>

However, larger, better controlled studies in human medicine show less promising results. A study of 34 patients receiving either needle acupuncture or a control sham acupuncture showed no changes in health-related quality of life scores between treatment and sham control.<sup>25</sup> Another study of 29 patients needle acupuncture, also sham controlled. Both groups had decreases in seizures, but neither was statistically significant.<sup>26</sup> Finally, there is a Cochrane Review of 17 randomized controlled trials comparing placebo/sham treatment. Overall, they found acupuncture was not effective compared to Chinese herbs, valproate or phenytoin. Catgut implants at acupuncture points showed some promise. One study had a significantly higher portion of people become seizure-free, and another did not show significance in that regard but did show a 50% reduction in the catgut implant group. Overall, acupuncture did not have excess adverse events. The Cochrane Review conclusion was that available studies are small, heterogeneous and have high risk of bias. They concluded current evidence does not support acupuncture for treating epilepsy.<sup>27</sup>

Personally, I remain skeptical of acupuncture as an effective therapy for epilepsy. The limited veterinary studies we have are positive. However, there are far more negative results from larger and better designed studies than positive ones. That being said, the treatment isn't harmful, so if my clients want to do this I take a "can't hurt, might help" approach. We really need larger and better studies to be sure of effectiveness. The trials that seem to work all seem to involve implants, so that may be a more effective way to pursue this therapy with interested clients.

## **Surgical therapy**

Surgery to remove a seizure focus is offered in people with medically intractable epilepsy. In a Meta-analysis of seizure outcomes following epilepsy surgery they reviewed 83 studies and found a long-term seizure free outcome in 66% of temporal lobe resections, 46% of Occipital/Parietal lobe resections, 27% of frontal lobe resections and 16% of multiple subpial transections.<sup>28</sup>

Broadly: Resective procedures, corpus callosotomy, and vagal nerve implantation have been considered for dogs with epilepsy.<sup>29, 30</sup> The reality is we need to get a whole lot better with EEG before resective surgeries can be truly considered in Vet Med. Corpus callosotomy is published in six normal dogs; 5/6 dogs were neurologically normal within 14 days of the procedure.<sup>29</sup> One dog had persistent deficits for the study period. Anecdotally, I worked with a neurologist who performed this procedure and he thought it was a reasonable risk and good choice for patients with intractable epilepsy

Vagal Nerve Stimulators have been studied in 10 dogs with epilepsy.<sup>31</sup> Overall seizure frequency was not reduced compared with the control period, however the last four weeks of the treatment period did show a statistically significant reduction in mean seizure frequency by 34.4%. Unfortunately these implants are **very** expensive (\$12,000 without markup last I checked).

## **DRUG FORMULARY**

Should these notes not be handy, Plumbs Veterinary Drug Handbook is an excellent resource. In the following section, I've added some additional literature, as well as my personal experience with the drugs.

Unless otherwise noted, levels are typically available through most large scale veterinary diagnostic companies (i.e. Antech, Idexx) or directly from the Clinical Pharmacology Department at Auburn University.

Cost of these drugs is highly variable and can change seemingly week to week. In general I recommend my client check [www.goodrx.com](http://www.goodrx.com) for current prices. I have found Costco pharmacy to be fairly consistent and reasonable priced versus other large chain pharmacies.

1. Diazepam
  - a. Proposed mechanism of action: Facilitation of GABA receptors.
  - b. Metabolism: High protein bound. Liver metabolized.
  - c. Toxicity/Side Effects:
    - i. Should not be used in dogs as routine therapy as they rapidly build tolerance
    - ii. Paradoxical excitement is possible in cats

- iii. Dose dependant lethargy, sedation and ataxia is common
    - iv. Rare but fatal oral hepatic toxicity in cats.<sup>32</sup> Should not be used as a first line, and owners should be informed of risk prior to use.
  - d. Dose:
    - i. 0.2-1mg/kg q 12h
  - e. Levels: Not commercially available.
  - f. Effectiveness/Personal Experience: This has been an effective therapy in cats, however the reports of hepatic toxicity have dramatically limited its use.
- 2. Felbamate aka Felbatol
  - a. Proposed method of action: Several mechanisms are proposed including action via the GABA channel, inhibition of voltage gated Ca<sup>2+</sup> channels and NMDA inhibition.
  - b. Metabolism
    - i. Organs: mixed hepatic metabolism (30%) and excretion in urine unchanged (70%)
    - ii. T<sub>1/2</sub>: 5 to 6 hours
  - c. Toxicity/Side Effects
    - i. Blood dyscrasias, liver enzyme induction, liver failure, agitation, KCS, tremors and limb rigidity have been reported in dogs.
    - ii. 4/12 dogs in one study developed liver disease. These patients were also on high dose phenobarbital.
  - d. Dose: 10-15mg/kg q8-12h
  - e. Levels: Not available commercially
  - f. Effectiveness/Personal Experience
    - i. I do not used this drug frequently due to expense
    - ii. Literature supports its use in dogs
      - 1. 12/16 dogs had a reduction in seizures
      - 2. 6/6 dogs with focal seizures improved, 2 of them became seizure free.
- 3. Gabapentin aka Neurontin
  - a. Proposed method of action: A structural analog of GABA presumed initially to work at the GABA channel (thus the name). This theory has fallen out of favor. It is now believed that Gabapentin most likely acts through inhibition of calcium channels.
  - b. Metabolism
    - i. Organs: 30-40% hepatic metabolized, the rest is excreted unchanged in the urine.
    - ii. T<sub>1/2</sub>: 3 to 4 hours
  - c. Toxicity/Side Effects
    - i. Sedation, ataxia, polyphagia, weight gain
    - ii. Generally, well tolerated and safe
  - d. Dose
    - i. 10-20mg/kg q8h
  - e. Levels
    - i. 4-16mg/L (However, I know of nowhere to run these samples commercially)
  - f. Effectiveness/Personal Experience
    - i. In humans, this drug is thought to be more effective as an anticonvulsant in partial seizures. I generally have found it to be the least effective anticonvulsant in my patients.
    - ii. There are two studies that support its use in dogs for seizures
      - 1. In 7/17 dogs had a 50% or greater reduction in seizures. This was not statistically significant.
      - 2. 6/11 dogs in another study had a 50% or greater reduction in seizures, which was statistically significant.

- iii. It does seem very helpful in multimodal pain therapy, and we use it commonly in both cases of neuropathic pain and chronic orthopedic pain.
- 4. Levetiracetam aka Keppra
  - a. Proposed method of action:
    - i. Unknown. Studies have failed to link this drug to any of the usual suspects (i.e. ion channels, GABA or NMDA receptors)
  - b. Metabolism:
    - i. Organs: 90% is excreted in the urine unchanged and the rest is hydrolyzed in the serum.
    - ii. T1/2: 3 to 4 hours
  - c. Toxicity/Side Effects:
    - i. This is an extremely safe drug in dogs. Studies have shown minimal side effects (mild GI side effects and ataxia) at doses of up to 20 times the normal starting dose given for a year. I rarely see any significant side effects with Keppra.
    - ii. Patients on the XR variety may pass whole “ghost pills” in feces. Active ingredients have been absorbed. If owners are concerned, serum levels can prove this.
  - d. Dose
    - i. 30-40mg/kg PO q8h. Increase as needed (usually in 10-20mg/kg increments) until cost prohibitive.
    - ii. Can be given IV in the event of cluster/status epilepticus at a dose of 40-60mg/kg as a bolus. The IV form is very expensive. One dose can be a couple of hundred dollars in a medium to large breed dog.
    - iii. There is an extended release form available, dosing is similar but in a q12h frequency (20-40mg/kg q12h). Capsules cannot be broken or split. Limited to 500 and 650mg tablet sizes, which precludes use in patients <10kg.
  - e. Levels: In humans 5 to 45mcg/ml. Some question as to the significance of levels in regards to seizure control in people.
  - f. Effectiveness/Personal Experience
    - i. May be neuroprotective (i.e. prevent seizure related brain damage)
    - ii. An excellent choice in patients with liver disease (primary hepatic encephalopathy patients or in dogs with phenobarbital associated liver toxicity)
    - iii. We use the IV form a good deal for refractory status epilepticus.
    - iv. This has been clinically studied in cats: 7/10 cats responded to therapy, and the mean reduction in seizures was 92%. A single 500mg extended-release tablet daily has also been shown to be effective. However, it is a large pill that cannot be crushed, so this dosing is still practically challenging.
    - v. In dogs 8/14 dogs studied prospectively responded, and had 77% reduction in seizure frequency.
    - vi. There is significant pharmacokinetic variability of the extended release Keppra, especially in dogs on phenobarbital.
    - vii. The “honeymoon” effect: The study noted above also showed a significant portion of the dogs (6 dogs) returned to base line seizure frequency or worse 4-8 months after starting therapy.
    - viii. In one study at 20mg/kg Keppra was no more effective than placebo (Munana et al 2012)
- 5. Phenobarbital
  - a. Mechanism of action: Via the GABA receptors, increases the flux of chloride ions
  - b. Metabolism:
    - i. Induces and is metabolized by the cytochrome P450 system in the liver
    - ii. Highly protein bound
    - iii. T1/2: 37-73h

- c. Toxicity/Side Effects:
    - i. The Annoying
      - 1. Sedation
      - 2. Ataxia
      - 3. Polyphagia
      - 4. Polyuria/polydipsia
      - 5. Interference with thyroid testing
      - 6. Increased liver enzymes
      - 7. Increased cholesterol and triglyceride concentrations
    - ii. The Dangerous
      - 1. Hepatotoxicity
      - 2. Blood dyscrasias
      - 3. Superficial necrolytic dermatitis
      - 4. Hypoalbuminemia
      - 5. Potentially pancreatitis
  - d. Dose
    - i. 2-3.5 mg/kg BID as a starting dose. Titrate upwards based on blood levels
    - ii. New Pheno Daily Dose in mg = (Desired Pb Level/Actual Pb Level) x Current Total Daily Pheno Dose
    - iii. Loading: Give 15-20mg/kg divided in a 24-hour period
  - e. Levels
    - i. Reported normal range varies slightly by labs, but generally from 15 to 40mg/L
    - ii. Levels >35 are associated with an increased risk of hepatotoxicity.
    - iii. Levels can be measured one to three days after loading, but will not necessarily correlate to levels on a maintenance oral dose.
  - f. Effectiveness/Personal Experience
    - i. The longest history of chronic use of all AED in veterinary medicine.
    - ii. Decreases seizure frequency in 60-93% of dogs with idiopathic epilepsy.
    - iii. In one trial, 85% of dogs became seizure-free for six months when given phenobarbital.
6. Potassium Bromide
- a. Mechanism of action: Bromide hyperpolarizes neurons via the GABA associated Cl channel
  - b. Metabolism:
    - i. T1/2: 25-45 days
    - ii. Excreted unchanged in the urine
  - c. Toxicity/Side Effects:
    - i. Cannot be used in cats due to reactive pneumonitis
    - ii. Sedation
    - iii. Ataxia
    - iv. PU/PD
    - v. Polyphagia
    - vi. GI upset: minimized by giving the medication with food or divide dose
    - vii. Uncommon
      - 1. Increased risk of pancreatitis
      - 2. Persistent cough
      - 3. Increased risk of megaesophagus
  - d. Dose
    - i. Dose: 15-30mg/kg
    - ii. Loading dose:
      - 1. 625mg/kg divided in eight or more doses over 48h
      - 2. 125mg/kg/day for 5 days

- e. Levels
    - i. 1-3mg/mL, widely available
  - f. Effectiveness/Personal Experience
    - i. I'm not a huge fan of this drug for most patients. I see relatively more annoying side effects with it, and the long half-life makes it difficult to react to changes quickly.
    - ii. That being said, for the flakey owner who can't remember to give meds, this medication is perfect.
    - iii. Don't prescribe for dogs that regularly go to the beach, as drinking the salt water can dangerously lower the KBr concentrations
    - iv. Similarly, dogs on this medication need to be loaded before being given a high rate of IV fluids, as fluids will rapidly decrease serum KBr levels.
7. Pregabalin aka Lyrica
- a. Proposed method of action: This is the next generation of gabapentin. Like gabapentin, it is also thought to act at the voltage gated calcium channel.
  - b. Metabolism
    - i. Organs: In humans, major excretion is unchanged in urine. However, the same can be said of gabapentin, which is known to be at least partially metabolized in the dog via the liver.
    - ii. T1/2: 7 hours
  - c. Toxicity/Side Effects
    - i. Sedation and ataxia (do not seem to adjust as dogs do to other anticonvulsants)
  - d. Dose: 2mg/kg q8h, wean up to 3 to 4mg/kg q8h.
  - e. Levels: Not available commercially. Human levels 2.8 to 8.2ug/mL. 2/11 dogs had levels lower than this in one study, but all other dogs in this study fell within the reported human level range.
  - f. Effectiveness/Personal Experience:
    - i. I have never used this drug due to cost and reported side effects.
    - ii. It is reported to be more effective than gabapentin for both pain and seizure control in humans.
    - iii. 7/11 dogs (9 completed the trial) responded in a recent study
8. Topiramate aka Topamax
- a. Proposed method of action: Increased GABA activity, inhibition of kainate/AMPA receptors
  - b. Metabolism
    - i. Organs: In humans 70% excreted in the urine unchanged. Some liver metabolism
    - ii. T1/2: 2-4 hours
  - c. Toxicity/Side Effects
    - i. Side effects reported in dogs: GI upset, inappetence, and irritability. In cats, sedation and inappetence noted.
  - d. Dose
    - i. Dogs: 5-10mg/kg q 12h reported, some question as to efficacy as the half life is 2-4 hours- may need more frequent dosing.
    - ii. Cats: 12.5-25mg divided q 8-12h
  - e. Levels: In humans, levels range from 2-25mg/L, but are not frequently monitored.
  - f. Effectiveness/Personal Experience
    - i. This has been studied in cat experimental model, but not in a clinical setting to my knowledge.
    - ii. There has been some pharmacokinetic and metabolism work done in the dog.

- iii. There are no clinical studies as to efficacy and I have not personally used this drug due to previous cost issues (Generic became available in 2009)

9. Zonisamide aka Zonegran

- a. Proposed method of action: Many mechanisms proposed, at least seven at my last count, including inhibition of sodium and calcium channels.
- b. Metabolism
  - i. Organs: 20% liver metabolism, the rest is excreted unchanged in the liver
  - ii. T1/2: 15 hours
  - iii. Activation of hepatic microsomal enzymes may lead to increased drug clearance
- c. Toxicity/Side Effects
  - i. Generally, side effects are very mild. Sedation and ataxia are the most common. Inappetance is the one I see most frequently, especially in picky small dogs and in cats.
  - ii. One report of liver disease in a dog also on high dose phenobarbital
  - iii. Can cause a renal tubular acidosis, which can lead to urinary stones
  - iv. Minimal side effects noted in dogs given 75mg/kg/day for 1 year.
  - v. Dose: 5mg/kg q12h as a starting dose.
    - 1. Dogs on drugs that increase hepatic enzymes (i.e. phenobarbital) should be started at a minimum of 10mg/kg q12h
  - vi. Levels: Human levels 10 to 40mcg/mL. Levels available through Auburn.
  - vii. Effectiveness/Personal Experience
    - 1. 7/12 dogs had a 50% or greater seizure reduction. Mean reduction in seizure frequency was 81.3%
    - 2. Another study showed 9/11 epileptics responded to therapy, with a median reduction of 92.9%
    - 3. I use this drug commonly as a sole anticonvulsant or as add on drug therapy. I find it particularly helpful in patients where we are trying to avoid side effects. Seems to work better in structural epilepsy than idiopathic epilepsy in my hands.

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# You Spin Me Round: Vestibular Disease

Carrie Journey DVM DACVIM (Neuro)

- Overview
  - a. Anatomy and Localization
  - b. Common Differentials and Treatment
    - i. Brief overview of diseases, diagnosis and treatments
    - ii. Most information regarding dogs, with some information about cats
- Neuroanatomy
  - a. Vestibular system anatomy upon first inspection can look a little complicated.
  - b. Knowing the relevant neuroanatomy is crucial to the ability to localize and therefore come up with a reasonable differential list.
  - c. I find it easiest to break it down in to two functional units
    - i. Middle ear: In addition to the auditory apparatus the middle ear contains:
      - 1. Facial nerve
      - 2. Vestibular apparatus: including the semicircular canals and vestibular portion of the vestibulocochlear nerve
      - 3. Post ganglionic sympathetic nerve fibers to the eye
    - ii. Brainstem
      - 1. The vestibular nuclei are in the cranial medulla, just lateral the fourth ventricle.
      - 2. The brainstem is packed with other functional systems such as:
        - a. The ascending reticular activating system which controls consciousness
        - b. The proprioceptive fibers
        - c. Other cranial nerve nuclei such as those that control facial sensation (CN5) and gag reflexes (CN 9-10).
      - 3. Portions of the cerebellum and cranial cervical spine are involved with the vestibular system. We will not be covering these aspects in detail in the interest of time.
      - 4. Remember the brain as a whole is confined in a rather small space, so forebrain problems can also result is referred problems in the brainstem due to herniation and fluid shifts.
- Horner's Syndrome
  - a. Loss of sympathetic innervations to the eye
  - b. Results in:
    - i. Miosis: pupil is smaller
    - ii. Ptosis: loss of eyelid tone
    - iii. Enophthalmus and elevation of the 3<sup>rd</sup> eyelid
  - c. Clinically HS can be seen with diseases affecting the middle/inner ear
  - d. Neuroanatomy:
    - i. Preganglionic fiber originates in the spinal cord C8-T2
    - ii. These fibers course cranially in the vagosympathetic trunk to the cranial cervical ganglion

- iii. The pathway from here is a bit fuzzy. Fibers may be located on the ventral aspect of the petrous temporal bone
  - iv. CN5 Ophthalmic is involved in pathway to the eye
- Symptoms of Peripheral Vestibular Disease
  - a. Mentation: disorientation is frequent- animals are trying to react to their environment, but their perception is that the room is spinning so it is difficult to react normally.
  - b. Posture: head tilt ipsilateral to the lesion
  - c. Gait:
    - i. Circling/Leaning/Rolling Falling ipsilateral to the lesion.
    - ii. Gait abnormalities tend to be more severe in peripheral disease
  - d. Cranial Nerves:
    - i. Nystagmus: horizontal or rotary, fast phase AWAY from the lesion. Speed of nystagmus tends to be faster in peripheral lesions.
    - ii. Positional ventrolateral strabismus ipsilateral
    - iii. Facial nerve paresis/paralysis ipsilateral:
      - 1. decreased palpebral
      - 2. decreased menace: some animals will still retract their globe and raise the 3<sup>rd</sup> eyelid.
      - 3. Lip/ear droop
    - iv. Horner's Syndrome: Ipsilateral miosis, ptosis and enophthalmus
    - v. Everything else is normal (gag, sensation, etc.)
  - e. Proprioception: Normal.
    - i. **\*\*\* This is a really important way to distinguish between central and peripheral disease. \*\*\***
  - f. Spinal Reflexes: Normal
  - g. Palpation: May hurt over the bullae (can look like cranial cervical pain)
- Symptoms of Central Vestibular Disease
  - a. Mentation:
    - i. Disorientation
    - ii. Alterations in consciousness: dull, obtunded, stupor, coma
  - b. Gait: In addition to vestibular ataxia, can also have cerebellar and proprioceptive ataxia
  - c. Posture: Head tilt to either side
  - d. Cranial Nerves:
    - i. Any cranial nerve sign possible
    - ii. Loss of sensation and gag are common co-morbidities
    - iii. Nystagmus:
      - 1. Can be horizontal, rotary and/or vertical
      - 2. Fast phase can be in any direction
      - 3. Can switch direction
    - iv. Strabismus
      - 1. Any type, any direction, static or positional
  - e. CPs: Deficits can be ipsilateral or contralateral to other signs
  - f. Spinal reflexes: normal to hyperreflexive
  - g. Hint's that it's Central

- i. **\*\*\*CP deficits\*\*\***
  - ii. Changes in mentation other than disorientation (i.e. stupor or coma)
  - iii. Sensation deficits
    - 1. Stick a pen in their nose- if they only react on one side it's central!
  - iv. Gag problems
  - v. Seizures
- Peripheral vestibular disease
  - a. Differentials Overview
    - i. Idiopathic
    - ii. Infectious
    - iii. Ototoxicity
    - iv. Metabolic
    - v. Masses/Neoplasia
    - vi. Trauma
- Idiopathic Vestibular Disease
  - a. Background
    - i. 39% of peripheral vestibular disease in dogs (Schunk and Averill 1983)
    - ii. Older dogs
    - iii. Uncommon but reported in all ages of cats
  - b. Theories
    - i. Endolymph production/absorption/drainage issue theorized
    - ii. Autoimmune disease theorized: never proven and steroids do not speed recovery.
  - c. Presenting Complaint: acute to peracute vestibular signs
  - d. Exam:
    - i. Purely peripheral vestibular
    - ii. Should not have facial paresis nor Horner's Syndrome
      - 1. We do occasionally see cases with multiple idiopathic abnormalities. It is my opinion that these should truly be a diagnosis of exclusion as it is far more likely that there is an underlying cause (i.e. otitis neoplasia, hypothyroidism, etc).
    - iii. No CP deficits
      - 1. Admittedly, many older dogs have CP deficits from chronic non-progressive spine lesions and then can develop idiopathic vestibular disease. There is no sure way to separate these CP deficits from CP deficits from central disease. Dogs with CP deficits should be assumed to have central disease until proven otherwise.
  - e. Treatment/Prognosis:
    - i. The only treatment is supportive care and time
      - 1. Consider anti-emetics: Ondasetron, Cerenia, Meclizine.
      - 2. Sedation when required
    - ii. 2-6 weeks to resolution
    - iii. Some improvement usually within 72 hours
    - iv. Some animals retain a head tilt
- Primary Secretory Otitis Media

- a. Aka “Glue Ear”: very tenacious mucous plug in middle ear
- b. Reported in Cavalier King Charles Spaniels
- c. Non infectious, not accompanied by otitis externa
- d. Usually causes pain
  - i. Often mistaken for or is a co-morbidity with Chiari malformation
  - ii. But can also result in vestibular and/or facial/horner’s signs.
- Otitis Media
  - a. Often secondary: 43/110 primary allergic dermatitis (Saridomichelakis, et al. 2007)
  - b. This is common!
    - i. 49% of peripheral vestibular disease (Schunk and Averill 1983)
    - ii. 16% of acute otitis externa cases (Gotthelf 2004)
    - iii. Up to 80% of chronic otitis externa cases (Gotthelf 2004)
  - c. Diagnosis:
    - i. In theory, you should be able to diagnose this via otoscopic exam, but unfortunately it’s not always possible to get a good otoscopic exam of the tympanum
      - 1. In one study the tympanum was only visible in 66% of exams. (Trower, et al. 1998)
    - ii. CT and radiographs have similar accuracy (Love, et al. 1995)
      - 1. However, we still consider advanced imaging important in many cases. This is most common when there is not obvious otitis media on otoscopic exam, or if there is some concern for central disease (i.e. CP deficits).
      - 2. When there is any question of co-existing central disease or the tentative diagnosis of otitis media is shakey, I strongly prefer an MRI.
      - 3. Excellent review of all three imaging techniques: Garosi et al. Review of Diagnostic Imaging of Ear Diseases in the Dog and Cat. Vet Rad and Ultrasound. 2003. 44(2): 137-146.
    - iii. Cytology and Culture: External Ear vs. Myringotomy
      - 1. The culture from the external ear and the middle ear are not always the same. However, I have spoken with dermatologists who will treat off of an external ear culture and only go to middle ear culture if treatment fails.
      - 2. *Malassezia*, Staphylococci, and *Pseudomonas* are most common (Cole, et al. 1998) (Oliveira, et al. 2008)
  - d. Treatment
    - i. Systemic and topical therapy in combination are recommended
      - 1. Antibiotics, antifungals depending on cytology and culture
        - a. >70% of bacteria isolated from dogs and cats susceptible to topical gentamycin and baytril in one study (Hariharan, et al. 2006)
        - b. However there is some evidence for growing resistance as only 18% pseudomonas susceptible to baytril in another more recent study. (Palmeiro, et al. 2004)

2. Steroids are often recommended at least initially to assist with inflammation that can lead to stenosis of the ear canal and treatment failure.
  3. Topical solutions: remember ototoxicity!
  - ii. Cleaning: More frequent ear cleaning is generally recommended.
  - iii. Flushing
    1. Successful in 82% of dogs (Palmeiro, et al. 2004)
    2. Mean time to resolution was 117 days
  - iv. Surgery
    1. VBO
      - a. Consider in cats due to polyps
    2. TECA
      - a. Success in 11/13 (12/14 ears) chronically affected dogs (Sharp 1990)
- Otic toxicity
    - a. If you don't know if there is an eardrum, be careful what you put down in the ear.
    - b. There are many toxins:
      - i. Chlorohexidine and aminoglycosides are commonly named ones, but far from the only ones.
    - c. Treatment:
      - i. Stop putting toxic substance in the ear, give it time- many will get better.
  - Metabolic: Hypothyroidism
    - a. Of 379 dogs that are hypothyroid, 29 had neurologic abnormalities.
      - i. 9 of those 29 had peripheral vestibular disease (Jaggy, et al. 1994)
    - b. However a recent prospective study of 9 dogs shows no evidence of peripheral nerve disease 18 months after radioactive thyroid ablation (Rossmeis 2010)
  - Neoplasia/Masses
    - a. External ear canal tumors
      - i. Can extend through tympanum
      - ii. Ceruminous gland adenomas and adenocarcinomas
        1. Most common
        2. Benign in dogs
        3. Malignant in cats
      - iii. Squamous cell carcinoma, basal cell tumors, papillomas, histiocytomas also reported
    - b. Primary middle ear masses
      - i. Cats
        1. Polyps
        2. Squamous cell carcinoma most common neoplasia
        3. Also fibrosarcoma, anaplastic carcinoma, lymphoblastic lymphoma, ceruminous gland adenocarcinoma
      - ii. Dogs
        1. Papillary adenomas and fibromas- benign, more common than malignant forms
        2. Cholesteatoma
  - Trauma

- a. Head trauma resulting in damage to petrous temporal bone
  - b. Often trauma is a mixed peripheral and central phenomenon. The force required to break the petrous temporal bone will frequently cause contusion and or hemorrhage to the underlying brainstem.
- Central Vestibular Disease
  - a. Differential Overview
    - i. Vascular
    - ii. Infectious/inflammatory
    - iii. Metabolic
    - iv. Neoplasia
    - v. Toxic
    - vi. Nutritional
    - vii. Trauma
  - b. Vascular
    - i. Classically characterized as either hemorrhagic vs. non-hemorrhagic
    - ii. Clinically, I group them as hypertensive vs. non-hypertensive
      - 1. 8/28 dogs hypertension in one study (Garosi, et al. 2005)
      - 2. I have seen this in cats too.
      - 3. I feel that in our practice we see a much higher incidence than that, which I didn't realize until we started checking blood pressure regularly.
      - 4. It can be difficult to sort out which pets are hypertensive due to White Coat Effect or the stress of their disease- however most dogs with clinical brain signs due to hypertension will have Doppler blood pressures over 180, often >200. These reading would be exceptionally high for stress related hypertension
    - iii. When speaking of vascular accidents as a whole concurrent medical conditions are reported in 18/33 dogs (55%) (Garosi, et al. 2005)
      - 1. Kidney disease (8/18)
      - 2. Cushing's disease (6/18)
      - 3. Diabetes mellitus
      - 4. Cardiac disease
      - 5. Hemangiosarcoma
      - 6. Pheochromocytoma
      - 7. Hypothyroidism reported in other studies. (Higgins, Rossmeisl and Panciera 2006) This is discussed at length further down in these notes.
    - iv. Diagnosis
      - 1. MRI +/- CSF
      - 2. Systemic workup:
        - a. BP, CBC/Chem/UA (+/- UP/C), T4/TSH
        - b. Coag Panel, Echo, AUS, LDDS: I usually reserve these test with dogs with suspect infarcts on imaging or confirmed hypertension.
    - v. Treatment/Prognosis
      - 1. Results from one study:

- a. 23/33 (70%) alive
    - b. 16/23 (70%) considered good to excellent recovery
    - c. 5/23 (22%) had recurrence 5 to 10 mo later. All had underlying conditions
  - 2. Personally I find given enough time and supportive care most pets will recover adequate function to be a happy pet in 3-4 weeks.
- c. Inflammatory Disease
  - i. Includes disease like granulomatous meningoencephalitis, necrotizing encephalitis, etc.
  - ii. Etiology is uncertain
    - 1. Despite extensive investigations no infectious agents found (Schatzberg, et al. 2005)
  - iii. Top differential in young to middle age small breed dogs with acute to peracute central signs
  - iv. Diagnosis:
    - 1. MRI
    - 2. CSF
    - 3. Infectious disease panel
    - 4. In theory a biopsy is required for definitive diagnosis, but this step is often skipped when other test are supportive.
  - v. Guarded prognosis
  - vi. Requires aggressive treatment with immunosuppressant therapy (prednisone, mycophenylate, cyclosporine etc) and sometimes chemotherapeutics (cytosar, CCNU).
- d. Infectious
  - i. Not as many cases around here
  - ii. Tick borne encephalitis
    - 1. Top differential with mild thrombocytopenia
    - 2. Very treatable- signs usually start to resolve on doxycycline within a day or two.
  - iii. Other infections
    - 1. Toxoplasma, FIP in cats
    - 2. Toxoplasma, Neospora, Distemper in dogs
    - 3. Cryptococcus possible in both. There is a strain of Cryptococcus creeping down the west coast- so this may be an emerging problem soon.
  - iv. Diagnosis:
    - 1. Blood titers for Toxoplasma, Neospora, Tick panel, +/- Cryptococcus (more accurate on CSF)
    - 2. Corona titer is of limited utility (as always)
    - 3. MRI/CSF can be supportive- especially important with FIP due to lack of serologic testing
  - v. Treatment
    - 1. The Good: toxoplasma, neospora and tick borne encephalities are very treatable
      - a. Toxoplasma/Neospora: Clindamycin

- b. Tick Borne Encephalitis: Doxycycline
  - 2. The Not As Good: Cryptococcus can be challenging to treat
    - a. Fluconazole is preferred treatment
    - b. Sometimes have to use amphotericin B.
    - c. Often start with a short anti-inflammatory course of steroids as there can be a huge inflammatory response in the brain when the fungus starts to die.
    - d. I strongly recommend hospitalization for the first week of treatment.
    - e. Generally a lifelong treatment (don't believe the book).
  - 3. The Bad: Distemper and FIP are not generally treatable (you can try steroids- they are a bandaid).
- e. Metabolic: Hypothyroidism
  - i. One study looking at purely central cases found that only 30% look hypothyroid (i.e. obese, alopecia, etc) (Higgins, Rossmeisl and Panciera 2006)
  - ii. 70% have hypercholesterolemia
    - 1. This may be an important part of pathogenesis. Cholesterol related vascular plaques are a known phenomenon in people, and have been reported sporadically in the dog.
  - iii. Treatment/Prognosis
    - 1. Thyroid supplementation
    - 2. 50% (2/4) improved in one study (Bichsel, Jacobs and Oliver 1988)
    - 3. 90% (9/10) complete resolution in another study (Higgins, Rossmeisl and Panciera 2006)
    - 4. Average time to resolution 4 weeks
- f. Neoplasia
  - i. Any primary brain tumor, skull tumor or metastatic tumor can affect this system.
  - ii. MRI +/- CSF required for diagnosis
  - iii. Prognosis and treatment depends on tumor type
    - 1. They can do better than you think!
- g. Toxins
  - i. Metronidazole is a very common vestibular toxin
    - 1. There is some evidence of dose relation
      - a. Average dose in toxicity cases in one study was 60mg/kg/day (Evan, et al. 2003)
      - b. I try to stick to 20mg/kg/day- no more than 30mg/kg/day.
    - 2. Onset of symptoms is usually 1 to 3 weeks after initiating therapy, but can occur in patients on it chronically
    - 3. Symptoms can be severe enough to warrant hospitalization (nonambulatory due to rolling/etc, nausea and inappetance)
    - 4. Treatment
      - a. Stop giving metronidazole

- b. Oral diazepam hastens recovery significantly (Evan, et al. 2003)
    - ii. Although not a frequently reported syndrome, I have seen several dogs with confirmed marijuana toxicity appear central vestibular with nystagmus etc in addition to or without the “classic” signs (disorientation, hyperesthesia, mydriasis, leaking urine, bradycardia, etc)
  - h. Thiamine Deficiency
    - i. Related to all fish diets in cats and overcooked food in dogs.
    - ii. Signs
      - 1. Acute and rapidly progressive
      - 2. Central Vestibular
      - 3. Obtunded
      - 4. Seizures
      - 5. Pupillary dialation
      - 6. No menace
      - 7. Cerebellar tremors and ataxia.
      - 8. Often have concurrent cardiac arrhythmias
    - iii. Treatment is thiamine
      - 1. 5-50mg/day in dogs
      - 2. 1-20mg/day in cats
    - iv. Good prognosis if caught early.

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# Top Marketing Mistakes Your Team is Making with Social Media

Cheyenne Flerx

[Hey Cheyanne, LLC](#)

Social media marketing can be a powerful tool for your practice. It can be leveraged well when you and your team manage and operate social media as an excellent business-generating asset.

However, knowing how to get your social media accounts to the point where they are this powerful asset can be tricky. This often leaves teams scrambling to figure out how to run their practice's social media accounts, leaving room for mistakes.

## **There are ten common mistakes I see practices making when it comes to their social media:**

- Not engaging with the audience on your social media posts
- Posting the same old type of posts without evaluating their effectiveness
- Posting to social media last minute
- Not proofreading your content before your post
- Not planning social media content ahead
- Forgetting to check the analytics and performance of your posts
- Not setting marketing goals and expectations for your marketing
- Not providing the proper support to those managing your social media accounts
- Not communicating well with the entire team
- Not continuing to learn and try new things about social media and digital marketing

## **Why are these actions ultimately hurting your practice's bottom line?**

When people look at your social media account, they are either evaluating to see if your practice would be a good fit to do business with you or decide if they want to be employed by you! People create an impression of your practice solely on your digital presence.

Your social media accounts, what you post, and HOW they are presented and managed speak volumes about your business. Whether people's impression of your practices matches reality, the

impression your social media (and your website, too) puts out plays a huge part in how you are generating clients and revenue!

The key thing to remember is that anything you post on your social media pages is perceived as a direct reflection of how you operate, practice medicine, and what your practice values.

Therefore, you attract the clients that align with what your practice embodies and supports, and your social media can be that avenue that can genuinely attract your ideal clients and repeal the clients you don't want to serve. But you must be aware of what you are posting and be strategic with your posts and how you show up on social media.

### **Steps to help you move forward:**

1. Use the list above to reflect on your team's current situation, output, and where your team needs to improve with your social media accounts.
2. Make a note for each mistake you see on the list, then write your desired outcome.
3. Then create a list of things you can do to improve for each mistake. Create your list with the thoughts of the desired outcome you want to achieve in mind. This list will serve as your next action steps and help you see the gaps you can close and reach the desired results!
4. Take action!

### **Suggested Resources:**

- My recommended software and tools for managing social media: <https://www.heycheyanne.com/fav-tools>
- Free templates to help plan social media posts and more: [www.heycheyanne.com/freestuff](http://www.heycheyanne.com/freestuff)



## Giving Engaging Team Meetings

Presented by Debbie Boone, CVPM

**Managers typically dread staff meetings. So do teams....**

*I once asked a group of students about staff meetings, and one young woman said, “they bring us in, yell at us . Send us back to work. “I certainly don’t blame anyone who is the subject of this torture to want to avoid staff meeting participation.*

Team meetings can be either fun, engaging, and productive — or they can be filled with complaining and boredom. You can’t expect that every meeting will be a cheerleading session. Yet, you can learn how all can be beneficial to the success of the practice, the growth of the team, and the care of our patients.

Step 1 . Build a good agenda and get it out to the team ahead of time If you plan to have team participation in growing your hospital you have to give people time to think about what needs to be done. Without some lead time they can’t come to the meeting with their own unique ideas. Giving people time to consider the work to be tackled will result in better engagement.

You can also solicit items for the agenda from the team.

Here is my Framework for meetings:

- Share Good Stuff – Storytelling and Spotlighting
- Discuss issues-but don’t point fingers, instead team problem solve
- Learn something -bring in outside trainers or learn from peers
- Set goals – not necessarily financial but still measurable

Remember the purpose of team meetings is to build a “team” of people all pulling together to create a positive outcome. It is also to share updates and be transparent so no negative undercurrents can pull down the group. They are to design “systems” that work for the group and to break down systems that cause frustration and stress. This is why team activities work.

**Storytelling** creates community.

*“It keeps you from a silo mentality, or thinking you are the only one getting anything done.*

*It gives you a reason to celebrate what is happening all across the organization.*

*It gives you hope and reenergizes your vision when your team may be going through a*

*tough season.”<sup>1</sup> **Spotlighting** allows us to shine a light on one of our team and learn about their life. Then we can share our appreciation for them and the value they bring to the team. Start with a simple question and answer session. Things like – where did you go to high school? What is your*

favorite food? Do you have a hobby? What kind of music do you love? These questions show how similar we are and help us connect with each other. Then you can ask team members to share what they admire about this person and what value they bring to the practice.

Recognition of good work is one of the keys to team retention. So is autonomy. Being in the “know” and having a safe space to be are others. Team meetings can help develop all these when used well.

Team meetings are places where we share plans, challenges and insights. Leaders work with groups to accomplish the goals of the business. Poor management hoards information and limits growth and stifles creativity of the group. They believe they are there to provide answers to the problems when they should be there to listen to solutions from the people actually doing the work.

Before you can have engaging team meetings your staff must feel they are a safe place to launch an idea without being dismissed or shot down.

When groups decide how to create a system then they are more likely to abide by the changes rather than a top-down dictation.

Foundational work on core values, mission statement and other behaviors are best discussed with the team in a “how do we manifest these in daily life” rather than just some impractical and unobtainable ideal. Most importantly they have to have **meaning** to the group.

Good Icebreakers – Story telling, Let’s Make Toast exercise, How Do We Live This – core Values exercises



## **You Said WHAT?! Training Your Team How to Say Things**

### **the RIGHT Way**

#### **Presented by Debbie Boone, CVPM**

OMG! Some of the things people say to our clients and each other are unbelievable! Yet, the speaker is often clueless about the word choice that set the person they were speaking with "on fire." Language is subtle and nuanced. Choosing the correct word at the right moment takes planning and training.

Today's public is skeptical. Lack of trust continues to escalate with every news story, conspiracy theory, and political campaign. Establishing trust with our clients is the key to compliance and practice success. Not to mention, that it also improves our work experience when we are not engaged in a battle with mistrustful clients on a daily basis.

I firmly believe that pushing information out to clients with the correct wording helps us avoid conflict. Sharing information with our team is equally important.

**Words are powerful tools that move our emotions to joy, sorrow, anger and glee. One challenge is the literacy of our clients and our staff.**

[Know your audience!](#)

**Half of U.S. adults can't read a book written at the 8th-grade level.**

— Organization for Economic Cooperation and Development

When we keep this in mind along with the fact that people are emotion driven rather than fact driven, we can make better word choices to reach them.

*"to convince skeptics, we must first learn how they view the world and accept that view is valid. From that position we can engage them and persuade them to listen to what we have to say. "*<sup>2</sup>

Understand the best way to train team members how to gracefully set client boundaries without making them angry is to teach them how to stand in the client's shoes, listen intently and communicate with empathy.

Example: When we say, "I am sorry, I don't have an open appointment for 2 weeks." We are putting negative messaging out. When we say, "Mrs. Jones, I am happy to help you. Our first appointment is

October 1. Will that work for your schedule? “ We are starting on a positive note and offer the exact same thing.

George Bernard Shaw wrote: *“The problem with communication is the illusion that it has been accomplished.*

*Recommended books: Crucial Conversations by Patterson, Grenny, McMillian and Switzler*

*The Language of Trust by Michael Maslansky.*



## Listening With More Than Your Ears — It's All About the Body

**Presented by Debbie Boone, CVPM**

At just two days old, babies can read the body language of their moms. When did we lose this intuitive skill? It's time to recapture it by learning how to interpret physical cues that provide insight into what others are really thinking and even if they're being truthful. It's vital to listen intently with focus and watch others with equal focus.

## bod·y lan·guage

[ˈbädē ˌlaŋgwiː]

NOUN

1. the process of communicating nonverbally through conscious or unconscious gestures and movements:

Non-verbal messages including body movements, facial expressions, vocal tone and volume, and other signals are collectively known as body language.

Microexpressions (brief displays of emotion on the face), hand gestures, and posture all register in the human brain almost immediately—even when a person is not consciously aware they have perceived anything. For this reason, body language can strongly color how an individual is perceived, and how he or she, in turn, interprets others' motivation, mood, and openness. It's natural to mirror; beginning as soon as infancy, a newborn moves its body to the rhythm of the voice he hears.

A common form of body language is mirroring another person's gestures and mannerisms; mirroring also includes mimicking another person's speech patterns and even attitudes. When you want to [persuade or influence](#) a person, mirroring can be an effective way to build rapport.

Tilting the head exposes the neck, and perhaps shows vulnerability. The person with a tilted head is perceived as more interested, attentive, caring, and having less of an agenda.

Eye blocking, or covering your eyes, expresses emotions such as frustration and worry.

Touching or stroking the neck signals a pacifying behavior. We all rub our necks at the back, the sides, and also under the chin. The fleshy area under the chin has nerve endings and stroking it lowers heart rate and calms us

Rubbing the hands conveys stress, steeping the fingers means that a person feels confident. When you feel confident, the space between your fingers grows

A light touch on the arm conveys harmony and trust.

Standing crossed legs will help you say that you are comfortable with the other person.

Fidgety hands mean anxiousness or even boredom and keeping your arms akimbo may telegraph arrogance. sitting with open arms invites the other person in. If you are sitting and want to appear neutral, it's best to hold your hands on your lap,

Crossed arms can signal many other things, including anxiety, self-restraint, and even interest,

Freezing in place, rocking back and forth, and contorting into a fetal position are all known as "reserved behaviors," as they are used only when a person experiences extreme stress. Facial expressions alone can signal this state, such as pursing or sucking in the lips, often seen when a person is upset or feels contrite.<sup>3</sup>

Recommended Reading: What Every Body is Saying by Joe Navarro <https://amzn.to/3R0pew2>

# Not your Typical Talk on Antimicrobial Resistance

## Deborah Thomson, DVM

September 16, 2022  
3:50-4:40pm

This highly applicable interactive lecture is designed for clinicians who want to be good antibiotic stewards but simply don't have the time to read the recent literature and do an in-depth review of newly available technologies.

During this time together, learners:

- (1) Understand the mechanisms behind Antimicrobial Resistance;
- (2) Learn methods to communicate the importance of Antimicrobial Resistance to clients; and
- (3) Become familiar with currently available veterinary diagnostic tools.

# Communication, One Health, and You

## A Workshop in 2 Parts

Deborah Thomson, DVM

Friday, September 16, 2022

10:00 am – 10:50 am

11:00 am – 11:50 am

### Overview:

*Ever come across a difficult client? Want to increase the number of times clients agree with your treatment plan? The key to increasing this number is to tailor your message to the individual family. Communication is an art that is not thoroughly taught in veterinary school. By participating in this workshop, you can (1) no longer fear difficult client conversations, (2) practice One Health in unexpected ways during your daily clinical shifts, and (3) increase the amount of client consent to your diagnostic plans.*

### Description:

This interactive series is designed for veterinary general practitioners, specialists and staff to win over challenging clients. This 2-part communication series includes masterclass-style teaching as well as small group work and role playing. Being an efficient clinician really comes down to practicing communication techniques in a safe setting before getting "back on the horse" in the clinic. Bring your pen and paper, sense of humor, and questions!

## What you *really* want to know about Canine Cushing's Syndrome

Heather L Kvitko-White, DVM, DACVIM

8.18.22

Having a high clinical suspicion for Cushing's Syndrome means that we have evaluated multiple data points and recognized a constellation of anticipated exam findings, lab work changes, and clinical signs. Remember that the signalment, history, and physical examination findings are some of the most important (*and free*) pieces of data available.

### *Signalment*

Spontaneous CS is a geriatric disease most commonly seen in small breed dogs around 10 years old. It is nearly unheard of in dogs <4 years old. In *general, CS is uncommon in the large breed dog even though they are overrepresented in the cases caused by an adrenal tumor.* Large breed dogs get PDH as well.

### *History*

Dogs with CS are rarely ill and should not have signs of illness (anorexia, vomiting, diarrhea, sneezing/coughing) at the time of workup.

The most common clinical signs of CS include the five "Ps" polyuria (PU), polydipsia (PD), polyphagia (PP), panting, and pot-bellied appearance (hepatomegaly and weakened abdominal girdle). Additional clinical signs include skin and hair-coat changes such as alopecia, thinning of skin, recurrent pyoderma, and calcinosis cutis as well as broader symptoms of muscle wasting and weakness (difficulty ambulating, jumping in the car, splaying out on slick surfaces) and a predisposition to recurrent infections. Insulin resistance and diabetes mellitus may occur 15% of the time.

Remember that clinical signs, while often "typical" are slow in onset and progression. Dogs rarely all have all the clinical signs at once and the clinical signs could be mild. For instant skin and haircoat changes alone.

Minimum database (CBC/Chemistry/UA)

***Cushing's should rarely be pursued because of the results of routine serum biochemistry,*** none-the-less the routine minimum database is an important screening test for CS.

Dogs confirmed to have CS have a constellation of lab work changes that are characterized by liver enzyme elevations, thrombocytosis, a stress leukogram (neutrophilia, lymphopenia, eosinopenia) and hyposthenuric urine. For instance, it is rare for the urine to be concentrated >1.020 in a dog with CS, although it may be isosthenuric if they also have CKD. One study found that a platelet count <450,000 was a very sensitive screening test to rule *OUT* CS.

The typical "pattern" of liver enzyme elevations includes an ALP elevation with elevated GGT, cholesterol, and triglycerides. The patient should not be icteric and T. bilirubin should be normal. ALT may be normal or mildly elevated (far-less so than the ALP). Additional albeit less specific findings might include elevated phosphorous, slight hyperglycemia, low BUN from medullary washout secondary to PU/PD, and importantly hyperkalemia which is actually a spurious test result which happens in blood samples packed with too many platelets.

Both hypertension and clinical proteinuria may be seen in dogs with CS, thus, if a combination of lab work findings suggests CS that patient should have a blood pressure performed and a urine protein: creatinine ratio assessed, assuming the urine sediment is inactive. Dogs with CS may be predisposed to bacterial cystitis and a urine culture might be considered as part of initial workup. However, routine urine culturing is not recommended in dogs with stable treated CS unless there are clinical signs of lower urinary tract disease or a significant change in health.

*If, based on signalment, history, physical examination and lab work the clinical suspicion of Cushing's syndrome is low, further testing is not recommended.*

If, based on signalment, history, PE, and lab work findings the clinical suspicion of Cushing's Syndrome is high, a low dose dexamethasone suppression test is recommended as the diagnostic test for CS.

*Serum Cortisol Assays*

Tests of serum cortisol in veterinary medicine are imperfect. First, cortisol levels naturally and sporadically fluctuate throughout the day in healthy pets, a reality that complicates determining even the normal reference ranges. Results between different assays at different laboratories cannot be directly compared.

Samples for cortisol measurement should be non-hemolytic, non-lipemic, and from serum that was spun off the blood and refrigerated or frozen within 1 hour. The patient does not technically need to be fasted for cortisol measurements however the risk of obtaining lipemic serum typically dictates a need for a fasted sample.

Any test that measures cortisol also measure nearly all other glucocorticoids (*\*except dexamethasone*).

Some helpful tips regarding glucocorticoids and cortisol testing

1. Patients receiving short-term glucocorticoids (< 1 –2 weeks) must have them discontinued for at least 72 hours before testing.
2. Patients receiving chronic (> 2 weeks) glucocorticoids, *including dexamethasone*, must have them tapered and discontinued for at least 3-4 weeks before cortisol testing.
3. Patients in need of treatment with glucocorticoids prior to cortisol testing should be treated with **dexamethasone** and testing should be performed ASAP\*

\* Applies only to Addisonian suspects. Dogs with CS should not be sick at the time of diagnosis.

#### *Low Dose Dexamethasone Suppression Test (LDDST)*

A LDDST is performed by measuring a basal cortisol and then administering 0.01mg/kg dexamethasone IV and measuring cortisol at 4 and 8 hours. A normal dog will respond to exogenous steroids by suppressing natural cortisol production and a normal cortisol 8 hours after dexamethasone should be low. Measuring the cortisol at 4 hours does not impact the diagnosis, however, it may be useful to differentiate ACTH dependent Pituitary Disease (PDH) from an Adrenal Tumor (AT). Suppression >50% of baseline is consistent with PDH while lack of suppression at 4 hours does not offer any additionally useful information.

When our clinical suspicion is high an abnormal LDDST provides support to the question “is there evidence of Cushing’s?” Yes. The diagnosis is made. When the suspicion is high, but the LDDST is normal we do not accept the result. The next step is to ask the same question in a different way.

### *ACTH Stimulation Test*

The ACTH stimulation test has the highest diagnostic specificity (ability to rule the disease *IN*) even though it also has a relatively high false positive risk. Thus, typically the ACTH stimulation test is reserved for use when, despite a high clinical suspicion, the LDDST returns normal.

However, in dogs with uncontrolled diabetes suspected of having CS the ACTH stimulation test *IS* the diagnostic test of choice. Here we do not want to risk missing the diagnosis because the potential consequences of poorly regulated diabetes can be fatal. In order to avoid the trap of a false positive the clinical suspicion for CS in the diabetic pet must be strong.

Indications of possible CS resulting in poorly controlled diabetes include...

- a. Insulin dose >1 unit/kg
- b. Persistent PU/PD/PP despite well controlled glucose
- c. Persistent PP despite controlled PU/PD
- d. PE findings consistent with CS but not diabetes (such as comedones, thinning skin, and potbellied appearance)

If the results of the ACTH stimulation test mimic Addison’s disease, meaning the cortisol is <2 at baseline and after ACTH administration, the findings are consistent with Iatrogenic Cushing’s. Additional evaluation into the source of glucocorticoids is recommended. Consider all sources including ophthalmic, topicals, inhaled, and budesonide.

### Abdominal Ultrasound

Differentiating between PDH and AT is important when the owner will pursue definitive therapy, namely- surgery. For an adrenal tumor, the most definitive therapy is adrenalectomy. For PDH, a hypophysectomy offers the only chance of a cure.

Regardless, an abdominal ultrasound is typically part of the workup for Cushing's disease as these are geriatric patients often with multiple comorbidities at the time of diagnosis. In skilled hands the adrenals can be reliably evaluated and used to help differentiate PDH from AT.

In PDH the adrenals are usually symmetrically enlarged and continue to grow overtime (even despite treatment with trilostane. Old dogs often have adrenal tumors on ultrasound- many of which are incidental- and therefore asymmetric adrenal enlargement is possible with PDH. "Normal" adrenal thickness is dependent on view and measuring and is relative to the body size of the patient making it nearly impossible to determine just one set-point for and adrenal being borderline vs too big. Adrenals may fall within guidelines of "normal" depending on sources.

When CS is due to an adrenal tumor assessment of the contralateral adrenal gland is crucial as it should be *small and atrophied*. Rarely cases have PDH and AT complicating everything.

The most common pitfalls in abdominal ultrasound for CS in dogs include studies that do not identify one or both adrenal glands, studies that have structures measured that are not the adrenal gland, studies lacking sufficient detail such that the structure cannot be confirmed to be the adrenal gland, and studies that lack evaluation of other important organ systems involved in CS such as the liver, gall bladder, and urinary tract.

Endogenous ACTH (eACTH) can be measured to help differentiate PDH from AT in dogs. A very labile test this has previously been limited to practices with advanced laboratory services on site. While adding eACTH measurement to ultrasound did not outperform ultrasound alone to differentiate PDH from AT, measurement of eACTH could be clinically useful in situations where ultrasound is not available. A new cageside diagnostic assay has been newly released to the veterinary market. The finding of low eACTH in a dog with diagnosed CS would support an adrenal tumor as the cause as long as you can trust your test results. *A low eACTH cannot be used to diagnose Cushing's disease.*

## **Treatment**

CS is nearly always a tumor and, as with many other types of cancer, the most definitive treatment is surgery. For the practical and financially able client- referral for surgery may provide the best long-term value (i.e. cost relative to outcome and client experience).

None-the-less, I have found that most of my clients forgo the path ending in surgery. In these cases, a common question posed is- should we treat at all?

My answer is nearly always yes. Untreated CS results in significant progressive morbidity and dogs who are being treated for Cushing's have higher quality of life scores than dogs who are not treated. With a median survival time of ~500 days when untreated most dogs do not die spontaneously of Cushing's disease. 80% of dogs with CS are euthanized due to clinical signs or complications of the untreated or poorly regulated process.

Examples include protein losing nephropathy, systemic hypertension, pulmonary thromboembolism (PTE) and pulmonary hypertension, secondary infections (dental, urinary, respiratory, skin and anal sacs, uveitis), cruciate ligament injuries, uroliths, gall bladder mucocele, and diabetes. Many of these complications are prevented or resolve with treatment although proteinuria and hypertension may persist requiring specific concurrent therapy. There is no direct treatment for calcinosis cutis which resolves over time (months) via the normal process of skin renewal. Secondary pyoderma and pruritus and/or non-pruritic demodex may need to be managed with oral and/or topical antibiotics and/or antiparasitics.

The complications of un-treated or poorly controlled CS occasionally result in critical illness such as massive PTE, bile peritonitis secondary to mucocele rupture, sepsis, and diabetic ketoacidosis. In these situations, diagnostic and treatment efforts must be directed at the specific disease process causing illness and not at CS. Trilostane must never be given to the sick pet and attempts to diagnose CS should wait until the patient has fully recovered from illness, at least 2-3 weeks later.

My treatment of choice is name brand Vetoryl<sup>®10</sup> even though I freely admit that I have almost no experience with mitotane which may still be a better choice for some adrenal tumors. Trilostane, the active drug within Vetoryl<sup>®</sup> is an enzyme inhibitor which blocks the pathway responsible for producing cortisol (and other glucocorticoids). Because this effect relies on normal adrenocortical cells to respond to the drug, some adrenal tumors may respond poorly to enzyme inhibition.

Studies have shown that compounded trilostane lack consistency and reliability, enough so that in my opinion the potential cost-saving benefits do not make up for the lack of or unreliable control that may result in general or from batch to batch. Only trilostane suspended in cod-liver

oil has been studied with all other varieties of oral suspensions having unknown absorption, stability, and efficacy characteristics.

The literature overwhelmingly points towards Vetoryl® being a twice or even three times daily drug. Where I once might have looked at once daily therapy for cost savings, I now recommend putting the money into treatment and being cost conscientious elsewhere (see the section on monitoring below).

I typically start a dose of 1-2 mg/kg every 12 hours which is generally rounded up or down based on capsule size. Consider the lean body mass or ideal body weight of the patient in your calculations. If you are uncertain or uncomfortable doing so, I recommend using the low end of the dose range.

Due to cost, I do my best to avoid doses that require multiple capsule sizes twice daily. This may mean a different dose in the morning than in the evening- for instance 50mg PO once daily in the morning and 30mg PO once daily in the evening versus 40mg (one 30mg capsule and one 10mg capsule) twice daily has a potential cost savings of over \$100/month.

### **Monitoring:**

Perhaps the most used monitoring protocol is published by Dechra, the makers of Vetoryl® although there are many variations on “target” cortisol and, as mentioned, the results of ACTH stimulation do not appear to correlate to clinical control.

***In my opinion, the most important emerging topic in canine CS is that routine ACTH stimulation monitoring is falling out of favor.***

First, research has failed to show that the ACTH stimulation test can predict an iatrogenic Addisonian crisis. The incidence of iatrogenic Addison's disease has proven to be low (15%) and 3 of every 4 cases were temporary. The risk did not appear to be related to the Vetoryl® dose and occurred anywhere from 5 days to 4 years after initiating therapy.

Further, the cost of synthetic ACTH cannot be ignored. In Europe, synthetic ACTH has become so limited and cost-prohibitive that the European labeling for Vetoryl® has been changed to use the pre-pill cortisol; a single cortisol measurement obtained just before the pet's morning dose is given. This test, like all tests of cortisol, are unreliable in sick and stressed dogs. A pre-pill cortisol is most helpful to confirm your clinical impression that the patient is doing well.

*As a result, consensus for the best monitoring protocol is still lacking amongst experts, although it is generally accepted that the goal should be a healthy dog and a happy owner.*

Presently, I perform an ACTH stimulation test about 30 days after starting a patient on Vetoryl®. This is probably unnecessary although I like to document a baseline (especially if diagnosed with a LDDST), potentially note an initial effect (ACTH stim improved from time of diagnosis), and screen for possible drug sensitivity (such as a dramatic response to typical doses early on).

In my experience the effects of Vetoryl® ramp up over time and I rarely increase the dose at the first recheck. At 60 days, if clinical signs persist, a dose increase is indicated and a recheck is scheduled in 30 days. In my hands dose increases are typically directed by the capsule sizes. The overall management goal should be to achieve a physical examination that is NOT consistent with CS meaning that the pets pot-bellied appearance, alopecia, and hepatomegaly have resolved.

If trends on routine lab work, clinical signs, or cortisol measurements indicate possible Addison's disease trilostane should be discontinued then resumed several days later at a lower dose. In a clinically healthy patient, a post-ACTH cortisol <1 mg/dL is not necessarily an indication to reduce the dose. If lab work is not suggestive for Addison's then the current dose might be maintained or an additional ACTH stim performed later that same day.

## **What you *really* want to know about diabetes**

Heather L Kvitko-White, DVM, DACVIM

Updated 8.18.22

Diabetes is generally straightforward to diagnose in both dogs and cats though management can seem overwhelming. Unlike many other chronic internal medicine diseases, a thorough understanding of the pathophysiology is not necessary for effective management.

### **Diagnosis**

In dogs, diagnosis is as straightforward as finding hyperglycemia on a portable glucose monitor or chemistry analyzer. If lab work is to be sent out to a reference laboratory, and diabetes is suspected, an in house blood glucose (BG) should always be performed. This will prevent a delay in diagnosis. Dogs do not have “stress” hyperglycemia although dogs occasionally have transient glucose elevations without being diabetic. I see this most commonly with concurrent pancreatic or hepatic disease.

In cats, diagnosis is more complicated due to stress hyperglycemia. BG elevations are typically seen during even routine wellness examination and thus a single BG measurement is not adequate to confirm diabetes. A cat’s BG may become elevated >400mg/dL and even an elevated BG combined with glucosuria (+/-) ketonuria is not adequate to confirm diabetes. A serum fructosamine must always be performed for confirmation. The definitive cause of “stress” hyperglycemia is not known in cats though it is most-likely due to a temporary surge of counter-regulatory hormones resulting in decreased insulin effect (ie insulin resistance).

### **Insulin**

After diagnosis insulin therapy should be started immediately to avoid further complication such as diabetic ketoacidosis (DKA). No change in diet, oral medications, or combination of the two will treat diabetes in either species. Do not delay insulin until referral, even if your plan is to refer.

Insulin is categorized by the anticipated effect or behavior. Short acting insulin (Humulin-R®, NPH), intermediate acting insulin (NPH, Vetsulin®, ProZinc®), and long-acting basal or

“peakless” insulins (detemir, glargine). Insulin should never be compounded. There are many other insulins that are not covered here but can be used in veterinary patients.

Insulin used in veterinary medicine comes in either human standard strength of 100 units/mL or veterinary exclusive strength of 40 units/mL (**Table 1**). Some human insulin is stronger (300-500 units/mL). Those products (such as Toujeo®) are still being evaluated for veterinary medicine. Only the appropriately sized insulin syringes should be used with the insulin. U-100 syringes have orange caps and U-40 syringes red caps. Attempting to convert the insulin to the syringe increases the risk of error and is strongly discouraged. Syringes are typically available in 0.3cc and 0.5cc sizes and may contain ½ or 1-unit markings. Some insulins (Vetsulin®, glargine, Levemir®) is available for use in an insulin-dosing pen. These devices have improved dosing accuracy over syringes and may be easier to use for clients with dexterity issues or needle phobia. For the most part, they do not come with the ability to administer ½ units. Pen needles must be prescribed at the same time. For the most part needles should be ½” in length however smaller pen needles are available and might be considered in certain cases. Alternatively, you can withdraw the insulin from the rubber stopper of the dosing pen. The only benefit to using a pen this way is usually to save cost. Savings are best if the pharmacy will dispense one pen at a time and veterinary pharmacies are often more likely to do so.

There are many factors to evaluate when choosing insulin for any specific pet. It is nearly impossible to predict which insulin will be the best fit for a patient although the majority of veterinary internal medicine specialists recommend insulin glargine for cats. In dogs, insulin choice should factor in size of the pet, owner commitment and ability for monitoring, individual owner and patient factors such as needle-phobia, ability to change diet or feeding schedule, the potential impact of a pet remaining hyperglycemic or becoming hypoglycemic, and long-term (not just short-term) cost.

Typically I start with Vetsulin® for most small dogs, detemir (name brand Levemir®) in larger dogs, and glargine (Lantus® or biosimilar Semglee®) in cats. Novalin-N® (the generic form of NPH insulin) is a reasonable choice in small to medium dogs due to anticipated cost-savings, however, given that the overall duration of effect is short (4-10 hours) I personally try to avoid it. As the cost for Humulin-R® (name brand NPH) exceeds other typically longer acting insulin, I almost never prescribe Humulin-N® in my patients. None-the-less, other internists note experiential differences in clinical control when comparing Humulin-N® and Novalin-N® therefore a switch from generic to name brand may still be reasonable if costs are not a concern.

Prescribe insulin to an estimation of lean body weight. Cats are typically between 5-6 kg, even in the most obese patients. If that makes you nervous, use 6.5 or even 7 kg. In dogs, use your best judgment, body condition score, and breed standards.

**Table 1: Insulin for standard veterinary use**

Insulin	Regular	NPH	Porcine Zinc	Protamine Zinc rH	Insulin glargine	Insulin detemir
<b>Name</b>	Humulin-R®	Humulin-N® Novalin-N®	Vetsulin® Caninsulin®	ProZinc®	Lantus® Basaglar® Semglee® *Toujeo®	Levemir®
<b>Duration</b>	Short	Short / Intermed	Intermed	Intermed	Long	Long
<b>Strength</b>	U-100	U-100	U-40	U-40	U-100 *U-300	U-100
<b>Dose</b>	0.1-0.2U/kg PRN or CRI	0.25-0.5U/kg q8-12h	0.25 - 0.5U/kg q12h	0.2-0.7 U/kg q12h in cats  0.5-1U q24 in dogs	0.25- 0.5u/kg or  1 unit per cat q12	0.1U/kg q12h in dogs 1 unit per cat q 12 hours in cats
<b>Use by</b>	30 days	30-60 days	42 days	60 days	~ 6 mos	~ 6 mos
<b>Cost/U</b>	~\$0.01	~\$0.03 – 0.15	~\$0.04	~\$0.25	~\$0.02- 0.18	~\$0.03- 0.30
<b>Notes</b>	Used for DKA or as an adjunct to long-acting insulin	Not for use in cats due to short duration	FDA approved dogs and cats  *Must be shaken	FDA approved in cats and dogs	No studies using the generic basaglar though anecdotal y similar	Difficult to dose in small dogs  In cats effect is similar to glargine

## Diet

Most newly diagnosed diabetic pets would benefit from some type of diet change at the time of diagnosis. In obese pets a weight-loss diet may be more appropriate than a prescription diabetic diet. In fact, both obese and cachectic diabetic dogs and cats may need higher protein content than what is provided in a typical prescription diabetic diet. Patients with concurrent comorbidities are often fed based on the needs of the other disease process. For instance, pets with concurrent chronic kidney disease may need a lower protein content than a standard diabetic prescription diet. In some cases, a specific over-the-counter food might be more appropriate than any prescription diet. A nutrition

consultation is an invaluable resource to any diabetic pet - especially those with comorbidities which include obesity and cachexia.

None-the-less, in a poorly controlled diabetic pet that is still eating a low-quality commercial diet, a change to a higher quality diet (i.e. prescription) may be the change necessary to improve control.

## Monitoring

Monitoring the pets' response to insulin is a critical part of managing a diabetic. In my experience the most common cause of poorly regulated diabetes is mismanagement. Sometimes the insulin is not the right match for the patient but most commonly the insulin has been adjusted using spot check BGs or based only on clinical signs without objective monitoring. In these cases it is easy to increase the insulin too rapidly and too high such that many pets are being overdosed by the time they are referred.

A blood glucose curve evaluates the response to insulin over time and consists of the BG just before the first meal and dose of insulin and then well-timed BGs throughout the rest of the day until the next dose of insulin is due. For intermediate acting insulin BGs are measured every 2 hours. For long-acting insulin BGs may only be measured every 3 hours due to the slower change rate throughout the day. Practically speaking, since most clinics are not open for 24 hours a day, the BG curve can stop once we have seen two points on the upswing of the curve which helps confirm that we have hit the nadir (ie the lowest glucose of the day) and that the BGs are now steadily increasing (**Image 1**).

Spot glucose checks are never appropriate for adjusting the insulin dose. When we choose a time to perform the spot BG check, we have made an assumption about when the nadir will be. Unless we have established a large data set for that patient and that insulin, it is impossible to predict when the nadir might occur. Most of the time, the nadir does not occur at exactly 6 hours after the dose. As already discussed, NPH is often quick to effect and short acting resulting in a nadir within 1-3 hours of the dose. When spot BG checks are performed in patients on NPH insulin the typical response is to continue to increase the insulin dose again and again because the spot BG checks do not reflect that the patient is becoming hypoglycemic at their true nadir at 10am (**Image 2- purple line**).

### Image 1: Blood Glucose Curve

A glucose curve is necessary to understand if the insulin is working, to estimate how low the BG gets (the nadir), how quickly after insulin administration is the nadir reached, the total duration of the insulin effect, and the average glucose throughout the day. A perfect BG curve is rarely achieved, however, for twice-daily insulin the nadir should occur around 6 hours and result in a BG between 80-120mg/dL. The total duration of insulin effect should be a BG < 250 for 10-18 hours of the day with average BG throughout the day of 150-200mg/dL.

The most typical “poorly controlled” BG curves result when the insulin dose is too low and the entire curve is shifted upwards, too high and the curve is shifted down, the insulin does not last long enough (checkmark shaped curve), insulin lasts too long (BGs still declining at the end of the day) or the insulin appears to have no effect at all making a flat-line. **(Image 2)**. The vertical line indicates the glucose value one would get across the various curves at a single point in time 6 hours after the morning dose of insulin. Note that this value is essentially the same across three different curves- insulin dose is too low, insulin does not last long enough, and insulin is lasting too long.

### **Image 2: Typical normal and poorly controlled BG curves**

Importantly, in pets receiving “peakless” insulin there may not be a nadir at all, or the nadir may occur just at the time the next dose of insulin is due. This can be alarming to the practitioner with less experience with these longer acting insulins and cause concern that the pet may become overdosed, however, it is important to remember that the next dose of insulin will work equally slowly meaning that an overlap causing hypoglycemia is not likely. In cases receiving intermediate acting insulin that have not yet reached the nadir before the clinic closes a 24-hour curve is indicated. Insulin duration of effect >18 hours indicates a patient who might benefit from once daily insulin. Though a minority of pets can be treated once daily to good control, in these cases, overlapping doses of insulin of long-duration does become problematic.

Many pet owners are willing and able to perform glucose monitoring at home. There are numerous resources available to help teach a client to do so using a portable glucometer (PGM) and ear or paw pad pricks with a lancet. Instruct the client to purchase the alpha-trak2 system as many studies have shown the results from human PGMs to be inconsistent to veterinary meters. This is due to differences in plasma binding to glucose between species. In cats, stress hyperglycemia interferes with the ability to perform reliable blood glucose curves in the clinic. Depending on the cat and owner, stress hyperglycemia may still complicate the results obtained at home.

Continuous glucose meters (CGMs) such as the Freestyle Libre ([www. freestyle.abbot](http://www.freestyle.abbot)) measure interstitial glucose automatically over long durations. Like all new diagnostic tools, we as a community are still working out many of the nuances. At the time of this update the Libre 3 system is newly emerging. For interstitial glucose monitoring the patient must be well hydrated otherwise the sensor fails to function. Further, there is a time lag for interstitial glucose to equilibrate with blood glucose. Successful attachment of the sensor can be difficult, especially in cachectic cats and 14 full days of monitoring may not be achievable. Current out of pocket cost for clients for the Libre2 system was about \$60-80 per sensor and about \$180 per reading device. Though iPhones can be used as a reading device, easy sharing of curve information from client to doctor via iPhone isn't available.

The Libre appears most accurate in the lower glucose range (<150mg/dL) and far less so in the higher ranges (>350 mg/dL). Personally, I have found it useful for routine monitoring of the newly diagnosed or stable diabetic, monitoring for *hydrated* hospitalized DKA patients to and beyond discharge, as a screening tool for unrecognized hypoglycemia, or to monitor for the possibility of prolonged insulin duration > 16 hours which suggests a need for once per day dosing. New competitor products, also for human use, are available but not yet tested in the veterinary field.

The serum fructosamine is an average of the glucose over roughly the proceeding two weeks. This information may be the only reliable data that can be collected in diabetic cats; however, it still has its limitations. In the clinically well diabetic pet, a fructosamine adds little useful additional information although a low fructosamine in a healthy cat suggests diabetic remission and insulin overdose. In a clinically unwell pet, a low fructosamine also indicates likely insulin excess, however, a high fructosamine can occur from insulin underdose -OR- insulin overdose. This is because of the "Somoygi effect," the over-swing phenomenon. In a patient experiencing significant hypoglycemia counter regulatory hormones are released causing insulin resistance, a numbing of insulin effects. This life-saving hormonal surge can persist for up to 72 hours. Clinical signs of diabetes worsen, BGs

and fructosamine typically measure high, and insulin dose is often *increased* when it needs reduced. This can occur even when the absolute nadir is not critically low however the drop in glucose from high to normal is so fast that the body senses the same hormonal panic. Clues to a likely Somoygi effect include a high insulin dose for the patient (anything approaching or exceeding 1 U /kg), the rate at which the dose was increased to the current dose noted from the history, the prior monitoring plan (which often is based on spot BGs or clinical signs), and the type of insulin. For instance, NPH and detemir are more likely to cause a Somoygi.

Urine monitoring is best used to screen for insulin overdose or diabetic remission. Urine monitoring should never be used to increase the insulin dose. Urine glucose is nearly inevitable in diabetic pets. On the other hand, a glucose-free urine sample indicates that at no point in time while that urine was forming and collecting in the bladder did the blood glucose exceed the renal threshold of >180mg/dl in dogs or 288mg/dL in cats. *When found consistently, the lack of glucose in the urine of a diabetic pet should alert the doctor to the possibility of insulin overdose.*

Urine ketones are seen not just in diabetic ketosis but in the urine of poorly controlled diabetic pets. While the finding of urine ketones does not always indicate an emergency, it does suggest inadequate control or concurrent illness (particularly in cats) and suggests further workup and BG curve are indicated. Remember that, due to the Somoygi effect, hyperglycemia, glucosuria, and ketonuria may result when the insulin dose is too high or too low.

In some cases of chronic overdose, the situation has become so complicated by counter-regulatory hormones that only *starting over with an initial small dose of insulin will resolve the confusion*. In these cases, given the legwork involved in starting over, I usually change to different insulin at the same time.

## **Conclusions**

There are many factors that effect diabetic management and not all of these are under our control. The owner may lack compliance or make changes at home without consultation. They may fail to replace the vial in a timely fashion or continue offering table scraps. Pet's may develop concurrent diseases that impact the insulin effect.

For our part, it is important that we don't make assumptions during treatment. While it is true that the nuances of how insulin usually behaves comes with experience, it is impossible to predict how any specific insulin will work in any specific pet. Only by collecting a large enough set of data for **that insulin in that pet** can we make the best treatment decisions. Starting over should not feel a failure but a second opportunity for the pet.

The secret to managing a diabetic pet is to monitor closely using blood glucose curves/continuous portable glucose monitoring, and to recognize the value AND limitations of the serum fructosamine and urine glucose/ketone strips. In outpatients, I start with a conservative dose of insulin and do not perform a BG curve with the first dose, however, one should perform a BG curve within 1-2 weeks of starting a dog on insulin. At a *minimum* a fructosamine should be performed in 2 weeks in the cat.

Insulin should not be increased by more than 10-20% which, nowadays, is functionally ½ to 2 units, 3+ in only the largest of dogs. Insulin should only be increased once every 7 days, perhaps twice weekly for pets wearing a CGM if free and easy data exchange is feasible in that case.

A BG curve should be rechecked for each dose change within 1-2 weeks (fructosamine in 2 weeks in cats). Avoid spot glucose checks for monitoring anything other than the pet's glucose at that moment in time, such as pre- or during anesthesia.

Promote a diet change and nutritionist input. Promote an early ophthalmology consult to prepare the owner for the options to help prevent and manage lens induced uveitis.

Ensure the owner is replacing the vial of insulin regularly and handling and administering the injections without issue. If, after multiple careful and well-monitored dose changes the pet is not doing well, start over with a new insulin or consider referral.

Ultimately, our goal in cats should be to try to achieve diabetic remission if possible. In dogs the goal is a happy pet without clinical signs and a satisfied owner without concern for hypoglycemia.



## **Feline Hyperthyroidism; A Few New Tricks for the Old Cat**

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First documented 1979, hyperthyroidism has grown from a disease once unrecognized to a condition screened for in routine wellness monitoring. The availability of the total T4 test on feline panels has allowed for earlier diagnosis such that the disease may now be confirmed before the first clinical sign.

In the normal cat, the pituitary gland produces thyroid stimulating hormone which acts on the thyroid gland to cause the production and release of thyroid hormones (T3 and T4). A classic negative endocrine feedback loop, once the circulating concentrations of T4 (thyroxine) reach suitable limits, TSH from the pituitary is suppressed. In the hyperthyroid cat, however, the thyroid gland is hyperplastic and autonomously secreting large amounts of excess thyroid hormone. Following the same pattern of negative feedback, circulating levels of thyroxine drop to nearly nothing.

90% of cats can be reliably diagnosed with a single elevated total thyroxine (TT4) measurement. However, for the remaining 10%, the diagnosis may be less clear. Individual patient variability, breed-specific nuances, and the high prevalence of concurrent disease in a geriatric patient population can complicate matters. False negative test results may delay the diagnosis and lead to significant morbidity for the pet whereas false positive results could contribute to misdiagnosis and iatrogenic hypothyroidism.

### **Signalment, Clinical Signs, Physical Examination, and Routine lab work**

Although hyperthyroidism has been diagnosed in cats as young as 4 years old, 95% of cats are > 8 years old at the time of diagnosis and 90% of cats are >10 years old.<sup>1</sup> Thyroid hormone has many effects on the body, particularly on metabolism, cardiac, and respiratory function. Thyrotoxicosis, whether caused by multinodular adenomatous goiter or the rare carcinoma, results in multisystemic illness and a wide variety of possible signs.

Since hyperthyroidism and its clinical signs are often insidiously progressive it can be months to years before the disease is recognized by the owner. Signs such as an increased appetite

and activity level can be misinterpreted as signs of well-being. Clinical signs may be vague, mild, absent, or in cases with significant concurrent illness may resemble other disease processes.

Cats with “apathetic” hyperthyroidism make up the minority (<10%) of cases. These cats typically have concurrent conditions such as GI tract disease, heart, or kidney disease and the combination of illnesses causes clinical signs of poor doing. Typical clinical signs of lethargy, anorexia, dehydration, and weakness can be difficult to differentiate from other feline illnesses.

### **Clinical signs of hyperthyroidism listed in relative order of frequency**

Weight loss

Polyphagia

PU/PD

Restlessness/behavior change

Gastrointestinal: Vomiting, diarrhea, anorexia

Haircoat and skin changes- alopecia, dry or greasy seborrhea, thinning of skin

Cardiorespiratory – tachypnea, dyspnea, coughing

Neuromuscular – seizures, tremors, cervical ventroflexion

### **Physical Examination:**

In the earlier stages of disease, weight loss results mainly from catabolic muscle wasting and not from decreased fat stores meaning that body condition scoring and muscle condition evaluation are important in addition to body weight.

Whereas it was once rare to diagnose hyperthyroidism in a cat without a palpable thyroid nodule this seem to be changing with roughly 1 in 5 cases now being diagnosed without a palpable thyroid slip. A reminder that 1 in 5 cats with a palpable thyroid slip do not have

hyperthyroidism and studies have shown the the severity of T4 elevations do not correlate to nodule size. Thus, more so now than before we can say that not all cats with hyperthyroidism have thyroid nodules and plenty of cats have nodules without hyperthyroidism.

### Routine Laboratory Findings

In cats, there is little on routine lab work that specifically indicates hyperthyroidism, however, *85% of hyperthyroid cats have a mild to moderate ALT elevation*. In my experience the ALT elevation is in the 130-200 mg/dl range, however in extreme toxicosis the ALT may increase into the moderate to severe range. ALP elevations are also possible. Of course, elevated liver enzymes are not specific to hyperthyroidism and could represent comorbidities, however, TT4 measurement is recommended in any feline patient with an elevated ALT, most especially if the remaining lab work is normal and the patient is >8 years of age.

### **Thyroid Hormone Testing**

In most cases an elevated TT4 combined with compatible clinical signs and a palpable cervical nodule confirms the diagnosis. The longer the duration of disease, the higher the TT4. With early diagnosis of hyperthyroidism, elevation of T4 levels is less extreme and T4 may even be normal.

There are several reasons why a cat with hyperthyroidism may have a normal T4. First, there is normal daily variability of T4 levels in both normal and hyperthyroid cats. In addition, non-thyroidal illness can affect thyroid testing results both directly and indirectly. Hypoproteinemia may result in a direct decrease in measured T4 levels due to decreased protein-bound thyroid hormone availability to the assay. Further, nearly any chronic illness can cause thyroid hormone suppression decreasing a mildly elevated thyroid into the high normal or even normal range.

### What if the T4 is not clearly diagnostic?

“Borderline” results are typically classified as a T4 in the upper 1/3<sup>rd</sup> of the reference interval. Reference intervals are assay dependent and test results should be interpreted within the known reference interval of the assay. None-the-less, researchers have shown that a TT4 > 3.5 ug/dL is a statistical outlier that should be evaluated further. If clinical suspicion is high, the

patient's thyroid hormone status should be investigated further, regardless of the initial T4 result.

A simple exercise to evaluate the clinical suspicion is included here with questions to be answered by the veterinarian based on the available history and examination findings.

1. Is this cat at least 8 years of age, Yes or No?
  - There is only a 5% chance of hyperthyroidism in a cat <8 years old.
  
2. Is there a palpable thyroid nodule, Yes or No?
  - ~80% of cats with hyperthyroidism have a palpable thyroid nodule, even though ~20% of cats with a cervical mass are not hyperthyroid.
  
3. Is the cat in poor body condition, Yes or No?
  - Overt hyperthyroidism would be atypical in an overweight or obese patient even though occult hyperthyroidism cannot be ruled out.
  
4. If thin/poor body condition- is the appetite maintained, Yes or No?
  - There are few differentials for weight loss and decreased body condition in a cat with a normal or increased appetite. In addition to hyperthyroidism, causes include diabetes mellitus, exocrine pancreatic insufficiency, and malabsorptive/maldigestive GI tract disease.
  
5. Is the ALT elevated, Yes or No?
  - Most hyperthyroid cats have increased ALT levels (+/- ALP)

With the number of "yes" answers to the question, the clinical suspicion increases. On the contrary, as the number of "no" answers increases, the clinical suspicion becomes lower.

*When clinical suspicion is high*, the practitioner elects to add additional tests of feline thyroid hormones or to repeat T4 measurement. In most cases hyperthyroidism is not an emergency

and repeat testing can be performed within 1-4 weeks. Urgency may increase in cases of apathetic hyperthyroidism with significant concurrent non-thyroidal illness.

*If clinical suspicion is low consider rechecking T4 immediately or in several months. If normal, clinically significant hyperthyroidism is ruled out. If borderline, then further thyroid testing is recommended at that time.*

### Free T4 (fT4)

A highly sensitive test (98.5%) the fT4 can be used to rule OUT hyperthyroidism with confidence. However, research has shown that the fT4 can both decrease or increase in cats with non-thyroidal illness. Thus, *fT4 measurement should not be used as the sole test to confirm hyperthyroidism.*<sup>7</sup>

This is particularly important to remember when using both T4 and fT4 for routine screening. In other words, while an elevated T4 and fT4 is consistent with the diagnosis of hyperthyroidism, a normal or borderline T4 and a high fT4 is not diagnostic for hyperthyroidism and requires additional testing. Free-T4 measured by equilibrium dialysis (fT4ED) is preferred over other types of free T4 assays however equilibrium dialysis has become less available than previously.

### Thyroid-Stimulating Hormone (TSH)

In human medicine, serum TSH levels are used for routine thyroid monitoring. In the case of hyperthyroidism, which is caused by autonomous secretion of thyroid hormone, TSH should be very low. In fact, TSH values compatible with hyperthyroidism in cats have been shown to be below the lower limit for quantification in current commercially available canine TSH assay (cTSH). Using available cTSH assays researchers found that an undetectable cTSH level in cats without clinical signs or thyroid hormone elevations went on to develop clinical hyperthyroidism within about 1 year.

In a newer study it was also shown that cTSH levels were too low to measure (<0.03 ng/mL) in 98% of hyperthyroid cats compared to low but measurable values in normal cats and in cats with euthyroid sick syndrome. *The cTSH is not able to reliably differentiate between normal and euthyroid sick cats and the cTSH test cannot be used as a sole diagnostic test for hyperthyroidism in cats. However, by combining the results of cTSH with TT4 and fT4ED cats could be accurately diagnosed with hyperthyroidism 98.8% of the time.*<sup>9</sup>

### Feline specific TSH

Recently, a feline-verified TSH test (fTSH) has been developed and integrated into a point-of-care diagnostic system with a lower limit for detection of fTSH of 0.008 ng/mL compared to the lower limit of the currently available cTSH of 0.03ng/mL. The increased diagnostic sensitivity in the low low range should allow for better characterization of the feline thyroid patient and may allow for differentiation of hyperthyroid, normal thyroid, and euthyroid sick cats. Clinical studies to investigate this further are currently underway. The fTSH test may replace fT4/fT4ED for confirmation of hyperthyroidism in cats.

The clinical implications of varying thyroid panel results are listed in **Table 1**.

**Table 1. Summarized Thyroid Hormone Panel Test Results and Clinical Implications**

	<b>TT4</b>	<b>fT4 / fT4ED</b>	<b>cTSH</b>	<b>fTSH</b>
<b>Normal Cat</b>	Normal	Normal	Low but measurable	Reference ranges being established
<b>Euthyroid Sick</b>	Low or Normal	Low, Normal, or High	Low but measurable	Studies ongoing
<b>Hyperthyroid</b>	Normal or High	High	Too low to measure (<0.03 ng/mL)	Studies ongoing

### **Iatrogenic Hypothyroidism (IH)**

Hypothyroidism occurs following treatment of the hyperthyroid cat in upwards of 50% of cats regardless of the treatment modality chosen (radioactive iodine, bilateral thyroidectomy, and methimazole). Studies have shown that when using T4 alone to monitor for IH upwards of 80% of cases could be missed. *If azotemia is present a low or low normal T4 and a high TSH suggests IH and supports the need to decrease the methimazole dose or to start oral thyroid supplementation in cats previously treated with radioactive iodine.*

This is important because studies have shown that azotemia is more common in hypothyroid cats and azotemic hypothyroid cats had a significant decrease in median survival time (456 days) compared to non-azotemic cats (905 days). Azotemia secondary to IH resolves with normalization of T4 about ½ of the time.

## **Conclusion**

In summary, feline hyperthyroidism is a common disease of geriatric cats. While most cats can be diagnosed based on history, physical exam findings and results of a single total T4 test, a small but important number of cats require more extensive work up including measurement of the complete thyroid hormone panel. A new feline specific TSH assay should help improve the diagnosis and management of complex cases and may allow for earlier diagnosis of disease and more precise management of therapy. Recognition and treatment of iatrogenic hypothyroidism as a side effect of treatment is important and may help delay the onset of clinical CKD.

## **Emotionally Intelligent Euthanasia**

Kathleen Cooney, DVM, CHPV, CCFP

Companion Animal Euthanasia Training Academy

Loveland, Colorado

Connexity 2022 event

A few years back now, a veterinary student shadowed me for a week to learn how I cope with the emotional strain of euthanasia-centric work. Her backstory was filled with highly sensitive post-euthanasia episodes, wherein she needed to cry; intense crying for upwards of 15 minutes. It was deeply affecting the rest of her day and building a sense of dread in her psyche towards the next one. After witnessing her first euthanasia with me, watching my protocols and approach, she cried again, but this time in relief. She had never seen a slow and smooth euthanasia like that before and expressed how a huge weight has been lifted off her shoulders. This student, just starting out in her journey in a long career, was granted an important view of not only a quality medical procedure, but on how attention to an Emotionally Intelligent Euthanasia (EIE) can forever shift our perception.

Emotional Intelligence (EI) is the capacity to be aware of, control, and express one's emotions, and to handle interpersonal relationships judiciously and empathetically. When we apply this to euthanasia procedures, filled with delicate terrain to begin with, it's vital the entire veterinary team, shelter staff, etc. is fully attentive to their personal behavior. This is understandably hard to do when you are focused on ensuring the medical procedure is properly carried out. Shifting attention to our own physical and mental state during euthanasia becomes paramount. Are we in control of our own emotions? Are we unintentionally internalizing our client's grief? How are we protecting ourselves from the start? EI has been around a long time. Many books have been written; studies conducted. I like to think of it as the blueprint for compassionate work. If

you can master even a bit of it, oh the places you'll go. It's powerful and life changing. People take years, entire lifetimes to master EI, but with even a little attention given to it, your ability to support pet patients and families during euthanasia is limitless.

There are 5 main components to EI. They are Self-awareness, Motivation, Empathy, Social Skills, and Self-regulation.

- Self-awareness is being mindful of your feelings, emotions.
  - Example: "Performing this euthanasia will be hard on me. Today is the anniversary of my own cat's death."
- Motivation / passion is appreciating what drives us. Identifying our motivation helps us understand why we feel the way we do about a given situation and ultimately how we respond to it. I often think of suffering with this one. If your motivation is to help people, and you cannot, it can feel like suffering, like despair. Suffering is when you are denied your true self.
  - Example: "I want to help this patient at home because that's where I want my own pet to be."
- Empathy is the ability to feel what others are feeling. It is the understanding of their emotions. It is the ability to put yourself in the other person's shoes.
  - Example: "My client is overwhelmed with this decision. I know what that's like."
- Social skills relate to how we interact with others and even influence them. You've been developing your social skills since childhood. Even if they don't come naturally to you, you still have them and they can always mature.
  - Example: "My client is angry. I can use my gentle body language and tone of voice to diffuse him."
- Self-regulation is having control over our physical and emotional state. I like this one. It helps me to act intentionally, with my mind in control, not my adrenaline or emotions themselves.
  - Example: "Things are getting challenging and my body is tense. Time to take some deep breaths."

If any of these 5 components feel unnatural, they are the ones you should devote the most time to. They are not low hanging fruit, so the reward when you master them will be that much sweeter.

We have deep, complex emotional lives and our pet patients do too. You will be watching their emotional states just like your clients. Intelligence comes from first identifying the emotion before you, and within you, then acting accordingly. If it's positive, you may be inclined to simply acknowledge it and reflect it back to them. Those around you are conveying trust for example, you can trust in return. If the emotion is negative, you may get pulled in that direction. The sooner you realize it, the sooner you can respond with intention and implement social skills to counter. Building awareness of emotions is as basic as telling yourself what you see or feel. "The person in front of me is feeling fear", "I am feeling optimism", "That cat is apprehensive". It might sound a bit absurd, but that's how it all starts. Recognize and adjust accordingly.

### **Building Self-Awareness**

Self-awareness is the idea of focusing on the here and now, and understanding the internal and external environment we are in. For me, it forces reflection on how I want to act right now and what emotions I want to convey to my client, my team. One of my favorite exercises before an appointment is to reflect on it for a few minutes. I write up my own euthanasia consent form to familiarize myself with the names, the pet's signalment, the reason for euthanasia. I look over the notes to see who will be in attendance, where they are traveling from to reach my Comfort Center, and what they've chosen for aftercare. If I've known the family for a while, I think back to what they've gone through caring for the pet, what my experiences have been with them, and end with a general sense of empathy for what they are about to undertake. And right before I step into the appointment, I look upon an image of the human animal bond and take a deep breath to ground myself in the moment.

Building self-awareness includes looking for triggers throughout the euthanasia. Maybe it's a similar pet to your own, the patient's diagnosis might be an old nemesis, or the woman is the spitting image of your

favorite grade schoolteacher. It all applies and can affect your emotional state. Another trigger for me, and could be for you, is when clients are ultra-quiet. I enjoy hearing a few stories and getting to know about the pet's life. Extreme quiet can feel disconcerting and bring on a sense of discontent. For others, it's exactly what they like.

And avoid auto pilot behavior. In the early days of your euthanasia work, this will never happen. There is so much to think about between client communication, the technical work at hand, etc. But after a while, comfort with euthanasia sets in and auto pilot happens. It means you are at peace in the setting, you feel confidence in your work and skilled enough to let your mind wander. It's great, but it means you are more likely to mentally and emotionally check out from what's in front of you. I'm guilty of this regularly but knowing what I should be focused on helps draw my attention back to the here and now, which has increased my capacity to see the emotions in the room.

Once you have labeled your emotions, acknowledge it, and feel gratitude for having done so. This is the trick to really tapping into your EI. And keep things in perspective by reminding yourself of truths. "I am not euthanizing my own pet right now. I am here to facilitate this request." "It's her cat, not mine." "This is her grief journey. I am here to bear witness and support."

### **Allowing Other's Grief**

**Pet owners have the right to feel the way they do. Their emotions come from deep within. Encourage them to keep it. This means you say to yourself, "I see your sadness and grief. You must go through this. This is your story and moment." To them, they see your patience and kindness and understanding. If we take away the grief they feel, or try to minimize the stages of grief themselves, we slow the mourning process. It will only complicate grief. Taking grief away could come in the form of telling people not to feel guilty, not to be sad or to be happy their pet is no longer in pain. People will feel what comes naturally and that natural state tends to be healthier than something synthetic or forced. There is a book out there called 'It's**

**Ok that You're Not OK' by Megan Devine. I appreciate the notion of meeting people where they are at in that moment. And if you try to take their pain and sadness away, you risk internalizing it yourself. I've seen veterinary professionals do this countless times. Showing compassion and empathy does not include welcoming in another's pain.... it's about walking with them and witnessing and lending a helping hand where you can.**

### **Who Are You?**

**Emotional Intelligence grows by knowing your core values and strengths. Find out what makes you 'you', at the very center of your soul. What things do you like or don't like? What makes you tick and gets you going day to day? Those who know their values and strengths tend to place themselves in settings where they can shine. They will do better in work environments that utilize the strengths. Core strengths in euthanasia tend to be ones like relating well with others, harmony, and empathy. If you find empathy is not a core strength of yours, what a great opportunity to find an assistant that radiates it. You will complement each other with your skills. One of my core values or principles is stewardship. I like to take care of people. Knowing this increases my awareness of negative emotions when I cannot. When I cannot help, I'm bummed and when I can, I'm happy.**

In preparing to write an EIE training module for the Companion Animal Euthanasia Training Academy (CAETA), the following outline was developed to support professionals before, during, and after euthanasia. It is a start in the long discussion around self-awareness and preservation.

### **Awareness of your own previous experiences**

**Does this situation remind me of a negative incident from my past?**

**In what ways are there similarities and differences?**

**How am I going to decipher between this current experience and my own previous ones?**

### **Ownership of what is mine to control**

**Have I approached this appointment following the right procedural steps? Am I aware of other's control over their own destiny?**

**How will I shift the focus of ownership to them, not me?**

### **Development of self-awareness and regulation**

**What is my body doing right now?**

**Have I identified stressful triggers around me?**

**Am I following the necessary steps to maintain myself in a relaxed state?**

### **Protection with the right people**

**Who is on my support team?**

**How often will I be connecting with them?**

**Am I prepared to share my true feelings and concerns for my personal growth?**

### **Recognition of individuality**

**How is my euthanasia approach similar and different from others? Am I comfortable with my own style?**

**What do I like best about my euthanasia protocols and behaviors?**

In closing, I'd like to share a direct sentiment from a veterinary student who was reflecting on EIE recently. "Establishing emotional intelligence & acceptance means recognizing how we feel, admitting it ourselves, and giving ourselves permission to feel negative emotions (that which you resist, persists!). Perhaps more importantly, you don't have to block out negative emotions or be emotionally 'porous' and let everything through; there is a middle ground where a healthy type of connection exists." This is true to many of life's complexities. I appreciate her words and the attention we all must pay to our emotional health around euthanasia work. It is in our own emotional protection that we are granted the ability to care for those who need us.

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## **The 7 Habits of Highly Effective Communicators; Good death dialogue for end-of-life professionals**

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Knowing how best to communicate with grieving clients is challenging for many. Some of us are naturally gifted at saying the right things, putting clients at ease and giving emotional support. Some of us have our hearts in the right place when supporting dying patients but struggle to convey what we want to say. The idea of good death dialogue is to wrap a client in safety and warmth, which anyone can do it with a little know-how. Let's take some time to unpack the 7 habits of highly effective communicators and gain confidence working with clients in a time of grief.

As we begin, there are a few key concepts to keep top of mind. There are differences between sympathy, empathy, and compassion. Sympathy is about recognizing the emotion of another person. Think...that person is sad. Empathy is recognizing and feeling similar emotions to our clients. Think...that person is sad, and I know how that feels OR that person is sad, and I am sad too. Compassion is empathy in action. In other words, think...that person is sad, and I know how that feels and now I want to relieve that sadness. Compassion comes in many forms, for example, trying to help relieve patient suffering, offering to walk someone to the door after a sad euthanasia, and calling them the next day to check on them. These are ways we relieve negative feelings and emotions, although we know that having these feelings are a natural part of the grieving process. It's important to allow clients to feel the way they want to feel.

Clients want to be understood but have little capacity to understand. When faced with the loss of a dear friend, they may feel confused, angry, isolated, helpless, and sad. Meeting them where they are at, mentally and emotionally, is vital to the interaction. This can be hard to do, especially when engrossed in a busy workday. The trick to good communication is to quiet oneself and really listen; listen to what is being said and not being said (aka reading between the lines) and then helping clients navigate through emotional experiences such as learning their pet is sick, a euthanasia procedure, or natural dying process, and the grief that will follow these. A great saying to remember is 'Mouth closed, ears open, presence available'. It will help you to listen first, then you can carefully craft your responses with intentionality. Let's look now at ways to improve our death dialogue.

## The 7 Habits of Highly Effective Communicators

### 1. Establish rapport

Give a warm welcome... "Glad you are here", "What an honor to care for you"

Make soft eye contact with an easy smile

Learn everyone's names

Greet the pet patient for a few seconds, with client watching

Set the right tone - offer empathy, remain calm, non-clinical

### 2. Be comfortable with silence

Offer an open-ended question to determine how much they want to talk

Remain 'present' even in silence...mouth closed, ears open, presence available

Take a deep sigh if you feel yourself needing to speak when unnecessary

### 3. Offer praise

Give praise and gratitude regarding anything you see worthy of it..." You have taken such good care of her", "She has had so many wonderful adventures with your family"

Offer "Thank you" a few times

#### 4. Use the basics

Rely on standard communication skills to stabilize the conversations

Examples: reflective listening, summary statements

#### 5. Piece it out,

'Piece is more peaceful', 'Find peace in pieces', 'Make it peaceful'

Give less information all at once

Avoid big speeches - edit down and use simple jargon

Small, tailored chunks are more manageable

#### 6. Attend to emotional intelligence

Self-awareness

Am I truly present?

What am I conveying?

What am I feeling?

Social skills

What is the client telling me?

What does my body language indicate?

Can I diffuse the tension?

Empathy

What is the client feeling?

What would I be feeling in a similar situation?

Self-regulation

Is my body relaxed or tense?

Do I perceive any threats here?

Motivations

Why does my client act that way?

Why am I feeling this?

How would I like to control the situation?

#### 7. Create partnership

Good or bad, we convey we are in this together

Give reassurance about decisions made

Include 'us and we' language..." We are going to take good care of you", "We are in this together", "Together we will determine the next steps", "Reach out to us"

Establishing rapport with clients during death and dying is one of the most important things you will do in your careers. For our clients heavily bonded with their pet(s), feeling safe and supported is paramount right from the start. This connection or rapport made with both the humans and pets sets the tone for the whole experience. Many times, the success of any end-of-life journey is not only measured in how peacefully the pet died, but how cherished and loved everyone felt in the room.

It's nice that it doesn't have to be complicated. A gentle smile, soft eye contact, getting to know the pet, and setting a compassionate tone is plenty for many of us. What's important is to start off on the right foot. When we begin with the idea of building rapport in mind, there is no reason to back track if things have gone awry.

Regardless of your comfort level, there are some simple communication skills we can strive for. From a Psychology Today article from 2015, titled [How to Build Rapport: A powerful technique](#), they list the following:

- Match and mirror behavior – this includes sitting when others are sitting, lean forward then they do, or matching up with another’s breathing rhythm. One of my favorite mirror behaviors is to take a deep sigh at least 1 or 2 times during the euthanasia appointment. When I sigh, my clients almost always sigh right after, and if not, there is still a subtle relaxing energy shift in the room. And remember, it’s always ok to cry with clients.
- Find a common energy level - you are looking for harmony between your bodies and mental states. If the client is sad, the veterinary team is somber. If the client is joyfully reflective on the pet’s life, the team can be cheerful with them. What we don’t do is mirror negative energy, such as anger or hostility. Instead, we diffuse it by remaining calm and composed, with the hope our clients will mirror us.
- Maintain a supportive tone of voice – safety and trust in the veterinary team may go out the window if our tone gets shrill, angry, or high-pitched. Even if the euthanasia procedure is getting complicated, and our adrenaline is kicking in, the tone of voice we project must remain placid. A supportive tone is soft and balanced will convey disciplined authority, leaving our clients feeling safer.

In Dr. Jane Shaw’s End-of-Life Communication Module, part of the International Association of Animal Hospice and Palliative Care’s (IAAHPC) Certification Program, she highlights the importance of asking open-ended questions when establishing rapport; to help people open and share their thoughts / feelings. “What do you know about the euthanasia procedure?”, “What have you and your family discussed in preparation for today?”, and “How would you like me to help with her aftercare?”. These open-ended questions empower clients to feel more in control. They feel heard and are more likely to believe in the veterinary team’s abilities. Dr. Shaw also talks about offering partnership to clients. Statements like “We are all here together to support Ollie in this transition” and “Let’s gather around to create a circle of love.” The notion of losing a beloved pet can feel very

isolating. Building a sincere relationship with clients, and everyone in the room, is like a warm hug to sustain them during such a sad time.

Sometimes we don't find the rapport we are looking for. Clients have the right to be gripped by their grief and ignore normal 'non-grief' social behavior. Building rapport may be the last thing on their minds. We nevertheless remain kind and open to their needs; offering gentle smiles and space to grieve. When rapport is found, the hormone oxytocin is released in both you and the client.

Oxytocin is a complex molecule that bonds people and animals together in love. It starts flowing the second a safe interaction begins but can lead to negative reactions too. In the wrong environment, oxytocin has a Jekyll and Hyde effect. While it can be very useful to insight trust, it may trigger aggression and strong defensive behavior when trust is not present and when fear is. If clients are feeling stressed and unsafe during a conversation or during euthanasia, such as when they see their pet in pain or distress, their circulating oxytocin can elicit negative emotions like distrust, hostility, and anxiousness (through the activation of vasopressin). This is one of the main reasons establishing rapport with clients is so important before and during euthanasia. Increasing preplanning and speaking with clients about what's important to them should deescalate stress. Oxytocin boosters include smiling, touch, familiar pleasing smells, pheromones, sunlight and biophilic elements like nature.

### Verbal Hug Phrases to Lend Support

Since 2006, I've built up a large library of useful phrases and sentiments to share with clients during euthanasia appointments. Knowing the exact time to offer them can be challenging, especially since you won't know the full context of the situation until you are engaged in it but take a moment and picture yourself with your pet patient and devoted family. Some of the

phrases are specific to grief support while others speak to more logistical factors. Set the right tone for yourself (my words are heavy with reverence and respect). I hope you find this useful in your euthanasia communication. Note that in place of pronouns, I encourage you to use the pet's name as much as possible to deepen the connections.

### In Support of Crying

- ~ We all need someone to cry for us. It's the sign of a life well lived.
- ~ You honor her with your tears.

### In Witnessing Active Signs of Death

- ~ She is embracing the change.
- ~ She is releasing energy.
- ~ He is passing now and knows you are with him.
- ~ His movements are very natural and normal. These can be expected even with natural death.
- ~ Muscle fasciculations = energy release
- ~ Agonal breaths = reflexive, sign of the transition, energy release

### Describing the Process to Children

- ~ Pre-euthanasia sedation: I am going to give her a medicine to help her to sleep. It is a medicine only for our animals. Never for humans. It goes under her skin and will help her to rest.
- ~ Euthanasia itself: The second medicine, also just for our animals, never for us, will be given under the skin too. This is the special medicine that will help her to die; this is why we are here. She is dying on her own and my job is to help her along with that.

### In Preparing to Give an Injection

~ I'm going to lay my hands on him now to get him used to my touch.

~ Her comfort is important to me. Please let me know if she is tender anywhere and I will avoid those areas.

### Remaining Present for Euthanasia

~ How do you feel about remaining present for his passing?

~ All are welcome to be present, and this is safe space for you too. If you feel you need to step out of the room, you may do so at any time. He is in good hands and will always feel your love.

~ Let me describe how I'll be supporting her so you know more of what to expect.

### In Handling Payment

~ You have the choice to handle financials with me now over the phone or during the appointment. Which would you prefer?

~ If it's ok with you, let's take care of our financials now so we can focus our full attention with him.

### With Potential Dysthanasia

~ Thank you for allowing me to take my time in helping him.

~ Her passing was different than expected. Let's explore this so we understand what happened.

### Additional Phrases of Support

~ Thank you for loving her so deeply.

~ How blessed he was to have such a loving family.

~ You take on great personal pain today to let her go.

Everything we express should feel like a verbal hug. We hope to convey to our clients a sense of safety and stability during a very delicate time. You are sure to have favorite support phrases that carry you through. Share them with others and together, we will build a language of love.

## **References and Resources**

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## Why Can't They Just – Notes – Senani Ratnayake

What if reality was merely an illusion? What if *your* reality is created by *your own* brain based entirely on *your* personal perceptions... making each person's reality unique to *them*?!

Well, if that's true, then it's not that difficult to imagine why sometimes the things that are obvious to us may not be for somebody else. Things that appear one way to me might appear a completely different way to you.

It is simple to sit back and observe a situation wondering why somebody else didn't handle it as you would have, or think about it the way that you did... and yet, based on what we know of how our brains work – it should actually be more surprising to us at times when somebody *does* see things exactly how we do!

The libraries in our brains begin to build before we are even born. We are exposed to sensations such as touch (heat, cold, pressure, pain), taste, smell and sound, developing preferences that will become forever logged in our brains. Over time, our brains will literally capture and catalogue thousands of experiences a day – from sensations to experiences, each learning building on the next, to become an individualized databank of what we know “to be true.”

Our perceptions of the things we encounter are based on everything we have encountered thus far – our brain must decide where to categorize each new thing and looks for commonality in order to sort and understand what we are experiencing. This can, at times, create problems as brand-new groupings do not yet have context and our brains may misinterpret what we are encountering.

Our perceptions are the reason for our responses to the things around us, and they can be influenced by many things. This can be both a positive, or a negative, depending on our circumstances.

But let's take a step back. It starts with our perception of ourselves. Our perception of our own lives, our own successes and our own struggles. It is easy to look at somebody else and believe that they have it better or worse than you based on your perception but we often don't truly know enough about another person's situation to be able to compare. That's perspective. That said, it is not just about what is on your plate, it's also about your capacity and your tolerance. Something that is easy for you to navigate may be complicated for another. Something that doesn't affect you may greatly affect somebody else. These differences from person to person make it tough to determine how somebody can truly cope with what is before them just by knowing what you know of their (literal) challenges.

As the old saying goes, we should be able to put ourselves in somebody else's shoes when we want to understand how they are feeling or perceiving their reality. Obviously, this is not possible and, almost insulting to imply that it is. How can one person possibly understand, in totality, what another is thinking or feeling when we cannot first appreciate the scope of their experiences and encounters to-date?

Nobody can fully understand what it is like to be you, but you. We know that drama within the team is often caused by failure to see a different perspective, and so we first start with our own perspective on our own circumstances. It's not because anybody's world is better or worse, it is merely for the sake of acknowledging that everybody is different.

You are likely not qualified to do anymore than simply help them see a bigger picture – affect their perspective – so don't attempt to do anymore than walk them through the process of capturing what their bigger picture actually is. For some it is easy to become the person who takes on everybody's problems but, this is neither healthy nor helpful, for anyone.

Next, we want to tackle professional goals. We know how we want people to behave in the workplace with respect to culture and values, and with respect to work ethic and job description. We often run into situations where our words are not interpreted equally by all, and everybody has a different version of what success actually looks like. This situation is often caused by people only looking at their role from their own perspective, they haven't (or are unable to without guidance) actually taken a look at the bigger picture to interpret how their role then affects other roles in the hospital, and how their diligence and care, or lack thereof, will affect the overall flow and operation of the hospital. This is where Starting with Why can be very helpful, and tying things back to patient and client care along with your Mission/Brand is a must. If we as a team are committed to something, then we need to assess what our individual roles are in making it so as a team.

It can be interesting to explore this in three steps, first individually based on your perspective of your role, second as a department (how does your group and it's effectiveness affect the other groups in the hospital; what is expected of us by them and what do we expect of them in order for us to be successful) and third as a full team in order to share and learn about whether our expectations of others, and their expectations of us are in fact aligned.

There is really no end from there as to how you can work with your team to put a new perspective on everyday circumstances. You can tackle how we perceive other people based on existing biases, how people perceive our value based on our existing environment, language and processes, how we deliver customer service. One of my favourite activities is putting a new lens on our perspective of ourselves in our roles. Let's each create a business case for ourselves – in the same way we would a new piece of equipment or renovation – can we articulate our unique value? This can be done in a fun way without creating a feeling of unworthiness by allowing partners who can build each other up. This should not be used on its own as a tool for performance critique, but could be used for developmental performance planning. While not all initial perceptions may be accurate based on the larger reality, this knowledge is invaluable for coaching and feedback.

Shifting perspectives and increasing awareness around personal perceptions can dramatically affect the level of drama on a team. Normalizing our differences, but also bringing commonality to the forefront will help to keep the "Why can't they just..." at bay, and also unite the team by securing and maintaining a greater, positive perspective.