Emerald Coast Veterinary Conference

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2021 Proceedings

ECVC is presented by the Alabama and Georgia Veterinary Medical Associations

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2021 Emerald Coast Veterinary Conference

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The Jedi Mind Trick

Andy Roark MS DVM
Tall Oaks Enterprises LLC
Greenville, South Carolina 29601

Calling on classic techniques in effective communication, persuasion, and negotiation, this session helps technicians motivate and provide feedback to their veterinarians.

Active listening, deep acting, and strategic planning all come into play when we set out to affect the behavior of organizational superiors. By considering these skills in-depth, our goal is to develop usable plans and techniques for initiating and carrying out conversations that matter.

This session is highly interactive, and content will be strongly affected by cases presented by audience members. We will review active listening skill, discuss and learn negotiation skills, and learn to provide effective and appropriate feedback.

Ten Steps to the Jedi Way:

10. Give a Reputation

Our reputations are built by others, not by ourselves. We can help people by giving them reputation. Anwar Sadat the President of Egypt gives reputation when he states: "I love coming here. You all are the most compassionate people." As a client, imagine the swelling of pride when you hear "You obviously have a very strong bond with this dog." What if you said? "You're the best teacher. You never lose patience when I ask questions because I want to know things, even if you're busy."

9. Reciprocity Norms

What is a reciprocity norm? Let's take the average veterinary conference. Candy bowls in the exhibit hall! Why do vendors have candy there? So, you will take it and feel obligated to chat with vendors.

Set the expectation of helping people so that they help you. "Do you need help with anything? Let me help restrain that cat for you. Now, would you be available to see a client for me?"

8. Make it about the Pet/Client/Practice

At my last practice. I wanted radiographs for a limping cat. The tech said "She'd be much more comfortable and a lot less scared if we sedated her. Her owners would probably think it was a great thing if I explained it to them that way. They want her to be comfortable more than anything." In hindsight, I know that she did not want to radiograph this painful cat, and I don't blame her at all. But she didn't say "I don't want

to do it." She made it easy for me to make the decision to sedate the patient because she made it about the patient, the client, and better medicine.

7. Create a Star
When I first started at my current practice, one of technicians talked me up to the clients. They were stoked when I got there. It made my life so much easier. Front desk staff picks up the positive comments and heightens them so that every experience builds stardom.

6. Forget managing and lead
When I started out in a single vet practice, I discovered the difference between management and leadership when the manage would ask "What do we need to do so that everybody can leave?"

5. Positive Reinforcement
We're animals. It works exactly the same way. Praise and thank you goes a long way. An example of behavior that people want to change in practices that regularly comes up is reluctance to get on the phone with clients. You can encourage it the way it was encouraged with me. " Thank you for getting on the phone with that client. I really appreciate it. You're the best. You're so good at handling those things, you're really the best when it comes to stepping in to help with client questions. Everybody says so." – They expressed gratitude which felt amazing, and it gave me a reputation to uphold.

4. Increase perceived value/Benefit/Sacrifice
Benefits can be abstract and hard to nail down. It's not impossible.
A client has called repeatedly, and you want your vet to call this woman back.
Benefits: "Hey, I know you want to get home tonight and I don't want you to get stuck here." Or "hey, I worry that she's getting frustrated and think it might be better if you got to her earlier."
Reduce sacrifice: "here's her phone number and I pulled the chart up for you in this computer in case you wanted it."

3. Focus on their problem
We all want solutions to our problem. If they have a bleeding neck issue and you have a solution, you're good to go. How does what you want solve the problem they are having?

2. Make it their idea
The technician in step 8 could have just talked about the pain and anxiety, and how the owners want the pet to be as comfortable as possible. When I then mentioned sedation, she could have jumped on it with praise and positive reinforcement. The hardest thing is not jumping in. You want to plant the seed and let it grow.

1. Ask

Don’t be shy. You’re a part of the team. Build a history of asking because you want to know. Getting the how we ask down part is trickier, but it just take practice.
Actively listen = paraphrase, but do not parrot. This makes conflict worse.
Acknowledge commonality
Develop your phrases: “It sounds like…”, “If I understand correctly, you’re saying …”, “I don’t think I follow you, can you explain…”, “Do I understand correctly?”

Make sure you are speaking for yourself, not for others, your opinion and feelings carry more weight than the “I’ve heard others have this issue”.

It can be tough sometimes, so use softening phrases like, “Perhaps…”, “Suppose…”, “What if…”, “May I suggest…”, “Would it help if…”

Most importantly, ask before the decision is made, don’t wait.

Pairing CBC data with bone marrow evaluation, a case based approach

Dr. Erica Behling-Kelly, Associate Professor
Cornell University, Department of Population Medicine and Diagnostic Sciences
eb58@cornell.edu

Indications for aspirating bone marrow: In small animal medicine, bone marrow is most frequently evaluated to identify or stage a neoplastic process, or to determine if intramedullary disease is contributing to changes identified in the CBC. These abnormalities include increases or decreases in cell counts, disorderly maturation, and the presence of circulating neoplastic cells. Less frequently, the bone marrow is evaluated in the search for an infectious agent.

Clinical history and timing of sample collection: Appropriate timing of sample collection and inclusion of pertinent historical data and clinical findings are essential in evaluating a hematological disease. For example, bone marrow aspiration is not indicated in cases of pre-regenerative, acute hemolytic anemias. If an anemia is nonregenerative, persistent, and cannot be explained by a concurrent disease, bone marrow evaluation is indicated. The recognition of ineffective erythropoiesis due to destruction of earlier stage of erythroid cells (precursor directed or non-regenerative immune-mediated anemias), hinges on evaluating the kinetics of the bone marrow response. Pairing enumeration of the various maturational stages in the erythroid lineage with the clinical findings, allows the pathologist to make this diagnosis. A similar construct is applied in the evaluation of myelopoiesis and thrombopoiesis. The pathologist should be informed of the duration of any reductions in hematopoietic cell lineages (cytopenias), drug history or potential exposure to toxins, and any co-morbidities. Some common drugs, such as fenbendazole, have been associated with pancytopenia secondary to intramedullary cell death.

Pairing the aspirate with a complete blood count (CBC) and core biopsy: A CBC, ideally in sample format but alternatively in numerical data, will help the clinical pathologist provide the most accurate diagnosis and useful recommendation. Pairing findings in the marrow with cell counts in the blood facilitates distinction between hypoplasia, normal cellularity, relative hyperplasia, and overt hyperplasia in each of the three lineages. The blood smear, as part of the CBC, should also be reviewed for evidence of circulating neoplastic cells. If an orderly response is evident in the peripheral blood, there may be little information to be gained by evaluating hyperplasia in the bone marrow. Bone marrow evaluation is indicated if the cause of the inflammation is suspected to be intramedullary (certain fungal infections, for example). Cellular morphology is carefully evaluated for evidence of any dysplasia, both in the bone marrow sample and the blood smear. Acquisition of a core biopsy allows for evaluation of the trabecular bone, fibrosis, and architecture of the tissue. Identification of a neoplastic infiltrate can be facilitate by immunohistochemical labeling of cells, and is often useful in identifying tumors of lymphoid and histiocytocytic origin.

A series of cases will be presented to demonstrate how numerical and morphological details provided by the CBC and blood smear evaluation inform the interpretation of bone marrow aspirates. A subset of cases will include discussion of advanced diagnostics.

Evaluation of body cavity fluids

Dr. Erica Behling-Kelly, Associate Professor
Cornell University, Department of Population Medicine and Diagnostic Sciences
eb58@cornell.edu

Parameters of body cavity fluid evaluation:
The evaluation of body cavity fluids includes evaluating the gross features of the fluid, microscopic enumeration of cells and specific cell types, as well as limited chemical testing of fluid constituents. The goal of this evaluation is ultimately to determine the underlying pathological process driving the effusive process. Gross features of the fluid include the color and clarity of the fluid. Clear fluids are more likely to be transudative in nature whereas markedly turbid fluids will likely be exudative or neoplastic in nature. A yellow-tinge to the fluid may indicate icterus, and the strawberry milkshake color of a chylous effusion is a very characteristic feature. The protein concentration is most frequently approximated by measuring the refractive index of the fluid. Nucleated cell counts are commonly determined using a hematology analyzer that employs flow cytometric methodology, or an impedence based cell counter. The cell counts should always be compared to microscopic review of a direct smear. This helps ensure that particles are not being counted as cells, and to that cellular clots are not trapping the cells and causing a falsely low count.

Classification of effusions:
The terminology applied to classify effusions can be a subject of debate. The term exudate is universally applied to fluids with a high nucleated cell count (typically above 3,000-5,000 cells/ul, depending on the laboratory) and protein concentration above 2.5 g/dL. These types of effusions form due to chemotaxis of inflammatory cells across the vessels and concurrent leakage of serum proteins. The classification of an effusion characterized by a nucleated cell count that does not strictly meet the criteria of an exudative process, but a protein concentration above 2.5 g/dL often invokes terms including “modified transudate” or “high protein transudate.” The underlying nature of these effusions often involves increased pressures involving post-sinusoidal vasculature. Transudates, effusions with low cell counts (less than 3,000 cells/ul) and a protein concentration < 2.5 g/dL, form due to decreased oncotic pressures. Pure transudates are relatively uncommon in small animals. Often, more than one process is occurring. Neoplastic and hemorrhagic effusions are termed separately.

Microscopic evaluation:
The WBC differential can help to classify the mechanism behind the effusion. For example, lymphocyte-rich effusions are often chylous in nature and associated with congestive heart failure in cats. The predominance of neutrophils should prompt a thorough inspection for infectious agents or evidence of a chemical irritant, such as bile.

Chemical analysis:
Quantification of substances that should not be present in the body cavity at a higher concentration that plasma are often helpful in identifying the underlying etiology. Examples include triglycerides, bilirubin, and creatinine.

Cytological review of common skin lesions and lymph node aspirates

Dr. Erica Behling-Kelly, Associate Professor
Cornell University, Department of Population Medicine and Diagnostic Sciences
eb58@cornell.edu

General goals of cytological evaluation of skin lesions:

Skin lesions (dermal and epidermal) should be distinguished, when possible, from those arising in the subcutaneous tissues. The goals of cytological evaluation of these samples are first to determine if a diagnostic sample was obtained, and second to determine if the evaluation of the sample by a clinical pathologist is indicated. The separation of inflammatory lesions from a neoplastic processes is the primary point of dichotomy, and can often been accomplished in clinic. When a recognizable infectious agent (bacteria consistent with a Staph or Strep) fit the clinical suspicion is found, addressing the infectious process if often prudent prior to re-sampling at a later date. If no cause for the inflammation is found, structures that are not readily identifiable are noted, or tissue cells are evident in the sample, submission of the sample is recommended. Neoplastic lesions can often be secondarily inflamed, which can confound the interpretation. This is very common in cases of squamous cell carcinomas, and extreme care must be taken not to over-interpret dysplastic changes secondary to the inflammation. These samples should always be submitted to a clinical pathologist, and may require further evaluation with histopathology. Many common adnexal tumors tend be benign. The goal of cytological evaluation should not necessarily be definitive identification of specific tumor type, but rather exclusion of a process that might warrant further evaluation prior to surgical removal (eg a mast cell tumor indicating the need for wider margins than a sebaceous adenoma). Tumors arising from cells within or near the blood vessels in the skin tend to exfoliate, and can often be diagnosed cytologically. Grading of these lesions often requires histopathological evaluation of tissue architecture and invasion.

Lymph node aspirates:

Aspiration of lymph nodes is a primary modality for the diagnosis of lymphoma, evaluation for metastatic processes, and less frequently the evaluation of inflammatory conditions. If more than one lymph node is enlarged, aspiration of multiple nodes and avoidance of the mandibular lymph node, if possible, is recommended. Neoplastic lymphocytes are often readily exfoliative, but unfortunately also rupture easily. Thus, gently spearing of the aspirate is critical. The quality of the sample collected can be checked by quick-staining the sample, taking care to avoid the common pitfall of overstaining nuclear material by prolonged contact with the basophilic/azure dye in use. When in doubt, go light! It is far easier for a clinical pathologist to put an under-stained sample through their preferred staining procedure than it is to de-colorize and overstained sample. Metastatic populations can be identified in lymph node aspirates, and occasionally this can occur without identification of the primary lesion! There are some challenges in determining if low numbers of cells from a tumor are found in an aspirate as a result of their presence in the sinuses of the node, or if they have set up a neoplastic niche and are propagating the node. The evaluation of lymph node aspirates for metastasis of a mast cell tumor can be particularly challenge, as these cells are not only found in the nodes of healthy animals but also for reasons unrelated to cancer. Hypersensitivity conditions and inflammation in general can call mast cells to a lymph node, as they are part of the fibroblastic response in would healing. Infectious causes of lymphadenitis can often be identified using this modality.

EVALUATION AND COLORS (opacities) OF THE CORNEA

Kathryn A. Diehl MS, DVM, DACVO
University of Georgia College of Veterinary Medicine
Athens, Georgia

Corneal functional anatomy

No discussion of the cornea is complete without at least a brief and basic anatomy review. This is a shamelessly simple but not dumbed down lesson; it's truly a way to think about the cornea clinically during evaluation and disease interpretation. Understanding the function of the cornea allows understanding of the anatomy to follow naturally. The function of the cornea is two-fold. It is a defensive barrier between the outside world and the inside of the eye (which allows vision and thus is fairly important for most creatures to do their jobs). Additionally, as a glorified window, the cornea transmits and bends light. In order to perform these functions, the cornea is intact and pretty strong AND, it's clear (so the animal and you can see through it).

The anatomical components and features that give rise to these corneal defense and clarity functions include the corneal layers (from outside in): the tear film, the epithelium, the stroma (making up the majority of the corneal thickness), Descemet's membrane (the basement membrane of the endothelium) and the endothelium (a non-regenerative single layer of cuboidal epithelial cells lining the inside of the cornea / front side of the anterior chamber). And, they include the cornea's make up: with it being avascular, lacking pigment, being relatively dehydrated and with intensely organized collagen fibrils.

Corneal relative dehydration is governed by the epithelium, which is a "water-hating" mechanical barrier to fluid uptake by the "water loving" stroma – this is why with corneal ulcers, where the epithelium is missing, there is associated edema; and it is governed by the endothelium, which is similarly a mechanical barrier but also and most importantly, contains a Na-K ATPase pump that constantly pumps water out of the cornea.

The cornea's PERFECTLY regularly arranged collagen fibrils also contribute to clarity and a nice analogy to make sense of that is to recall that transparent glass and glass block are both made of the same material but it is the organization (specifically the variation in band gaps in different glasses, which are considered amorphous solids) that dictates whether a photon of light passes through or is absorbed or reflected. The cornea and the sclera are both made up of mostly similar collagen and yet the cornea is clear and the sclera opaque white, and this is due to the regular collagen arrangement in the cornea.

Corneal evaluation

Examination

With an understanding of what the cornea is supposed to do, what about it gives rise to that and thus what it is supposed to look like, you can start to examine it. First off, just look at it!! Do not be intimidated by the different anatomy and physical exam skill set requiring different equipment, or the different differential list and treatments including drugs and surgeries (requiring different and expensive instruments and with fragile, un-forgiving tissue), or the often highly owner-visible outcome and level of importance/value on eyes.

Examination equipment

What do you need to look? Really, if you could only have one thing for this, I'd recommend it be a Finnoff transilluminator. Other options include direct or panoptic ophthalmoscopes (as light sources and usually NOT via the ocular for corneal examination) or a good penlight and some loupes for magnification. A hand-held slit lamp or slit lamp biomicroscope

Examination technique

Start with a lights on, hands off exam – this is where you make your across the room observations, looking for overt rubbing at the eye(s), squinting, ocular discharge, periocular redness or swelling, and corneal cloudiness, redness or surface changes. You also can make an initial visual function assessment as the animal moves about in the lobby or exam room and you talk to clients about the history and presenting complaint.

Next up is a lights on, hands on exam – an exam table is often helpful here to get the patient's head and face at your eye level. Check the head, face, eyelids and each eye in turn for overt issues, then assess each eye in turn with direct, diffuse AND focal illumination for a quick overview.

For lights off hands on exam, an exam table and proper, steady head restraint, are even more important. Using a transilluminator or an ophthalmoscope with the light aperture set to a large spot and light intensity high enough to permit/facilitate the exam but also still reasonable comfort and tolerance by the patient, start arm's length away and use retroillumination (back-lighting with the tapetal reflex) to assess pupil size, shape and symmetry and notably, to highlight even subtle changes / opacities / interruptions in the clear optical media (including the cornea) that you might otherwise miss. Seeing them at this stage will allow you to be aware of them and look for and localize (cornea, anterior chamber, lens, vitreous) and define them on further specific exam whereas I guarantee, many would be missed altogether on up close and personal exam if not "spotted" here first. It's akin to jumping to 40 or 100x on a microscopic evaluation of a cytology specimen. Again, just look for anything "off" even if you know nothing about eyes. The beauty of this is that the cornea is normally intact, smooth and clear, so anything altering its surface or opaque, is abnormal.

Next up is close corneal inspection using diffuse and focal (i.e., with the light source closer to the eye, +/- smaller light aperture/spot) direct illumination at a variety of angles and

across the entire corneal surface. Look for appropriate sheen (versus lackluster/dull/dry or gritty surface), any opacities, and any other surface irregularities

You might stop there, but if you want to get into this cornea business a little, the meat of the matter is with a focused slit beam (and ideally magnified view) exam. You can either "be a slit lamp" – use a focused slit aperture light (from a slit lamp or direct or panoptic ophthalmoscope using the slit or smallest spot) and ideally magnified view (through magnifying head loupes of some sort) at a convergent angle OR use an actual slit lamp (hand held Heine, Eidolon, etc. or slit lamp biomicroscope) to provide you both.

Project the small and sharply focused light source obliquely across/through the eye to create an optical cross section/Purkinje images. This allows the observer to localize and determine depth of lesions within the corneal stroma, as well as to assess for corneal thickening/expansion/raised lesions and/or corneal stromal loss/thinning/devoting. These principles also apply to the anterior chamber, lens and anterior vitreous.

The concept (physics of light / optics, not really ophthalmology at all) behind slit beam use relies on passing light being bent differently by different substances and thus highlighted at interfaces of different / transitions from different media (with Purkinje images) such as from air to tear film/corneal epithelium – Purkinje image #1; and from aqueous humor to the anterior lens capsule. If we go more in depth, though, really there are more of these transitions: 1 - from air to tear film/corneal epithelium, 2 - from corneal endothelium to aqueous humor in the anterior chamber, 3 - from aqueous humor to the anterior lens capsule, and 4 - from the posterior lens capsule to the vitreous (Figure 1). Understanding the ocular cross sectional anatomy and these transitions and how associated visualized light from an obliquely projected slit beam appears across them normally, and also how it may be altered by lesions/disease states allows excellent exam capability, demonstrating localization and depth of lesions within the ocular structures. Figure 2 provides a diagrammatic example showing the distortion of the first and second Purkinje images with a thickened cornea, a mid-depth stromal corneal opacity (between the first and second highlighted Purkinje images), corneal endothelial debris (right up against the second Purkinje image), aqueous humor flare, cell and fibrin clot (all between the second and third Purkinje images), an anterior cortical lens opacity or cataract (just behind the third Purkinje image), and anterior vitreal cell (behind the fourth Purkinje image) (Figure 2A); and the distortion of the first and second Purkinje images with a thinned cornea (specifically a Descemetocele, where they essentially touch because there is no remaining stroma between them) (Figure 2B). This highlighting / identification of corneal thinning is a very important function of a slit beam as not all stromal / deep corneal ulcers look "divoted" to the naked eye even with close inspection, and yet recognizing such stromal loss or thinning of a cornea is a critical part of corneal disease assessment.

Other diagnostics

Schirmer tear test (STT)

A healthy tear film is essential to maintain a healthy cornea and thus the STT is a component of a thorough corneal evaluation. It is performed with the goal of ruling in or out an aqueous tear deficiency having a role in corneal disease. In veterinary medicine, the routinely

clinically utilized Schirmer tear test I uses a standardized STT strip to provide a quantitative measure of the basal plus reflex aqueous component of tear production in response to a noxious stimulus over one minute. (The rarely used Schirmer tear test II provides a quantitative approximation of the basal aqueous component of tear production with the ocular surface topically anesthetized and dried.) Normal canine reflex tear production (STT I) is equal to or greater than 15mm of wetting in one minute. Wetting values between 10 and 15mm are borderline and should be serially monitored with significance interpreted in conjunction with history and clinical signs and findings. Notably, the STT does not fully assess tear quality, and thus it may be normal despite a tear film lipid or mucin deficiency or abnormality and associated ocular surface disease.

STT technique and interpretation "pearls"

The STT (I) should be performed FIRST in the ophthalmic examination, before anything aside from your initial peek and without topical anesthetic, lubricants, etc. applied, to reduce the effect of reflex tearing from ocular manipulation and stimulus.

You should always* check both eyes as it may provide a built in control/normal or at least frame reference or important information to take into account. For example, the STT I of an eye affected with a painful (and reflex tear promoting) corneal ulcer may be stimulated into the normal range by the problem, prompting a missed diagnosis of a contributory tear film issue if the other eye (perhaps measuring low due to underlying keratoconjunctivitis sicca (KCS) in both eyes) is not assessed.

*The STT should not be performed in the face of deep corneal ulceration or other cases of significant corneal thinning and thus fragility/risk of perforation/rupture. Even minor trauma of the STT may also be contraindicated in a recently healed non-healing corneal ulcer with still fragile / weakly adherent corneal epithelium. Finally, though most species of animals can develop dry eye (and associated ocular surface/corneal disease) warranting it as a differential diagnosis consideration to always have at least in the back of your mind, the STT is usually only routinely performed/assessed in dogs, and infrequently performed in cats, exotic species, etc., as it may be difficult to interpret (e.g., normal cats may have a STT I of 0mm of wetting in one minute) or there may not be available normal reference value ranges. The phenol red thread tear test provides an alternative option for measurement of basal plus reflex aqueous tear production in cats' and smaller animal species' eyes.

Tear film break up time (TFBUT)

Again, because a healthy tear film is essential to maintain a healthy cornea, the TFBUT is a component of a thorough corneal evaluation. It is performed with the goal of ruling in or out a qualitative (lipid and/or mucin) tear deficiency having a role in ocular surface or corneal disease. This test measures the time it takes for a uniformly distributed (through the observer manually blinking the eyelids after fluorescein dye application, then holding them open) layer of fluorescein dye (highlighting the tear film) to break up or dissociate from the corneal surface and form dark spots. Normal TFBUT is ~20 seconds and this is reduced (accelerated) in many ocular surface disease states.

Other methods of tear quality assessment

Tear film quality may also be evaluated through meniscometry (measuring the tear meniscus radius), interferometry (measuring tear film thickness and the lipid layer), meibometry (measuring the meibomian lipid along the eyelid margin) and or conjunctival biopsy with analysis of goblet cell density/index, as these cells are responsible for producing the mucin component of the tear film.

Fluorescein and Rose Bengal staining

These stains evaluate the surface (tear film and epithelium) health of the cornea and are thus also integral to thorough corneal evaluation.

Fluorescein dye

Though fluorescein dye has many uses in ophthalmology, including assessment of tear film quality through TFBUT above, assessment of nasolacrimal duct patency (Jones test) and assessment for active aqueous humor leakage via a corneal wound (Seidel test), the most common is to detect corneal ulceration through fluorescein staining. Fluorescein dye (which is hydrophilic) is normally not retained by the "water hating" epithelium, but when that is not intact or missing, it IS taken up by the "water loving" stroma and highlights the epithelial defect / stromal exposure. This is best viewed after application of fluorescein dye +/- saline eye wash rinsing, with a cobalt blue filtered light source in a dimly lit or dark environment.

Fluorescein technique and interpretation "pearls"

Fluorescein dye solutions are excellent growth media for bacteria and should not be saved/stored once made up.

Fluorescein "pseudostaining" uptake is not uncommon where a STT strip contacted the cornea, especially if the animal's tear film is unhealthy; with tear film disorders; and/or corneal surface irregularities (gritty or raised lesions; or facets (epithelialized stromal loss or healed corneal ulcers with residual thinning) where dye may pool).

Two types of corneal ulcers may have characteristic fluorescein staining patterns. In primary or secondary superficial non-healing ("Boxer" or "indolent") ulcers, fluorescein may slowly leak under the defining loose epithelial edge, resulting in an area of bright green uptake surrounded by hazier uptake with time. With Descemetoceles, which it may be safest to not stain with fluorescein to reduce patient and ocular manipulation and increased risk of perforation, the exposed hydrophilic stromal walls take up fluorescein but the hydrophobic Descemet's ("water hating" epithelial basement membrane) does not. It is exposed because by definition with this type of ulceration, there is extreme (total) stromal loss down to that layer.

Rose Bengal dye

Rose Bengal staining is used in the same manner as fluorescein (though viewed with non-filtered white light), but detects / is taken up by devitalized (not necessarily missing) epithelium and or regions deficient in tear film mucin.

Corneal cytology

Cytologic evaluation of corneal lesions is indicated in cases with corneal infiltrate. After application of proparacaine or other topical anesthetic, a cytobrush or back end of a scalpel blade is used to collect a sample from the infiltrated cornea by gentle scraping (this may be dangerous and even contraindicated in cases of deep corneal ulceration at risk of perforation). If the lesion is epithelialized (fluorescein negative), it will be less likely to yield a diagnostic sample unless iatrogenic corneal ulceration is created; the pros and cons of doing so for this test must be weighed. Regardless, the sample is spread onto a clean glass microscope slide and allowed to dry. Clean (no "ears or rears") diff-quick staining of the slide allows in-house cytologic evaluation, or unstained slides may be submitted to a clinical pathologist for assessment.

Corneal cytology technique and interpretation "pearls"

Again, sample the area of / the infiltrate (where the "action" is), not the surrounding cornea or conjunctiva.

On analysis, look for epithelial cells, which indicate a good sample, and assess their health and for intracellular organisms, inclusions, etc.. Also look for inflammatory cells and assess who's there? how many? status? Similarly, look for organisms including bacterial (cocci, rods), fungi/yeast, as well as dysplastic or neoplastic cells.

Corneal culture and sensitivity

Culture of corneal lesions is indicated in cases with corneal infiltrate, especially if infection suspected. Ideally, sample(s) are collected early in the corneal evaluation, without topical anesthetic if safely/comfortably possible, using a microtip culturette swab gently rubbed over the area of infiltrate. Be careful to avoid contamination with the conjunctiva, eyelids, etc.. These may be used to inoculate growth medium plates or sent to a microbiology lab (prompt plating maximizes yield) for aerobic bacterial culture and sensitivity. Fungal culture in small animals is uncommonly indicated in cases of specific predisposition/concern (patient status (systemically and/or topically immunocompromised), infiltrate plaque, cytology results); but almost always indicated in addition to bacterial culture in horses and other large animals due to their increased environmental risk of fungal keratitis.
Sensitivity results should be interpreted "with a grain of salt" as well as taking in to account the clinical picture, as with topical therapy safely achievable drug concentrations may exceed those tested and yield different results.

Serial evaluation and response to therapy

Serial monitoring evaluation to gauge disease progression, especially with assessment of response to empiric or specific directed treatment, can be very useful in the evaluation of corneal lesions. Never underestimate the value of rechecks to help you determine what is coming and what is going / what is active or inactive!

Corneal biopsy

Biopsy of the cornea may be indicated for refractory, especially progressive mass or other undiagnosed corneal lesions and this may be diagnostic (via cytology and or histopathology) and or therapeutic. Corneal biopsy is generally a referral procedure and is frequently combined with adjunct therapy (cryotherapy or other).

Corneal lesion interpretation

Corneal opacities – the color wheel

So now we know how to look at and evaluate the cornea; what do we do with what we see? Interpretation begins with having a list of differentials for the corneal opacities or corneal colors.

Red

Red corneal opacities include: vascularization/vessels, granulation tissue and stromal hemorrhage/blood – all indicating chronic corneal irritation (keratitis). Less common red corneal opacity differentials include hemangioma/hemangiosarcoma and conjunctiva (pink-red) such as after grafting/flap surgery or via aberrant migration onto the cornea.

Brown / black

Brown corneal opacities include: pigment, neoplasia (limbal/conjunctival or iris/ciliary body melanocytoma/melanoma), dematiaceous fungal infection, foreign body (usually plant material) and sequestrum (cats only).

Corneal pigment may arise/migrate from the conjunctiva (superficial) and indicate chronic corneal irritation and/or previous vascularization (keratitis); arise/migrate from the endothelial limbus (endothelial pigment migration) as a usually incidental finding in middle-aged and older dogs; or it may have come from the iris through persistent pupillary membrane attachment, anterior synechia, corneal perforation with iris prolapse, iridociliary cyst exfoliation or rupture, or iridociliary mass exfoliation or contact.

Feline corneal sequestra may be from faint/weak tea staining to golden brown to burnt cookie to brown (to black) in color. They indicate chronic corneal irritation associated with breed/facial/ocular conformation and or feline herpes virus 1 infection.

Silver

Metallic foreign bodies may present silver corneal opacities.

Blue

Blue corneal opacification may be subtle/hazy grey-white “blue” to overt blue; and focal to diffuse. It indicates corneal edema, which can arise from epithelial (ulceration) and/or endothelial dysfunction (dystrophy, degeneration, endothelitis/uveitis, glaucoma, direct damage from lens luxation or other contact (ppm, cyst, synechia, neoplasia)), or leakage from corneal vascularization of any cause. In addition to blue opacification with corneal edema, the cornea thickens and bullae (blisters) may be present.

White

White corneal opacities are trickiest because they have the most differential diagnoses that are also often harder to differentiate. The differentials include: scar/fibrosis, Haab’s stria, infiltrate (cells present that should not be) and deposit; and as above, when more subtle (and not blue), edema.

Corneal scar/fibrosis may be subtle/hazy grey-white to dense white; and focal or diffuse. By definition it is "inactive", and usually appears that way. Considering the clinical picture (history – e.g., previous issue now static and comfortable; ophthalmic exam findings – e.g., "quiet eye", relatively well-defined opacity (at least with close and critical inspection), fluorescein negative, etc.) may help allow determination of a white corneal opacity as scar.

Haab's stria(e) are grey-white, softly linear Descemet's membrane (deep corneal) defects occurring with or after elevated intraocular pressure (glaucoma).

Corneal infiltrate may present as diversely as its causes (from infectious, sterile/immune-mediated inflammatory, to neoplasia or cystic) and thus appear creamy yellow/green-white, "soft or melting" to "white-white", as well as sometimes thickened/raised, cystic or plaque-like, all mostly/usually with ill-defined borders. The presence of corneal infiltrate, both because it is by definition an active and potentially eye threatening condition, and because it has varied underlying causes with different therapies, warrants at least consideration of signalment, history, overall ophthalmic examination findings and further corneal diagnostic evaluation (cytology, culture, serial evaluation and response to therapy +/- biopsy) to aid differentiation and diagnosis as discussed above.

Examples of corneal infection associated infiltrate (from subtle to plaque (raised) include infected corneal ulcers and stromal abscesses (bacterial or fungal).

Examples of sterile/immune-mediated inflammation include German Shepherd-like pannus and feline eosinophilic keratitis.

Keratic precipitates (KPs) are technically corneal opacities (usually in the white category though they may also be red or brown depending on their cell type) but indicate intraocular, not corneal disease (specifically uveitis). KPs are white blood (white, creamy yellow to tan in visible color), red blood, pigmented or neoplastic cells in the anterior chamber smattered against the corneal endothelium (generally more inferiorly due to gravity and aqueous humor convection current).

Neoplastic corneal infiltrate may have variable appearance from thickened/raised, grey to white-white, or pinkish/vascularized lesions. The most common (whitish) corneal tumor types are squamous cell carcinoma and papilloma. (The other most common corneal neoplasias are hemangioma/hemangiosarcoma (red as above), and melanoma (arising from, and brown as above)).

Corneal epithelial inclusion cysts may occur after trauma (commonly surgical or not always witnessed (spontaneous?)) "implants/entraps" surface epithelium within the corneal stroma. They are usually clear-ish to "fatty"-creamy-yellow-white fluid-filled/cystic (round, raised/nodular/thickening the cornea) structures. They may enlarge with time and potentially cause usually minor complications (though they can be full thickness and even rupture in to the eye), and thus may warrant intervention from "de-roofing" and debridement, to excision via keratectomy.

Corneal deposits generally have sharp borders, though they may be irregular. Lipid/cholesterol deposit opacities are usually sparkly or opalescent and occur with inherited corneal dystrophy (typically seen in young dogs of certain breeds (Cavalier King Charles Spaniel, Boxer, etc.) as bilateral and symmetric, non-painful lesions); post-inflammation (degenerative); topical steroid use (steroid keratopathy); systemic metabolic disease (hypothyroidism or other issue with fat metabolism); or infrequently solely high dietary fat intake. Mineral deposit opacities are usually more chunky, like a "dried soap bar", sometimes plaque-like, and may slough like scabs. These occur with calcific corneal degeneration, which is an age related / degenerative process in older and often metabolically compromised dogs, with secondary ulcerative keratitis as a common complication; post-inflammation (degenerative); or systemic metabolic disease (uncommon mineral imbalance, hyperadrenocorticism). Corneal drug precipitates are infrequently seen with topical ciprofloxacin, and rare other medications, administration.

Green

Aside from the occasional green plant foreign body, green corneal opacities are seen with positive fluorescein staining / fluorescein dye uptake, defining corneal epithelial loss or defect (erosion/abrasion/ulceration) +/- or stromal exposure. Corneal ulceration indicates trauma, an unhealthy / predisposed cornea (e.g., from KCS), or viral infection (herpes – a rare true primary epithelial pathogen); and when present risks secondary infection thus warranting topical antibiotic therapy until resolved.

Pink

Pink corneal opacities include Rose Bengal dye uptake (indicating corneal epithelial devitalization and or mucin deficiency), conjunctivalization (from previous surgery or aberrant migration), neoplasia, or vascularization/granulation tissue especially when combined with corneal edema and or fibrosis, which make them appear less red.

Haired skin &/or conjunctiva

Dermoids (choristomas) or aberrant islands of haired skin and or conjunctiva with or without pigment may occur on the cornea, resulting in opacification.

"Zebras"/other weirdos

It is not possible to predict all corneal opacity "colors" or other appearances and thus every differential, but the information above provides pretty close to an exhaustive list. And if you can detect and identify the opacity, you're well on your way to making the diagnosis….appropriate management (intervention, monitoring, implications) follows from there. Voila, corneal disease made simple/simpler.

Corneal ulcers and alterations of thickness

Seeing and identifying an opacity and differential diagnosis list is most of the story but for corneal examination and interpretation, there is a little more besides opacities.

Corneal erosions/abrasions/superficial ulceration (by definition not affecting the thickness of the cornea) may be diagnosed with positive fluorescein staining/uptake and

frequently associated discomfort, ocular discharge and redness, and or focal, usually relatively mild, corneal edema.

Corneal ulceration with stromal loss (thinning the cornea) is where the use/importance of a slit beam to determine depth and thus urgency and option considerations come in to play. These stromal ulcers may be superficial, mid or deep (including down to Descemet's membrane (Descemetocele)); sometimes obviously "divoted" or thinned as indicated by clearing in an otherwise cloudy and thickened corneal region (where little to no stroma remains to be opaque with edema and vessels in the surrounding area), but sometimes very hard to judge depth. In addition to determining their depth, appropriate corneal ulceration management is aided by assessing for and addressing any associated corneal infiltrate, "softness/melting/malacia and or (reflex) uveitis, as well as taking in to account lesion vascularization and location.

Corneal facets are areas of epithelialized (fluorescein negative) stromal loss (thinning) post previous ulceration.

Increased corneal thickness may occur with vascularization, edema*, infiltrate, or neoplasia/cysts as above.

Summary and Purkinje image figures

Armed with these differential considerations for almost every corneal lesion/disease, assessment/interpretation includes looking for/processing opacities, loss of thickness, or thickening, and then appropriate diagnostic testing. The other eye and the clinical picture (signalment dictating breed predispositions and or environmental risk; history – general, ophthalmic including topical and other medications/exposure and response; ophthalmic signs and exam findings; and systemic status) may also provide very useful information to guide and refine differential diagnosis and ultimate case management.

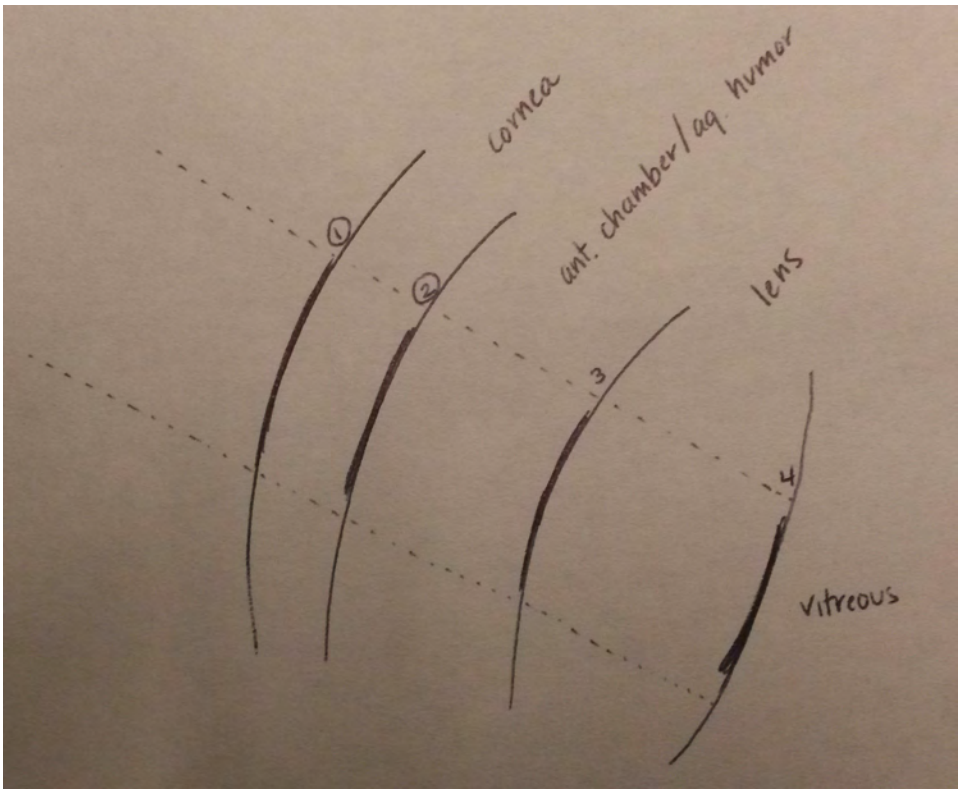

Figure 1 The Purkinje images. Light from a slit aperture is projected from left to right and across the eye. 1- from air to tear film/corneal epithelium, 2- from corneal endothelium to aqueous humor in the anterior chamber, 3- from aqueous humor to the anterior lens capsule, and 4- (often difficult to visualize without mydriasis) from the posterior lens capsule to the vitreous. This view and these images highlight the outside and inside of the cornea and its uniform thickness, the uniformly deep anterior chamber, and the anterior and posterior lens surfaces with the vitreous behind it.

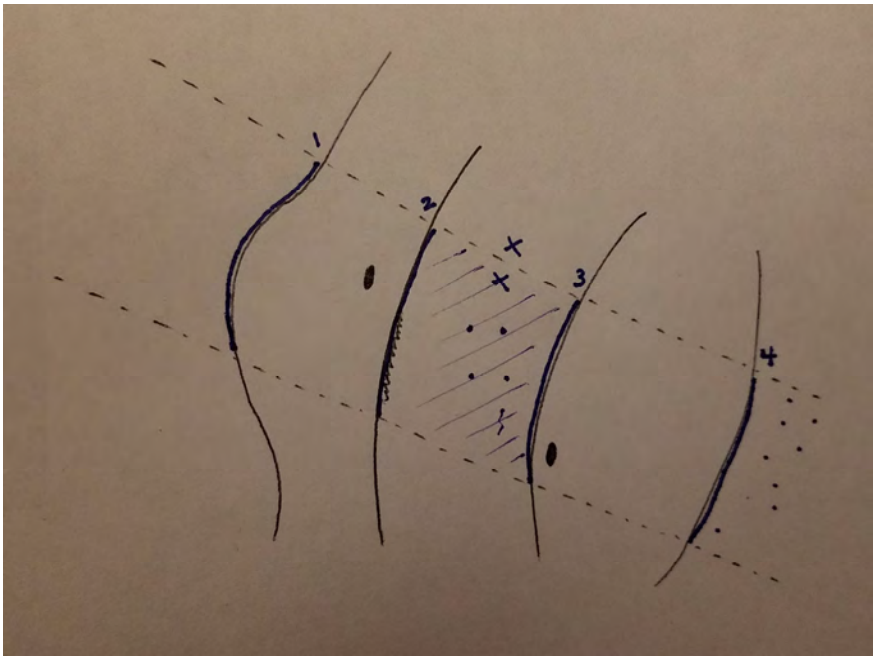

Figure 2A The Purkinje images highlighting and allowing localization of pathology. Light from a slit aperture is projected from left to right and across the eye. This view and these images highlight the thickened cornea, mid-depth stromal corneal opacity, corneal endothelial debris, aqueous humor flare, cell and fibrin clot, an anterior cortical lens opacity or cataract, and anterior vitreal cell.

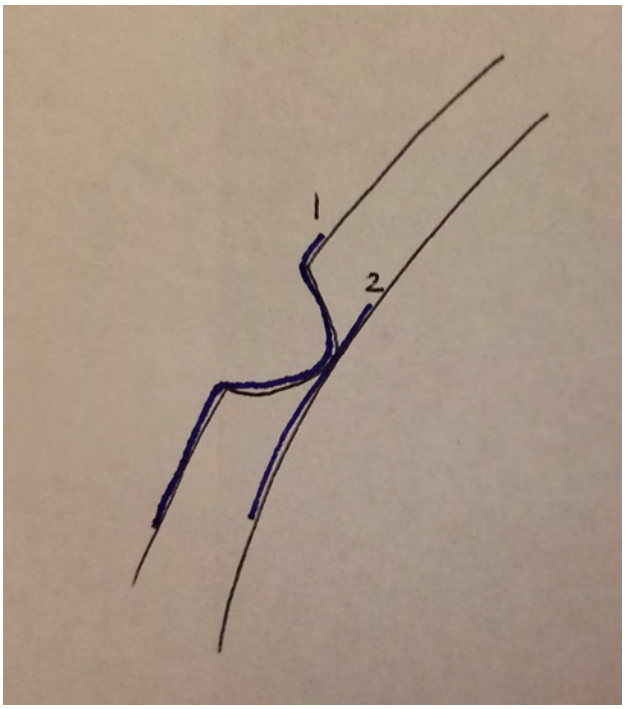

Figure 2B The Purkinje images highlighting and determining depth of pathology. In this diagrammatic cross sectional representation of a Descemetocele, light from a slit aperture is projected from left to right and across the eye. This view and these images highlight the thinned cornea with Purkinje image 1 approaches 2 because there is no corneal stroma remaining between them.

THE LENS AND CATARACTS

Kathryn A. Diehl MS, DVM, DACVO
University of Georgia College of Veterinary Medicine
Athens, Georgia

This lecture will briefly review clinically relevant lens anatomy and examination techniques to help identify and localize cataracts. It will then cover in depth different classification schemes of cataracts including causes. Finally, treatment of cataracts will be explained with concentration on a soon to be available promising medical treatment for diabetics, as well as surgery and indications for such.

With respect to clinically applicable lens anatomy, it is often useful to think of and explain the lens like a peanut m & m shaped like a regular m & m. It is comprised of the (continuous anterior, equatorial and posterior) lens capsule and anteriorly, the associated epithelium as the "candy shell", the cortex as the chocolate, and the dense central nucleus as the peanut. The actual lens cells or fibers are elongate, arising from the metabolically active equatorial region or lens bow and spanning from the front to the back of the lens, meeting at / forming the lens sutures. After being produced at the equator lens cells are sequentially compressed in to the lens center by new growth/cells.

A cataract, by definition, is any opacity within the lens. Such opacities occur due to disruption of the normally perfect/orderly lamellar arrangement of the lens fibers and thus light's passage through/interaction with (refraction (bending of light) and reflection) the structure. This disruption in varying degrees, may then affect the ability of the lens to "do its job" of focusing light (through refraction) and images onto the retina, with light scatter and ultimately blurred vision. Despite this potential visual impairment (with loss of menace response and/or object tracking), even with a complete cataract, the afferent (retina, optic nerve) and efferent (parasympathetic fibers of cranial nerve III, iris sphincter / pupil constrictor muscle) arms of the pupillary light reflex should be intact/functioning, and the pupil should react normally to light.

To evaluate for cataracts, especially to pick up more subtle and/or posterior lesions, first perform an ophthalmic examination from arm's distance away using a light source (pen light, transilluminator or otoscope head, direct ophthalmoscope (set at 0 diopters if looking through the ocular) in a darkened area. Collect a tapetal reflex to "back-light" the lens. Lens opacities will block the tapetal reflex and be highlighted as a darker or shadowed area within the pupil. Then perform a closer evaluation, ideally with a slit beam or other focused and bright light source (creating an optic cross section view or image) +/- magnification to localize a cataract within the lens and pharmacologic mydriasis (pupil dilation) with short-acting/diagnostic tropicamide (a topical anti-cholinergic / parasympatholytic agent). An optic cross section view provides Purkinje images where the first beam highlights the cornea, the black space the anterior chamber, the second beam the anterior lens capsule, the Tyndall effect of light scatter through the lens (protein) thickness including the anterior cortex, the nucleus and the posterior cortex, and finally the third beam the posterior lens capsule.

Cataract cause classification scheme / reasons for cataract development

Cataracts may be classified and named/diagnosed within several different categories, including their reason for development, size, location within the lens and age of onset.

Inherited/genetic

This is the most common cause of cataracts in dogs. In this species cataracts should thus be presumed inherited until proven otherwise (known diabetes, trauma, etc.). Canine inherited cataracts occur in many breeds and in some there is a classic appearance. One common example is dominantly inherited triangular incipient posterior polar cataracts of Golden and Labrador Retrievers as well as some other large breed dogs. Fortunately these only rarely progress to cause clinical significance for the individual patient, but obviously affected animals should not be bred to avoid worse issues in the offspring.

Metabolic

Diabetes mellitus – This is a common cause of canine cataracts; the incidence of cataract formation in diabetic dogs is 80% within 16 months of diagnosis (Beam, S; Correa, MT: Davidson, MG. Vet. Ophthalmol. 1999; 2(3) 169-172), while it is rare in diabetic cats. Diabetic cataracts are usually bilateral, complete, develop rapidly and cause vision loss. Due to their tendency to occur and progress rapidly, they also commonly have Y suture clefting.

Cataracts occur with diabetes due to altered lens metabolism of glucose. In a normoglycemic state, lens metabolism of glucose occurs first via breakdown of glucose by the enzyme hexokinase to glucose-6-phosphate. In a hyperglycemic state, hexokinase is overwhelmed and glucose metabolism is shifted to the enzyme aldose reductase and a pathway that produces sorbitol, which accumulates in the lens cells. Sorbitol is a large, osmotically-active molecule, and its accumulation ultimately leads to lens cell swelling and rupture. The disruption of lens fibers then leads to cataract formation.

Hypocalcemia – This is an uncommon cause of cataracts with a typically bilateral, multi-focal punctate appearance similar to a “snow-globe” opacity appearance within the lens. This should not to be confused with that appearance within the vitreous, which indicates asteroid hyalosis, a degenerative or post-inflammatory change not uncommonly seen. The ophthalmic examination with an “optic cross section” is used to differentiate these locations/depth within the eye.

(Post-)inflammatory (in a way, toxic)

Inflammation (uveitis/vitritis of any of many causes (systemic or local/ocular)) is the most common cause of cataract formation in cats and horses. Particularly in those species then, but in dogs as well, it is important to look for hallmarks of (current/active with anterior chamber flare +/- relative miosis, red eye and low intraocular pressure and) prior inflammation (uveitis), including posterior synechiae (adhesions of the iris to the lens) and iris hyperpigmentation. Patients/eyes with cataracts in this category are generally poor candidates for cataract surgery due to significantly increased risk of post-operative complications of retinal detachment and secondary glaucoma.

Traumatic

Cataracts may be caused by **blunt or sharp/penetrating trauma to the eye**. Blunt cataracts are usually actually caused by inflammation/uveitis ("toxin") caused by the trauma. Sharp cataracts may occur due to direct lens fiber disruption as well as from uveitis. Blunt traumatic cataracts are managed as any other along with any other traumatic injury and likely uveitis present. Sharp traumatic cataracts are similarly managed but lens capsule integrity must also be considered as lens capsule tears pose an additional problem. Capsular tears greater than 1.5mm in length generally will not self-seal and this can lead to severe lens-induced uveitis that is refractory to treatment (phacoclastic uveitis) – an exception to this rule is found in puppies, which can often (though not always) overcome severe phacoclastic uveitis. Generally though in cases of phacoclastic uveitis, treatment is thus more urgent cataract surgery not only to address the lens opacity as with typical cataract surgery, but also and almost more importantly to remove the inflammatory-inciting leaking lens material from the eye before secondary complications (retinal detachment, glaucoma) occur. In cats lens capsule damage is associated with the risk of post-traumatic sarcoma development, thus especially with sharp traumatic cataracts, long term close monitoring and ultimately possibly enucleation over cataract surgery is warranted.

Electrocution may cause cataracts and is most often encountered in young animals chewing on electric cords or with lightning strikes. Obviously there may be other issues/injuries to attend to that "trump" the lens opacities.

Dietary

Nutritional cataracts may occur in orphaned or nutritionally supplemented/supported puppies (and some other species) fed milk replacer due to arginine and other amino acid deficiency. Some ophthalmologists advocate adding beef or liver baby food to the milk replacer to reduce this risk. Milk replacer-related cataracts typically occur bilaterally at the nuclear – cortical junction and fortunately, usually don't progress and in fact become relatively smaller with age as the lens grows around them and compresses them centrally.

Age-related or senile / degenerative with oxidative stress

These cataracts are incredibly common in older dogs with cumulative damage to the lens cells (by UV light radiation, free radicals, etc.) but fortunately rarely affect vision and thus are often rightfully benignly neglected. They are often located at the equatorial cortex and wedge-shaped, as well as punctate cortical lesions.

Radiation induced

In addition to UV light radiation contributing to inducing senile cataracts as above, radiation induced cataracts have been reported to occur in 10-28% of dogs with the eye in the field of ionizing radiation, even when appropriately/adequately protected or shielded. They generally occur 6-12 months following radiation therapy. The often start in the lens equator, as well as anterior and posterior subcapsular regions. They may or may not progress but are rarely treated surgically because of ocular surface / corneal disease (dry eye, keratitis) that is even more common in eyes in fields of RT +/- radiation retinopathy change preempting good candidacy and prognosis. Additionally there is often the "big-picture" disease and systemic status to consider in these cases (i.e., why the patient was receiving RT in the first place).

Toxic

Toxic cataracts may occur after exposure to certain drugs, most notably though still infrequent and not well described, ketoconazole. Toxic cataracts may also occur secondary to exposure to "natural" toxins such as those released into the vitreous and ultimately permeating the posterior lens capsule and lens tissue, from dying retinal cells (dialdehydes). This is common in dogs with progressive retinal atrophy (PRA) and is important to recognize as these patients are not good surgical candidates. This cause of / association with cataracts is one major reason for performing electroretinograms to assess retinal function before cataract surgery is performed, as if it is abnormally low, surgery may not be indicated to remove the opaque lens because the visual impairment is also and regardless (without treatment options available), retinally based. Prior to going down the road of pursuing pre-operative testing for cataract surgery though, signalment (PRA and secondary cataracts common in Labs and Poodles) and history can help suggest this underlying disease in patients being evaluated for cataracts as owners (especially when probed) often report dim light visual deficits and even blindness before, THEN the ocular cloudiness/opacity (of the cataracts).

Anomalous/congenital

Congenital cataracts by definition are present at birth. They may be due to developmental "hiccups" or less often, inherited.

Vascular

Aberrant retention of (sometimes hyperplastic) ocular fetal vasculature (which may be inherited or a random developmental "hiccup") that normally regresses before or soon after birth, may result in cataracts (usually congenital) if it contacts the lens. Examples include iris to lens persistent pupillary membranes (ppms) anteriorly and persistent hyaloid artery (potentially with blood or bleeding into the lens if there is an associated defect of the back of the lens (posterior lenticonus and or absent capsule/capsular coloboma))/persistent (hyperplastic) primary vitreous/persistent (hyperplastic) tunica vasculosa lentis (PHPV/PHTVL) posteriorly.

Infectious

Infection within the lens may occur via septic implantation during penetrating trauma. This may result in cataract formation and progression even years after the inciting incident. The most common example of this scenario is after penetrating cat scratch/claw trauma/injury.

In rabbits, *Encephalitozoon cuniculi* (usually through intrauterine transplacental vertical transmission though spore ingestion or even inhalation is possible) or *Pasteurella* may infect the lens and or iris forming an abscess/granuloma and associated cataract, as well as uveitis. *E. cuniculi* lens abscessation and cataract has also been described in cats (rare).

Iattrogenic/post-operative

After cataract surgery, some degree of post operative faint hazy to even dense white opacification of the retained lens capsular bag, left in place to hold an intraocular lens implant if placed and or serve as a barrier to vitreal prolapse, is expected. This is called (posterior) capsular opacification (PCO) or capsular after "cataract" or capsular fibrosis and occurs due to migration and abnormal proliferation of residual viable lens epithelial cells that are not completely removed or killed at surgery.

Cataract size classification scheme

Punctate

With the advantage of slit lamp biomicroscope evaluations, this is first category, which is as it sounds, a pinhead sized opacity.

Incipient

These cataracts involve less than 10% of the lens volume and typically don't affect vision. They can easily be missed on exam, especially when posterior, if diffuse retroillumination to back-light them with the tapetal reflex is not employed. Additionally, due to their small size and varying with location, they may be best visualized after pharmacologic mydriasis or dilation of the pupil (with tropicamide).

Incomplete (immature)

These opacities involve greater than 10% of the lens volume but not the entire lens. As this is obviously a broad category, it is sometimes further subdivided into early and late incomplete cataracts. These lesions variably affect vision, depending on their size and location within the lens.

Complete (mature)

These cataracts, as their name implies, involve the entire lens and are almost always associated with visual impairment (though slightly variable due to variable density of the opacity and "coping" function/ability of the patient.

Resorbing (hypermature)

These cataracts are starting to liquefy; sort of the body's way of doing its own cataract surgery. Cataract resorption often occurs with chronicity but also in very rapid-onset and progressive cataracts, for example in inherited, juvenile cataracts of Cocker Spaniels and several other breeds. Hallmarks of resorbing cataracts include a sparkly appearance, wrinkling of the anterior lens capsule, a deep anterior chamber and sometimes discernable (on exam itself and definitely with ocular ultrasound) decreased lens thickness. As lens resorption and leakage of lens proteins out of / through the lens capsule and into the eye often causes phacolytic lens-induced uveitis, other signs to look for are those associated with intraocular inflammation, including anterior chamber flare +/- relative miosis, red eye, low intraocular pressure, posterior synechiae and a velvety smooth and/or hyperpigmented iris. An uncommon specific type of resorbing cataract is a Morgagnian cataract, which occurs when the lens cortex is so markedly resorbed away that the residual nucleus sinks inferiorly within the lens capsule.

Intumescent

In these cataracts, the lens fibers and thus the lens itself becomes markedly swollen and stretching the lens capsule. This results in shallowing of the anterior chamber and sometimes secondary pressure elevation or even overt glaucoma. It can also result in rupture (bursting) of the lens capsule (usually posteriorly where it is thinnest or near the equator) and then phacoclastic uveitis. This type of cataract is most commonly associated with diabetes (rapid onset osmotic cataract) or other rapidly progressive cataracts.

Cataract location classification scheme

Recall the lens anatomy. An incomplete or smaller cataract can occupy a specific/focal region(s) within the lens and can thus be classified based on this location as below. During clinical examination, the location of lens opacities is most readily and best determined using a slit beam to create an optic cross section or slice through the eye and lens, highlighting the anterior and posterior lens capsule with bright, convex and concave respectively, lines of light, and then assessing relative depth and position of lesions.

Capsular

Capsular cataracts may affect the anterior or posterior lens capsule or shell. They rarely progress. Typical causes include uveitis, congenital / developmental abnormalities (especially incomplete regression of the embryologic vascular supply to the lens) and genetic.

Subcapsular

Cataracts just under the capsule may be anterior or posterior.

Cortical

Cortical cataracts may affect the anterior or posterior cortex. Anterior cortical cataracts are more likely to progress than posterior cortical ones.

Equatorial cortical cataracts involve the equator or periphery of the lens and are often missed or difficult to see without retroillumination and ideally, for more thorough evaluation / assessment, pharmacologic mydriasis or pupil dilation. Cataracts in this location often progress (unless senile) because this is the most metabolically active region of the lens.

Nuclear

Nuclear cataracts form in utero and are thus usually congenital. They may be inherited or associated with developmental accidents or “hiccups”. Fortunately they rarely progress and in fact, relatively-speaking, often become smaller with age as the nucleus is compressed centrally within the lens.

Cataract age of onset classification

Congenital

By definition, congenital cataracts are present at birth. They may or may not be inherited.

Juvenile

Juvenile cataract develops after birth and varying with breed, up to about six years of age. They are very commonly inherited in cause.

Senile

As the name implies, senile cataracts occur after about 6-10 years of age. Age-related cataracts are very common and one study documented some degree of cataract formation in all dogs greater than 13.5 years of age (Williams, DL; Heath, MF; Wallis, C. Vet. Ophthalmol. 2004; 7(1): 29-35). Fortunately these usually do not significantly affect vision nor progress.

***Nuclear sclerosis** is a normal age-related opacification of the lens nucleus associated with increased density due to lens fiber growth around it throughout life (without increase in lens size/volume) and resultant compression centrally. In dogs and cats it begins around age 6 years – though not really generally visibly so until 8-10 years. In humans it begins about age 40 years and results in decreased accommodative ability and presbyopia. Nuclear sclerosis should be differentiated from a true cataract opacity and can be as the former generally does not affect vision (whereas a complete or near complete cataract in the same central location likely would); and with nuclear sclerosis the tapetal reflex is still present and the fundus/retina can be visualized (not be the case with a complete or near complete cataract). Finally, differentiating nuclear sclerosis from cataract is often easier with pupil dilation allowing visualization of the clear(-er) cortical halo around the dense central nucleus.

Cataract therapeutic interventions

Once cataracts are identified and ideally classified, treatment options come into play. In terms of medical management, there have been some highly publicized/advertised/marketed topical therapies that are touted to "melt away" cataracts. The old adage that says if it sounds too good to be true it probably is applies here. These eye drops are generally anti-oxidants, specifically N-acetyl carnosine and other ocular health vitamin supplement agents marketed under several names. They may in fact reduce oxidative damage to the lens and in a controversial study (as lens changes were very subtle and difficult to measure objectively with photographs as lighting and angle of exposure alter the results significantly and are nearly impossible to keep totally consistent, and the principle investigator refutes the findings stating his words have been manipulated by the pharmaceutical companies) did decrease lens opacity in cases of nuclear sclerosis and incomplete cataracts (Williams, DL; Munday, P. Vet. Ophthalmol. 2006; 9(5) 311-316. However, they do not eliminate or slow further progression of significant cataracts that we see in dogs that actually warrant treatment due to their visual impact (late incomplete and complete), probably due to the relatively large size of the canine lens and the high density of cataract opacities in this species. The bottom line is that these may be "useful" for cases where treatment is not really indicated as there is no visual impairment or other complication, but not for those already visually impaired. Furthermore, these medications are generally expensive, and can provide a false sense of security to clients as they think they're managing things with the drops and thus seek veterinary evaluation and attention later in the disease course when secondary changes like lens induced uveitis, resorption, lens luxation, retinal detachment and/or secondary glaucoma, etc. may have already occurred with chronicity, and now preempt successful intervention.

Ocu-GLO Rx™ is a maybe more worthwhile oral nutraceutical containing a combination of 12 safe (and effective in generally supporting and protecting ocular health and normal function, boosting overall immune health, and scavenging destructive free radicals) antioxidant ingredients and formulated specifically for dogs. Considered a vision supplement, it is probably most useful in potentially delaying progression of retinal disease (progressive retinal atrophy (PRA) and other degenerative diseases, maybe sudden acquired retinal degeneration syndrome (SARDS) and immune-mediated retinopathies, etc) and cataracts that are secondary to such retinal disease (toxic cataracts) or prior to formation/early diabetic cataracts. It will not reduce existing opacities but depending on cause, might delay progression of such, and has possible utility in decreasing post-cataract surgery capsular scarring/fibrosis opacification. Finally, Ocu-

GLO Rx™ may also benefit (and it is certainly unlikely to harm though it is expensive) other ocular disease conditions such as uveitis, glaucoma, and Golden Retriever uveitis.

A more promising potential medical therapy for diabetic cataracts is the use of aldose reductase inhibitors. This has been shown to be effective in delaying the onset and severity of cataracts in galactosemic (essentially an experimentally induced diabetic state) (Sato, S; Mori, K; Wyman, M; et. al.. Exp Eye Research 1998; 66(2) 217-222) and more recently diabetic dogs, and may ultimately also even be effective in treating these cataracts once they occur. Unfortunately these drugs (topical and systemic) are not commercially available for use at this time.

Cataract surgery

Considering these issues with medical treatment options, at this time, surgery is the only proven and reliable, effective way to restore vision lost due to cataracts. Surgery employs phacoemulsification or ultrasound energy to break up the opaque lens and it is irrigated and aspirated out of the capsule. The success rate in good/ideal candidate canine patients is 90-95%.

The ideal candidate for cataract surgery is:
-systemically healthy or at least managed/regulated/stable,
-a manageable patient - intensive post-operative medical therapy and follow-up are vital to success and the patient, client/owner and veterinary ophthalmologist must be able to tolerate and handle this!
-has vision affected by cataract/is impaired or functionally blind at least in the affected eye(s), making the potential benefit/gain of surgery worth the cost/risks,
-has no or at least controlled lens induced uveitis - previous or refractory intraocular inflammation poses an increased risk of post-operative complication(s), especially retinal detachment and glaucoma)
-does not have lens resorption – if present, this significantly increases the risk of pre-existing or post-operative retinal detachment
-has normal pre-operative electroretinogram (ERG) and ocular ultrasound (normal lens shape and no capsule disruption, non-degenerate vitreous (as when present slightly increases the risk of retinal detachment), no pre-existing retinal detachment, no residual embryologic vascular supply)

Risk factors for cataract surgery sort of naturally follow from the above list of qualities/factors of an ideal candidate. Specifically though, risk factors include lens induced uveitis (LIU) and lens resorption as above; breed predisposition to retinal detachment (in Bichon Frises and some other breeds as well as patients with significant vitreal degeneration); breed predisposition to glaucoma (Cocker Spaniels and many other breeds predisposed to primary glaucoma (and thus also secondary) with/by goniodysgenesis (an abnormal (narrowed) drainage angle)); and being a Boston Terrier – multiple studies have shown that the single biggest risk factor for serious (potentially devastating with ultimate blindness and loss of the eye/need for removal due to pain) post-operative complications (corneal ulceration, inflammation, retinal detachment, GLAUCOMA) is being a Boston Terrier!

Several of the potential complications of surgery are eluded to above but most importantly, as they are vision threatening and in the latter case, painful, include retinal detachment and glaucoma. At surgery, inability to place an intraocular lens implant may occur,

though even in this case, vision should still be improved, though far-sighted or hyperopic, like our vision underwater. Other possible issues include infection (potentially devastating endophthalmitis as the eye does not tolerate/handle infection and the associated secondary inflammation well); corneal ulceration, chronic and refractory inflammation/uveitis, posterior synechiae which may be cosmetic, increase the risk of secondary glaucoma or rarely be visually significant; and especially in young dogs, lens fiber regrowth often inciting lens induced uveitis and possibly though rarely affecting vision again – sometimes warranting surgery to alleviate the stimulus of inflammation and any visually significant opacity (remove regrowth).

Also as mentioned above when discussing the ideal candidate for cataract surgery, indicated/appropriate pre-operative testing includes electroretinogram (measuring/assessing hopefully normal/adequate retinal function) and ocular ultrasound (hopefully ruling out pre-existing retinal detachment, etc. as above). These tests are warranted as for good surgical candidates likely complete or nearly so cataracts preclude retinal examination and these tests allow the best possible evaluation and assessment to determine that the retina "checks out" okay. If it does not, it probably doesn't make sense to remove the cataract through somewhat invasive, expensive surgery – this is sort of like the situation if there is no film/data card in the camera, there's no point in removing the lens cover.

Other cataract pre-operative testing indicated includes screening lab work to assess for underlying systemic disease that may have caused the cataracts in the first place but more importantly for safe candidacy for general anesthesia and aggressive peri- and post-operative anti-inflammatory medical therapy needed with this procedure. In diabetic patients for the same reasons, glucose regulation should be good and stable (clinical signs controlled, blood glucose curves with nadirs not lower than 80mg/dl and peaks less than 350mg/dl; fructosamine < 450-500). It is expected that they will suffer some dysregulation with the stress of anesthesia and surgery, and frequently with necessary aggressive peri-operative anti-inflammatory therapy, etc., so it is ideal if everyone is comfortable and on-board with where they were and stay the course without reactionary insulin dose changes in the "rough" period shortly afterwards. Additionally in diabetic patients a urine culture, even if not indicated by the sediment findings of the urinalysis, should be performed pre-operatively as these dogs are prone to silent urinary tract infections and any bacteria in the body can seed the eye particularly when peri-operative uveitis breaks down the blood aqueous barrier. Bacterial infection within the eye can result in devastating endophthalmitis, frequently with vision and ultimately eye loss (enucleation). For this same reason, the skin (and ears) should be assessed, especially in allergic or otherwise predisposed patients, for signs of pyoderma (and otitis). In the case of UTI or pyoderma, appropriate antibiotic therapy should be initiated and continued through a negative culture and possibly the post-operative period to reduce risk of infection especially with intraocular lens implant (nidus potential). Finally, another pre-operative requirement or at least strong recommendation is good/reasonable periodontal health, again due to the risk of bacteremia with periodontal disease readily seeding the eye at surgery. Thus if/as indicated by oral/dental disease, a dental cleaning should be performed at least two weeks prior to cataract surgery, with peri-procedural antibiotics as indicated and also just time for expected bacteremia to clear. Though this all may seem excessive, remember that cataract surgery is an elective procedure with significant risk. Having all of the "ducks in a row" in an ideal candidate can maximize success.

Surgery is performed with phacoemulsification under an operating microscope. An incision is made superiorly in the cornea paralleling the limbus. A capsulorrhexis (hole in the anterior lens capsule) is then made to access the lens contents. The phaco. needle delivers ultrasound energy to fragment the hard nucleus of the lens while continuous irrigation and aspiration flushes and vacuums it out. The remaining lens cortex is then aspirated out of the hopefully still intact (it is very thin and easy to rupture or tear during the procedure) lens capsular bag. An intraocular lens is then inserted into the capsular bag. These are generally foldable acrylic lenses of varying diameter sizes as indicated for the patient, and in dogs usually with dioptric power of 41 to restore normal/emmetropic vision (through bending of light rays through/by the lens into focus on the retina) in the majority of patients. They are injected with special cartridges and instruments. Previously and sometimes still currently on occasion, a rigid polymethylmethacrylate lens is used – this requires a larger corneal incision and generally longer surgical time. Regardless, the corneal incision is then closed and the road to recovery begins. The postoperative therapy is as important as what happens in the operating room / at surgery – it is absolutely vital to the success of the procedure and entails initially somewhat intense topical and systemic anti-inflammatory medications (ultimately slowly tapered as indicated) and close/frequent monitoring with serial rechecks at increasing intervals. Because the inflammatory response incited by dogs to exposure to their own lens proteins (with any pre-operative lens induced uveitis and then at and after surgery itself) and the ultrasound energy requirement (high and long) to break up the relatively large and dense canine lens (about 8 times the size of a human lens and usually operated relatively later in the game when the lens is harder…) result in significant inflammation, afterwards weeks to months and even years are spent combating / hopefully controlling it and many canine post-cataract surgery patients remain on some level of topical therapy and monitoring long term to forever.

What if cataract surgery is not an option? It may not be elected or the patient may not be a candidate, etc.. In these cases, it is still indicated to monitor and treat any lens induced uveitis (with topical anti-inflammatory medication) to avoid or at least decrease the risk of discomfort and secondary glaucoma or other complications. Occasionally, if lens resorption occurs and is advanced, without in the process inciting significant inflammation and causing retinal detachment or secondary glaucoma, it may result in enough clearing of the lens and visual axis that some vision (albeit far sighted/de-focused) may be restored/gained.

In terms of visual impairment, if only one eye is affected, most veterinary patients function essentially normally, though longer-nosed and highly visually reliant working dogs may demonstrate some/more deficits on the affected side and astute owners may notice this. If patients are bilaterally affected and visually impaired to even completely functionally blind, fortunately the vast majority of dogs adapt amazingly well and have good quality of life, especially when their vision loss is gradual (with slow onset or progressing cataracts) or given time to adjust even when it is rapid. They will frequently memorize their environments and some owners don’t even recognize how impaired they are. However, with things out of place or novel environments, not surprisingly they may have more trouble, so ideally their environments should be kept as stable as possible to help them adjust and cope. This and other useful tips are available through many great resources for owners of visually impaired pets, in book and online forms.

GET TO KNOW THE NICTITATING MEMBRANE

Kathryn A. Diehl MS, DVM, DACVO
University of Georgia College of Veterinary Medicine
Athens, Georgia

Conjunctiva review

Reminder that the conjunctiva (conj) is the light pink (vascularized with thin vessels) tissue/mucosa that lines the eyelids and eyeball and allows smooth movement of the eyelids and globe. It has lymphoid tissue/immune function, frequently pigment (melanocytes/melanin), and connective tissue. It is not sterile (surface microbial flora – mostly gram + bacteria).

Palpebral conjunctiva lines the inside of the eyelids, reflects back at the fornix and then somewhat loosely lines the front of the globe (up to the limbus) as the bulbar conjunctiva. Palpebral conjunctiva of the third eyelid lines its outer/anterior side and bulbar conjunctiva of the third eyelid lines its inner side.

Nictitating membrane

Nictare = to blink in Latin.

The nictitating membrane/membrana nictitans, nictitans, TEL, haw, palpebra tertia, or plica semilunaris, as its name implies, is an eyelid structure. It lies between the inferior eyelid and the cornea in the nasal/medial inferior conjunctival fornix. It is variably pigmented with the margin (or leading or free edge) and a portion of the palpebral or anterior face (aspect/side) commonly pigmented. This variation, particularly when asymmetric can give rise to concerns of pathology (may create optical illusion of abnormal position, etc. or make an eye seem “red”).

Poorly defined attachments secure the base of the NM to the periorbita surrounding the inferior rectus and inferior oblique extraocular muscles. Musculature of the NM is largely vestigial in domestic species and movement is generally passive (except in cats where there is some muscular contribution to active protrusion) across the eye with retraction / retropulsion of the globe (in the former case by the retractor bulbi m innervated by the abducens n (CN VI)) – some animals with eyelid or exposure related conditions may “learn” to blink with the NM in this manner.

Movement (except in birds) is from inferior nasal/medial where the NM originates, to superior temporal/lateral.

Normal NM position is determined by a combination of orbital, eyelid and globe conformation and sympathetic tone of orbital and periocular smooth muscles, which govern globe position within the orbit and thus the NM’s passive movement associated with that position.

Again, lined on both sides by conjunctiva that allows its smooth movement.

Like “regular eyelids” it serves to provide physical protection to the globe/ocular surface/cornea. It also has important functions in contribution to tear production as well as distribution and drainage of the tears / tear film across the ocular surface through blinking. Finally it serves an

immune function through the presence of lymphoid tissue (CALT), specifically numerous superficial lymphoid follicles on its bulbar surface.

The conjunctival bi-lined (tightly adherent at the free margin, loose deeper and over the gland) nictitating membrane gains structure / support / rigidity from its internal T cartilage with a vertical portion extending perpendicularly from near its margin/free edge towards the inferior medial canthus/orbit where its base is encircled by the gland, and its horizontal portion extending near (about 1.5mm away) and along its margin or the leading edge.

The gland of the NM is located at the (orbital) base of the third eyelid on its bulbar or back side, again, at the base of the vertical T cartilage – hence, like the cartilage, it is not visible in the normal state. The gland is seromucoid and in the dog, has adrenergic and (denser) cholinergic innervation. In the pig and many rodents, a portion of the gland or a separate Harderian gland is found deeper within the orbit.

Tear film

Recall tear film is trilaminar with outer fatty / oily layer, middle aqueous portion (with up to 50% produced by the GNM), then mucin layer. There is a basal tear production and then an additional reflex component in response to stimuli. Tear film disease may have roots in NM disease.

Nictitating membrane examination

Look at it! Appearance, position, movement. Palpebral and bulbar aspects.

Diagnostics: cytology, culture and sensitivity, biopsy, response to therapy, work up of underlying issue

Nictitating membrane disease conditions

Abnormal position/movement – Horner syndrome, orbital disease, enophthalmos of other cause, tetanus, dysautonomia, Haws syndrome, physical restriction,

Inflammation, follicles – episcleritis (nodular granulomatous episcleritis), follicular conjunctivitis, pannus (“atypical”)

Follicular conjunctivitis: young, usually large breed dogs; response to environmental exposure; possible precursor to overt allergic conjunctivitis; bulbar conjunctival follicles with variable irritation; treatment = topical steroid +/- anti-histamine; rinsing

Glandular disorders including prolapsed gland of the NM and post-operative complications; keratoconjunctivitis sicca

Prolapsed gland of the nictitating membrane (PGNM): Inherited (or post-inflammatory) weakness of connective tissue attachment anchoring GNM; treatment = surgical repositioning / replacement; risks = keratoconjunctivitis sicca, cyst formation, failure/recurrence

Cartilage anomaly/eversion

Young large breed dogs; chronic exposure conjunctivitis and associated redness & discharge, but mainly cosmetic; may predispose to, cause or be associated with PGNM;

treatment = benign neglect, medical management (lubricant, steroid), manual correction, third eyelid flap, excision of bent cartilage portion, thermal cautery to straighten

Tumor/mass – inflammatory (immune-mediated), infectious (esp. parasitic), neoplastic

Neoplasia – adenoma / adenocarcinoma of GNM most common primary tumor but many possible – lymphoma, mast cell tumor, melanoma (AGGRESSIVE here), squamous cell carcinoma, hemangioma/hemangiosarcoma (USUALLY more benignly behaving)

Trauma – laceration, other damage; foreign body – look!!

Excision – with risk of iatrogenic keratoconjunctivitis (sicca)

EXCERPTED FROM

Drs. Katie Diehl and Kate Myrna present:

OPHTHALMOLOGY SURGERY WET LAB

UGA College of Veterinary Medicine Continuing Education

Athens, GA

August 7, 2016

INTRODUCTION

Ophthalmic surgery instruments/supplies

Small, sharp and precise! Designed to be handled with the fingertips under adequate lighting and often with magnification.

Essential instruments include:

Barraquer wire eyelid speculum (small or pediatric)

Tissue forceps

-Delicate Adson or other rat tooth 1 x 2 forceps

-Bishop-Harmon forceps (1 x 2 0.5mm teeth for eyelids; 1 x 2 0.3mm teeth (delicate) for eyelids and conjunctiva)

-(0.3mm Colibri-style forceps for conjunctiva)

Scalpels and blades

-#3 Bard-Parker scalpel handle with #15 blade for eyelids

-(Beaver handle/chuck with #6400 or #6500 blade for conjunctiva)

Scissors

- Stevens tenotomy scissors (curved or straight)

-Westcott tenotomy scissors (spring action handles) for conjunctiva and fine suture

Needle holders

-Derf needle holders / drivers for 5-0 to 4-0 suture material/needles

-Microsurgical needle holders (fingertip-controlled, spring action handles; curved or straight, locking or non-locking) for 6-0 and smaller suture material/needles

Additional useful instruments and supplies include:

Clear plastic, adhesive drapes (Steri-Drape™), Schaedel cross action towel clamps, tongue depressors or Jaeger lid plate, Jameson caliper or STT strips, cotton tipped applicators, cellulose sponge spears (Weck-Cel® sponges), 2x2 or 3x3 gauze sponges, irrigation cannulas, Jameson muscle hook, Gelfoam®, small Metzenbaum scissors, small Mayo scissors, Mosquito and Kelly hemostats, sterile tubing for stents, IV catheters

Surgeon's stool, adequate lighting – options via Universal Surgical Instruments

Ocular surface lubricants: Celluvisc/hyaluronic acid, Genteal gel, Puralube

Small clippers

Magnification – OptiVisor, surgical loupes

Instrument Care

Ophthalmic instruments are easily damaged and quickly worthless unless properly handled.

Storage: Store in a separate pack (various specialized trays are available) where instruments cannot rub against each other or individually wrap and sterilize them.

Cleaning: After use, rinse with distilled water and gently brush to remove blood and tissue. Subsequent ultrasonic cleaning with mild detergent, distilled water rinsing and air drying is best. Inadequate cleaning results in rust, which can be removed by soaking affected instruments for 12hrs in equal parts ethyl alcohol and aqueous ammonia.

Sterilization: Gas sterilization is best. Small sections of silicone tubing may be placed over instrument tips to reduce risk of damage during packing and sterilization. Steam autoclaving may be used but may dull instruments and cause corrosion. Cold sterilization is not recommended.

MOST DULLING AND CORROSION of instruments IS DUE TO IMPROPER CLEANING AND HANDLING.

Ophthalmic Suture

Suture **needles**: Ophthalmic microsurgical needles have "spatula" tips to allow suture to pass in the same layer (lamellae) of the tissue without cutting deeper or shallower. The higher the number assigned to a needle the smaller its radius of curvature. Handle (some 5-0 and all) 6-0 and smaller suture material/needles with microsurgical needle holders as Derf needle holders will bend the needles.

Suture **material**:

Absorbable for subcutaneous, conjunctival and episcleral tissues

Polyglactin 910 (vicryl) – generally braided except very small sizes; good tensile strength for ~20 days; fairly reactive

Poliglecaprone 25 (Monocryl) - monofilament

Polydioxanone (PDS II) - monofilament

Nonabsorbable for skin

Monofilament nylon – chronically strong and inert

Braided nylon – strong and inert with softer tags; requires additional throws to hold knot; wicking potential in between monofilament nylon and silk

Silk – braided with potential wicking (uncommon issue if sutures removed in timely manner (~10 days))

Ophthalmic surgery patient considerations

Anesthesia

-For extraocular procedures, sedation/anesthesia is much the same as for other surgeries.

-In sedated/anesthetized animals, **exposure-related corneal damage** risk is increased due to altered palpebral reflexes with frequent lagophthalmia and decreased tear production. Un-operated eyes should be generously lubricated with an artificial tear

ointment. Operated eyes (unless being enucleated) should also be lubricated with artificial tear solutions/gels or irrigating saline.

-**Monitoring** anesthetic depth is often more difficult in ophthalmic surgery because of the presence of draping over the animal's head. Pulse, pulse quality, respiratory rate, blood pressure and ECG should be observed.

-Traction on extraocular muscles and the optic nerve or pressure on the globe may incite the **oculocardiac reflex**, with resultant bradycardia or even cardiac arrest. Both the anesthetist and surgeon should be aware of this potential, avoid it if possible and quickly correct it if encountered, by promptly releasing the globe and when resuming intervention, proceeding more gently.

Patient and surgical field preparation: Differ slightly with procedure, but in general:

-Elevate the down eye off the table (with a rolled towel under the neck or otherwise) to prevent it from trauma and/or contacting a pool of betadine that has run off from preparation of the eye to be operated.

-**Trim the eyelashes** near flush with the eyelid margin with small scissors lightly coated with K-Y jelly or artificial tear ointment along the blade away from the cornea to catch cut lashes and reduce risk of them contacting the ocular surface/being retained there.

-When applicable (eyelid procedures, enucleation, etc.), carefully **clip** an appropriately sized border around the margins of the eyelids using a #40 blade on an electric clipper or other small electric clipper. Gently blot the area with tape to pick up remaining loose hairs.

-Either **lubricate** the ocular surface before clipping to protect it from stray hairs OR do so AFTER cleaning/rinsing the conjunctiva (including fornices) post clipping to reduce risk of loose hairs actually getting caught up in the lubricated ocular surface.

-To avoid excessive lid and conjunctival edema, BE GENTLE. All antiseptic preparations are toxic to intraocular structures, and should not be used to flush the conjunctival sac if the corneal/scleral shell has been breached or intraocular surgery is anticipated.

-**Conjunctival sac prep**: Rinse and wipe with dilute (half-strength aqueous / "weak tea") povidone-iodine solution (NEVER SCRUB) with flushes of sterile saline. Sterile cotton tipped applicators should be used to apply and wipe the povidone-iodine, taking care not to touch the cornea. The fornix should be swabbed first working out toward the eyelid margins. Rinse immediately with saline and repeat the cycle for a total of three times.

-**Periocular skin prep**: Prepare with gentle alternate applications of dilute (half-strength aqueous / "weak tea") povidone iodine solution (NEVER SCRUB and NEVER chlorhexidine which may cause a severe toxic keratitis) and sterile saline. Work from the eyelid margins outward. Repeat the cycle for a total of three times.

Patient and Surgeon Positioning:

-Patient**:** Depending on the surgery to be performed, the animal is placed in dorsal or lateral (or ventral for some eyelid and third eyelid procedures) recumbency with the **head close to the head of the table** and rotated such that the **palpebral fissure of the operated eye is parallel to the table/floor**. This position allows improved ease of focus especially if magnification is used for surgery. Vacuum (Vac-packs), sandbags, or other moldable beanbags or other padding or supports, as well as adhesive tape, to maintain this head position are particularly helpful for ophthalmic procedures, again especially those requiring magnification. **Be careful to avoid excessive kinking of the neck with impact on patient ventilation / CO2 level.**

-Surgeon**:** The surgeon usually **sits** at the head of the table, **resting his or her forearms for stabilization**. Care must be taken to not put pressure on the patient or anesthetic equipment.

Postoperative Care:

-Trauma to the surgical site should be avoided and this is accomplished by ensuring a smooth anesthetic recovery and placement of an Elizabethan collar as needed.

-Discomfort should be managed with appropriate anti-inflammatory and/or opioid medications.

-Indicated medical therapy, monitoring and follow-up should be pursued as indicated.

THIRD EYELID FLAP

Indications: **Uncommonly performed to encourage/expedite healing of non-healing corneal ulcers** when bandage contact lenses are not retained, tolerated or effective. **Or to "un-train" everted third eyelid cartilage anomalies.**

Presurgical considerations:

-A third eyelid flap is **contraindicated for corneal ulceration complicated by infection, malacia, and/or stromal loss (including perforation)**

-A third eyelid flap does NOT provide blood supply nor structural support to the cornea

-**Complete corneal coverage by the third eyelid may not be physically possible in breeds/dogs with conformational/physiologic exophthalmos** (e.g., Pugs) and **an alternative option should be considered and pursued** as indicated

-**Topical therapy will be significantly impaired** by the blockage of the third eyelid tissue over the ocular surface.

-**Visualization of the ocular surface for disease monitoring will not be possible** with the third eyelid flap in place.

-Third eyelid flap placement may apply **undesirable rubbing forces and pressure on the cornea/ globe**.

-**Ensure proper indication**, client understanding and, that there are no foreign bodies, aberrant cilia or even excessive irregular follicles (which may cause irritation to the ocular surface and cause, perpetuate or otherwise exacerbate corneal ulceration) under the eyelids or especially under the third eyelid.

Technique:

-**General anesthesia** (usually); or sedation and/or regional blocks is needed.

-Surgical preparation includes carefully clipping and prepping (with povidone-iodine as above) a small area of skin over the superior orbital rim. The eyelashes are also clipped and the ocular surface cleaned as above.

-Using **2-0 to 4-0 (based on patient size) non-absorbable suture**, (if a stent/stents are to be used, first pass the suture through one at this time) **enter the deep superior lateral**

conjunctival fornix via a bite through the overlying prepared skin. Take care to exit cleanly and avoid damage to the globe

-With awareness and caution regarding suture from the fornix laying across the ocular surface, **elevate the third eyelid and take a bite paralleling its leading margin from lateral to medial and incorporating/around the vertical T cartilage near its junction with the horizontal T cartilage. Ensure that this bite is not full thickness**/does not breach the bulbar aspect of the third eyelid.

-The final bite is back out the deep superior conjunctival fornix medial to the initial entry about equidistant to the length of the third eyelid bite (so that the suture bites form a rectangle) to the skin.

-The suture is tightened, elevating the third eyelid margin deep into the superior lateral conjunctival fornix, pulling the third eyelid over the ocular surface, and tied with or without stent placement.

-Leave suture tags long if attempted untying and replacement without a second procedure is anticipated/desired.

Possible complications and further considerations:

-An e-collar should be used as needed to prevent self-traumatic exacerbation of disease.

-Appropriate medical management of the ulcer (topical antibiotic, oral NSAID/opioid, e-collar, etc.) should be continued until it is healed though again, topical therapy effectiveness will be significantly reduced with the third eyelid flap in place.

-Recheck in 10-14 days (or sooner if the patient seems worse after 24-48h though this may be hard for the client and veterinarian to assess). Sometimes, it is possible to untie the knot securing the third eyelid flap and temporarily reduce it to examine and evaluate the ocular surface and globe conditions, then replace the flap as indicated – this may require sedation. Regardless, repeat or other interventions to promote healing until the ulcer heals are indicated.

-There is risk of suture rub or "cheese-wire" irritation/ulceration during third eyelid flap placement, or if such is positioned wrong (not deep enough in the fornix or full thickness through the third eyelid) or loosens post-operatively.

-The corneal ulcer or other ocular surface condition as well as intraocular disease may worsen under the flap, especially if exacerbated by pressure applied to the globe (e.g., a deep corneal ulcer may perforate).

-The patient will be at least temporarily blinded by the third eyelid flap, and this can be a significant concern/issue in patients with bilateral disease or only one eye.

-The eyelid skin may develop pressure necrosis if the sutures are placed too tightly

-The suture may pull through, releasing the flap but also harboring retained suture that may irritate the ocular surface.

PROLAPSED GNM REPLACEMENT

Indications: Replacement/repositioning of prolapsed gland of the third eyelid ("cherry eye") to reduce risk of development of dry eye and exposure conjunctivitis, and for cosmesis.

Presurgical considerations:

-Replace, **do NOT remove these glands**!! Removal carries a significantly increased risk of development of **dry eye (KCS)**. Even with replacement, animals are at a slightly increased risk of development of dry eye.

-There are several options for replacement with two basic categories: pocket imbrication and tacking (most commonly to the orbital rim)

-The condition is common in young dogs (under 2 years of age) and certain breeds, especially brachycephalics. Beware the "cherry eye" in older patients and especially atypical breeds as these may be associated with other issues including third eyelid neoplasia or chronic inflammation.

-In predisposed breeds, the condition is commonly bilateral – if only one eye is affected at the time of the surgery, the client should be warned that the other eye could be affected and warrant surgery in short order. It is reasonable to wait for this potential for a short period of time so that bilateral surgery may be performed under the same anesthesia if indicated.

-Chronic prolapse has a poorer the prognosis for successful repositioning and avoidance of dry eye.

Technique:

Pocket imbrication: Creates a subconjunctival pocket to house the gland. Does not impact third eyelid mobility/function. **Magnification necessary**.

-**General anesthesia** is needed.

-Surgical preparation includes carefully clipping the eyelashes and povidone-iodine cleaning as above.

-Elevate and evert the third eyelid by grasping it medially and laterally near (not at) its margin on the palpebral side with mosquito hemostats (pinching just a bit of overlying conjunctiva). This will expose the prolapsed gland and its base on the bulbar surface of the third eyelid.

-Using a #15 blade, make an incision in the conjunctiva inferior to and along the base of the gland. Do not go full thickness through the third eyelid. Make a similar incision along the superior aspect of the gland (this will be near the back side of the leading edge of the third eyelid). **These incisions** form a sort-of "moat" **around the base of the gland** but they **SHOULD NOT MEET** at their ends.

-A subconjunctival **pocket is created** by blunt dissection via these incisions in both directions away from the gland (it is often difficult to dissect very far if at all superiorly due to the third eyelid leading edge, but only enough to take suture bites on that side of the incision is necessary).

-The **pocket is then closed, imbricating the gland** into it as follows:

To reduce risk of suture rub, knots are tied on the palpebral side of the third eyelid. Using **6-0 vicryl** take a bite of conjunctiva roughly over one end of the incision and tie a knot. Pass the suture through the third eyelid to the bulbar aspect exiting at the same end of the incision.

Appose the inferior and superior incisions* over the gland using a simple continuous/running pattern – initial sutures may be challenging due to the prolapsed gland being in the way but as bites are taken, the gland should "slurp" into the pocket and be out of the way, allowing completion of the closure.

*__With the non-connected inferior and superior incisions, the ends of the wound should be slightly open or puckered (i.e., closure is not complete/sealed)__ to allow egress of gland secretions and reduced risk of cyst formation.

With the incision now apposed, pass the needle back into the incision and through the third eyelid to the palpebral aspect overlying the same end of the incision (opposite end as the initial knot). Ensure appropriate tension and closure of the incision without "spanners" or undue puckering and take a bite of conjunctiva and tie the final knot.

Orbital rim tacking: Anchors the gland to the periosteum of the inferior orbital rim. This reduces third eyelid mobility/excursions and thus function. Magnification is not necessary.

-**General anesthesia** is needed.

-Surgical preparation includes carefully clipping the eyelashes +/- an area of skin over the orbital rim. The remainder of the preparation is as previously described above.

-Incise the skin (or conjunctiva accessed via the fornix) over the inferior nasal orbital rim with a #15 blade

-Using **2-0 non-absorbable suture (nylon, prolene)**, take a **bite of the periosteal rim** – this should be SOLID!

-**Tunnel up into the third eyelid and around the edges of the prolapsed gland (in a purse string like pattern)**, ultimately **returning to the periosteal bite** region (+/- taking a second bite at this point), and **tie**.

-If a skin incision was made over the orbital rim, it should be closed routinely.

Possible complications and further considerations:

-An e-collar should be placed during recovery and healing (~ 10-14 days) to prevent self-trauma and associated complications with possible immediate re-prolapse of the gland or otherwise delayed or complicated healing.

-Prophylactic topical antibiotic TID x 10-14 days, and oral NSAID (unless contraindicated by patient issue) x 3-5 days are indicated

-**Complications** include: **suture rub** with irritation and possible corneal ulceration especially with the pocket imbrication and especially if the knots are not tied on the palpebral aspect of the third eyelid, infection, wound dehiscence, **cyst formation**, **dry eye**, and **failure with re-prolapse of the gland/recurrence**, as well as again, prolapse of the contralateral gland.

-**Monitor tear production** long term due to risk of dry eye

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Canine Heartworm Disease

Chris Adolph, DVM, MS, DACVM (Parasitology)
Zoetis
Tulsa, Oklahoma, USA

Our ability to diagnose certain parasites has improved over time but is still limited by several factors. Ability to recover diagnostic stages, length of prepatent period, and host-parasite interactions are but a few considerations when determining if the result of the diagnostic test performed is accurate. In this presentation we will cover new information regarding the diagnosis of *Dirofilaria immitis.*

Antigen testing has long been the gold standard for heartworm detection in dogs and cats. However, we now know that some dogs and most cats infected with *Dirofilaria immitis* will fall below detectable limits when tested. Historically, low worm burdens, single sex infection, and testing in the prepatent period have been listed as reasons why a truly positive patient will fail to be detected. Additionally, most infective stages (L3) will not reach adulthood in the feline patient (while extensive pulmonary damage is done by the presence of larval stages) resulting in low prevalence when antigen testing alone is utilized. Several new studies indicate another phenomenon, known as antigen blocking, plays a role in false negative antigen tests. When a patient is infected with heartworms, the body mounts an immune response to those parasites. The degree of the response depends on the burden, length of infection, and the unique host-parasite interaction. Some patients will mount such a robust response that circulation heartworm antigen is bound by antibodies to the point that not enough antigen is available to be captured by the testing platform utilized, and the result will fall below detectable limits. To address this, serum samples from suspected cases can be heated to 103-104ºC for 10 minutes. This will destroy the antibodies, freeing the antigen, and making the available antigen available for detection. You will need to check with your laboratory to see if they are currently preforming this testing. Oklahoma State University is currently preforming this test and the form is available here https://cvhs.okstate.edu/sites/default/files/OADDL%20submittal%20REC-FM-001.07_%20fillable.pdf

Studies have shown that 7.1% of randomized dogs that were initially heartworm antigen negative were positive after heating the sample. In addition, 53.3% of dogs diagnosed with heartworm disease, placed on monthly macrocyclic lactone therapy in lieu of proper adulticidal therapy with melarsomine, tested negative at some time point were positive when heat treated. In another study, 64.7% of dogs suspected to be heartworm positive by the veterinarian and initially below detectable limits by antigen testing were positive when heat treated. Also, antigen has been detected in in dogs at 128 days post infection by this method.
Veterinarians should consider submitting samples for heat treating when clinical suspicion is high and results are BDL. In addition, dogs on monthly macrocyclic lactone therapy for adult heartworm infection in lieu of proper adulticidal therapy with

melarsomine should be monitored with heat treated sample testing every 6 months until 2 consecutive BDL tests are obtained.

Suggested Reading

1. Velasquez L., et al., 2014. Increased prevalence of *Dirofilaria immitis* antigen in canine samples after heat treatment. Veterinary Parasitology 206, 67-70

2. Drake J., et al., 2015. False negative antigen tests in dogs infected with heartworm and placed on macrocyclic lactone preventives. Parasites and Vectors 8, 68.

3. Little S., et al., 2014. Prevalence of *Dirofilaria immitis* antigen in feline samples after heat treatment. Proceedings, American Association of Veterinary Parasitologists Annual meeting, Denver, CO.

4. Gruntmeir J., et al., 2015. False negative antigen tests in dogs infected with heartworm (*Dirofilaria immitis*)-an update and case series. Proceedings, American Association of Veterinary Parasitologists Annual Meeting, Boston, MA.

Canine Ectoparasites

Chris Adolph, DVM, MS, DACVM (Parasitology)
Zoetis
Tulsa, Oklahoma, USA

The conversation between veterinary team member and client around ectoparasites (e.g. fleas, ticks) will vary greatly depending on geographic region. And within the same geographic region, the conversation will vary greatly depending on the ectoparasites being discussed. The goal of this conversation is to communicate the importance of year-round flea and tick control in every pet. A client that is well educated and has a full understanding flea and tick biology, ecology, and pathogens that can be potentially vectored is far more likely to invest in year-round protection than a client that believes their pets are not at risk. Here we review the basic concepts to communicate and an effective way in which to do so.

Fleas and ticks are completely different organisms. Fleas are insects and are more closely related to house flies. Basically, fleas are smaller, laterally compressed wingless flies. The life cycle is the same; egg, maggot, pupae, and adult. Ticks, in contrast, are arachnids and more closely related to spiders. The tick life cycle, like the flea, starts with an egg. However, the next stage, the larva, does not resemble a maggot. Rather, the tick larva is a very small six-legged version of the adult. After feeding on a host and detaching, the larvae will molt to a nymph. The nymph is slightly larger and has eight legs. After feeding on a host and detaching, the nymph will molt in the environment into an adult and will either be a male or female. So fleas undergo a complex (or holometabolous) metamorphosis in which all life stages look different than each other. And only the adult flea is parasitic. Tick undergo a simple (or hemimetabolous) metamorphosis, where the larvae, nymph, and adult all look similar, getting progressively larger with each molt. And all 3 of these stages are parasitic. All these differences will be important when addressing client pushback.

We have all discussed the flea life cycle with clients. I find the most compelling way to communicate how fleas work is to tell a story. I start with the fact that the adult flea starts from an egg laid in the environment week to months prior to the adult being seen. The egg quickly falls into the environment and hatches into a maggot. It will remain a maggot for a week or two, then form pupae. It can remain pupae for days to months. Once it emerges as an adult it acquires a host by jumping at shadows until landing on a suitable host. Adult fleas being feeding within minutes, reproducing, and producing the first egg as soon as 20-24 after acquiring the host. And the whole thing begins again. Adult fleas are obligate parasites, meaning they need to be on their host their entire adult lifespan. If they are dislodged, they will die within one to four days. This is a factor in more fleas being detected than ticks.

Ticks, on the other hand, have a different story. There are multiple species of ticks in North America that each behave differently. It is important to be familiar with the ticks in your geographic area. They differ in habitat, host preference, seasonal occurrence, longevity, and the pathogens they vector. All

hard ticks share similar characteristics. They acquire a host either by questing or ambushing a host. Ticks vary greatly in how soon they start feeding. Once feeding begins, they are attached for several days before detaching. Larval and nymphal ticks are quite small and often go undetected. Adult male ticks feed, but do not engorge, therefore male and unfed female ticks are hard to detect also.

A thorough knowledge of these differences will allow the team member to address client pushback. The most common reason clients don't use tick control is the perceived lack of need. They honestly believe they don't have a tick problem because they don't see them. Utilization of distribution maps from the Center for Disease Control can be very helpful here. https://www.cdc.gov/ticks/

But this often is not enough. Clients still can't see the ticks. Education centered around these facts can often convince a client; 1. Ticks often live for years. 2. They only feed on a host three times 3. 2 of those stages are so small they often escape detection 4. The largest stage, the adult, when unfed is also very small. So, when they say they don't see them, I believe them. Most human with a tick-borne pathogen do not remember being fed upon by a tick.

Another powerful message is to share your practices data on number of dogs positive for tick-borne pathogens. If you are not screening dogs every year for common pathogens, this is an important thing to add to your protocols. In the meantime, the Companion Animal Parasite Council has maps for the USA and Canada you can refer to. https://www.capcvet.org/maps/#2017/all/lyme-disease/dog/united-states/

Clients will often push back with lifestyle arguments. We let them know that any trip outside (e.g. outside to eliminate, short walk) is a threat. Some species of ticks do very well in urban environments.

Some clients believe that only seasonal control is necessary. Fleas are a year-round threat. And some species of ticks are most active as adults in the winter months. Therefore, year-round control is a must.

One last hurdle to overcome is client's safety concerns about using topically applied non-systemic or oral medications, and their desire to use a "natural" product. I praise them for using something and gain agreement that fleas and ticks are a concern. I also praise them on caring enough about their pet to research the topic. Here I pivot to what I use for my on pets and why. I use medications that have a demonstrated safety and efficacy profile. While what they are using may not have any outward adverse events, there is no compelling data on the efficacy of these products. Then I share my concern that their pets are at risk for contraction a tick-borne pathogen. Keep in mind that clients have strong opinions on this topic and not all will follow your recommendation, but it is worth the educational effort.

By slightly changing the way the veterinary health care team discusses fleas and ticks, clients will be better engaged and informed, and this will result in better compliance. When compliance is up, the patient, the owner, and the practice all benefit.

References/Suggested Reading

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Doggie Diarrhea: The microbiome, pre- and probiotics and what this means for K9 Gut Health in 2021

Michelle Evason, BSc DVM, DACVIM
Michelle Evason Veterinary Internal Medicine & Nutrition Consulting
Atlantic Veterinary College; IDEXX UK
mevason@upei.ca

Evaluation of the microbiome is a rapidly expanding area of research. However, evidence-based application of research as it relates to clinical canine cases (i.e., doggie diarrhea) can seem unclear. This session will summarize current knowledge on the microbiome and GI microbiota and discuss practical and specific therapeutic gut manipulation therapies (nutrition, pre- and probiotics, fecal transplants) in canine diarrhea (acute and chronic).

Definitions of key terms are important to understanding of the session:

Microbiome refers to the total number of microorganisms and genetic material.
Microbiota refers to the microorganisms in different ecosystems in the body, e.g., the gut.
Microbiota dysbiosis is an altered composition (change, shift) in the host's 'normal' bacterial microbiota.
Probiotics are live microorganisms which are thought to provide a health benefit when administered in adequate amounts.
Prebiotics are nondigestible food ingredients that stimulate the growth and/or metabolism of one or more health promoting bacteria.
Fecal microbial transplant (FMT) is the administration of a fecal suspension from a donor into the gastrointestinal tract (colon) of a recipient.

The microbiota of the intestinal tract provides the animal (dog, human, etc.) with nutrition, modulation of the immune system, and defence against pathogens. At this time, we think that the intestinal microbiota is composed of 4 main bacterial phyla: *Firmicutes, Bacteroidetes, Proteobacteria,* and *Fusobacteria*. While characterization of *gut dysbiosis* is in the early stages, recent metabolomic studies indicate the consequences (and role) of gut dysbiosis in some disease states.

In healthy animals, ‘normal bacteria’ (aka, non-pathogenic) work towards synthesizing vitamins (e.g., folate, biotin, B12, vitamin K) and essential amino acids. Additionally, these bacterium deconjugate bile acids, ferment gut luminal contents, and produce hydrogen, methane, ammonia, sulphur dioxide and short chain fatty acids (SCFAs).

As research on the microbiome evolves, we have also become more aware of the impacts of antimicrobials and other drugs on gut health. Specifically, studies on metronidazole and tylosin, which are commonly used in canine GI disease. This research seems to indicate that we may be doing ‘more harm than good’ with these treatments for various canine diarrhea presentations.

Canine diarrhea (acute and chronic):

To date, only a few studies have described the fecal microbiota of dogs with acute and chronic GI disorders. In dogs with acute diarrhea, particularly those with acute haemorrhagic diarrhea (AHD) there are significant alterations in the microbiome vs. healthy dogs. Dogs with clinically active IBD demonstrate decreased *Faecalibacterium* spp. and *Fusobacteria*, which subsequently increase upon resolution of the IBD. Overall, work seems to indicate that the bacteria that decrease during disease states are those that produce short-chain fatty acids that promote gut health. Assessment of the canine microbiota is currently offered through a commercialized Dysbiosis Index.

Therapeutic gut manipulation:

Most studies on therapeutic gut manipulation are on pre- and probiotics and dietary fibre. The research (human and veterinary-canine) will be reviewed on these studies during the session, along with information on fecal microbial transplants (FMT).

Probiotics:

The proposed role of probiotics in reducing the severity or duration of diarrhea is that they compete with pathogenic bacteria or viruses and provide antagonism of these through antimicrobial metabolites. In dogs, product manufacturers are working to create and market probiotics that meet quality assurance (QA) standards (safe, pure, stable, efficacious) and are beneficial.

Prebiotics:

The most common prebiotics are fructans, followed by mannans, lactosucrose, and lactulose. There is limited research on these products in dogs, with studies limited to nutrient digestibility, microbial concentrations in feces, and fecal protein catabolites.

Fecal microbial transplant (FMT)

In dogs, FMT has been performed for a variety of chronic and acute (e.g., parvo) enteropathies. Research is limited; however, published studies and practical application appears to indicate a benefit. FMT in dogs is commonly administered through colonoscopy or retention enema. Appropriate care and screening of donors (donor stool) is advised.

Resources:

The Gastrointestinal Microbiome: A Review. P.C. Barko, J Vet Intern Med 2018;32:9–25

Clinical effect of probiotics in prevention or treatment of gastrointestinal disease in dogs: A systematic review. Jensen, Bjørnvad. J Vet Intern Med 2019;33:1849–1864.

Texas A& M: Dysbiosis Index, accessed Feb 2021: https://vetmed.tamu.edu/gilab/service/assays/canine-microbiota-dysbiosis-index/

Therapeutic Manipulation of the Gut Microbiome in Veterinary Patients, Stanley L. Marks, Proceedings, WSAVA Microbiome, July 2019

Use of metronidazole in dogs with acute diarrhoea. Holden R, Brennan M BestBETS for Vets, 2020-11-09. Retrieved December 30, 2020, from https://bestbetsforvets.org/bet/574

A randomized double blinded placebo-controlled clinical trial of a probiotic or metronidazole for acute canine diarrhea. Shmalberg JW, Montalbano C, Morelli G, et al Front Vet Sci 2019:163:1-8.

Metronidazole treatment of acute diarrhea in dogs: A randomized double blinded placebo-controlled clinical trial. Langlois DK, Koenigshof AM, Mani R J Vet Intern Med 2020:34:98-104.

Canine parvovirus: Updates on evidence-based spectrum of care therapy considerations

Michelle Evason, BSc DVM, DACVIM
Michelle Evason Veterinary Internal Medicine & Nutrition Consulting
Atlantic Veterinary College; IDEXX UK
mevason@upei.ca

Canine parvovirus is a common GI infectious disease presentation to veterinary practitioners. However, these cases (and pet-owners) often come with unique spectrum of care needs, i.e., evidence-based care option needs along the socio-economic (financial) spectrum.

Evidence-based veterinary medicine (EBVM) can be defined as *the use of the best relevant evidence in conjunction with clinical expertise to make the best possible decision about a veterinary patient*. An evidence-based veterinary medicine (EBVM) approach to patients (and concepts applicable to many other diseases) are critically important, particularly in light of current economic challenges facing pet owners and veterinary care. A recent publication (Stull et al. 2018) sought to raise awareness of the need for this, along with spectrum of care (defined above). Additionally, the commentary proposed initiatives to improve access to care for dog-owners and overcome barriers that prevent veterinary interventions across a wide spectrum of canine health care needs.

This case-based interactive presentation will describe current evidence-based therapies for canine parvovirus and address spectrum of care considerations (and conversations) for the individual dog-owner. Resources on evidence-based (EBVM) practice and PICO question creation are provided below and will be demonstrated during the session.

Parvo virus can cause one of the most common presentations of infectious GI disease in unvaccinated dogs, particularly young dogs. These patients can present with a variety of clinical signs and severity of these, which frequently makes therapy choice and discussion with owners challenging. An EVBM approach can assist with management decision-making and allow for incorporation into a spectrum of care approach with practical cost ranges. This technique is outlined in the session and summarized below.

One of the first steps in the EBVM process is asking a clinical question, which would next lead to the creation of a PICO question. As such, one way to approach a patient with parvovirus might be with the following PICO question: In dogs with parvoviral enteritis infection (P), what is the impact of outpatient treatment (I) as compared with inpatient hospitalisation (C) on patient survival (O)?

The next EBVM steps might include the following assessment of the literature:

Table: Summary of literature review findings, limitations, and strength of evidence.

Title	Source (Date Published	Study Design	Population (Study Participants)	Outcomes	Key Findings-Risks-Prognosis	Limitations
Evaluation of an outpatient protocol in the treatment of canine parvoviral enteritis	JVECC (2017)	Prospective randomised trial	40 client-owned dogs that had not received a parvovirus vaccine, tested positive on in-house faecal ELISA and had not previously received any treatment. Dogs divided into 2 treatment groups: inpatient protocol (IP) and outpatient protocol OP	Survival to discharge, death/euthanasia, or 'failure' of outpatient treatment protocol	16/20 dogs in the OP successfully survived to discharge, 3/20 died, and 1/20 was transitioned to the IP 18/20 dogs on the IP survived to discharge No statistically significant difference in survival or length of hospitalisation between the two protocols All non-survivors were less than 4kg 4/5 non-survivors and the one OP failure were less than 4 months old IP group had significantly lower lactate, and significantly higher increase in potassium during hospitalisation All non-survivors and 1 OP failure were persistentl neutropenic	The small sample size meant this study was underpowered and may not have detected all differences in outcome between the two protocols The OP was a very intense protocol that would require the owners to be able to dedicate significant time to at home care and daily vet visits. The cost of the OP was still high for most financially constrained owners
Evaluation of mortality rate and predictors of outcome in dogs receiving outpatient treatment for parvoviral enteritis	JAVMA (2017)	Retrospective case series	130 client-owned dogs presenting to a first opinion private practice that tested positive for parvovirus on in-house faecal ELISA and were not hospitalised for treatment	Dogs either alive or euthanised at 3 days post diagnosis as determined by phone call with owner.	97/130 were alive at 3 days post diagnosis 33/130 had died or been euthanised within 3 days The majority of dogs were treated with sub-cutaneous fluids and maropitant, with most also given pain relief, antimicrobials or de-wormers Only a prescribed caloric supplement was shown to have a significant effect on survival (p=0.02)	Measured outcomes are fairly limited. Hospital protocol was to follow up at 3 days but for patients where more data were available this may have given more information than just a mortality rate Retrospective study, so no standardisation of treatment protocol Case series rather than controlled trial so no inpatient "control group" to compare mortality rate No indication of what diagnostics were done other than faecal ELIS

Clinical question: In dogs with **parvoviral enteritis infection** (P), what is the impact of outpatient treatment (I) as compared with in-patient hospitalisation (C) on patient survival (O)?

Summary: Two papers were identified that address this PICO. Overall, the evidence available to evaluate the PICO was considered moderate (1 RCT, 1 case series; both with important study limitations).

Based on the above, outpatient treatment of parvovirus does appear to reduce survival when compared to inpatient treatment, but it should (and can) still be considered as an acceptable option for financially constrained clients with a thorough discussion regarding outcomes.

In this spectrum of care scenario, owners should be cautioned that there may be a lower survival (vs. in-patient protocol) and that treatments at home can be intensive. Daily physical examination by a veterinarian and point of care testing should be performed where possible (1), and patients may benefit from a high calorie diet (2).

Outpatient treatment is less likely to be successful (and might not be recommended) if the patient is young (<4 months), small breed (<4kg) or presenting with severe disease signs (1).

Spectrum of care and cost ranges for treatment options: All prices as recommended in the CVMA fee guidelines for PEI.

<u><$100 dollars and no veterinary consultation included:</u>

Risks and benefits to discuss with clients – Unfortunately due to the nature of parvovirus it is unlikely that puppies will survive without significant (OP or IP) supportive care. If finances are limited to this degree and particularly in scenarios where the patient has the risk factors above (i.e. young, small breed, etc.), then euthanasia may be the most humane option.

Euthanasia - $87.50
At home burial – no cost
Total - $87.50

$100-$500:

Risks and benefits to discuss with clients – Even without hospitalisation, at home care for parvovirus is very intensive and will require someone to be at home with the puppy to ensure that temperature and hydration status is well controlled. Survival on the Colorado protocol was 80% (1), but following the complete protocol would be upwards of $500 dollars, so this is not possible. Survival rate may be further reduced using this modified protocol. If the dog is <4kg or <4months or presenting with severe signs, then survival is less likely. In this spectrum setting, an outpatient protocol is less likely to be successful and may not be recommended (1).

Consultation - $71.90
Parvo ELISA - $50
PCV and total protein once - $22.80
Complete blood count once - $67.80
Venous blood gas and electrolytes once- $50
Medications - $150
Food - $50
Home supplies - $20
Total: $482.50

>$500:

Risks and benefits to discuss with clients – While this is the “gold standard”, Parvo is a serious disease and despite following IP treatment protocols, the prognosis may remain guarded and therapy may not be successful.

This IP treatment was taken from in inpatient protocol used in the randomised control trial. It is the protocol used at Colorado State University (a large referral centre and teaching hospital) and will be comparable to the treatment protocols used in many facilities of a similar standard. Survival on this protocol was 90%.

Consultation - $71.90
IV set up and maintenance - $166.70
IV maintenance for 2-3 days - $131.80 - $197.70

Hospitalisation with extensive care for 3-4 days: $229.50 - $306.00
Daily treatment with multiple injections 3-4 days – $170.10 - $226.80
Faecal flotation – $28.50
Parvo ELISA - $50
PCV and total protein 3-4 days - $68.40 – $91.20
Complete blood count 3-4 days - $203.40 – $271.2
Venous blood gas and electrolytes 3-4 days - $150 - $200
Medications - $200
Total - $1470.30 – 1810.00, This fee may be increased dependent on hospital protocols for infection control needs for parvovirus puppies.

Resources:

1. Venn EC, Preisner K, Boscan PL, Twedt DC, Sullivan LA. Evaluation of an outpatient protocol in the treatment of canine parvoviral enteritis. J Vet Emerg Crit Care. 2017 Jan 1;27(1):52–65.
2. Sarpong KJ, Lukowski JM, Knapp CG. Evaluation of mortality rate and predictors of outcome in dogs receiving outpatient treatment for parvoviral enteritis. J Am Vet Med Assoc. 2017 Nov 1;251(9):1035–41.

Other resources:

Infectious Diseases of the Dog and Cat. Weese and Evason. CRC Press. July 2019.

Barriers and next steps to providing a spectrum of effective health care to companion animals. Stull, Jason W., et al. *Journal of the American Veterinary Medical Association* 253.11 (2018): 1386-1389.

Stanton Foundation: Canine Spectrum of Care for General Practitioners Funding Grant. Evason, Stull. February 2019.

How the evidence-based veterinary medicine manifesto for better animal health was developed. Vet Rec 2020:187:e38.

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Risk reduction and management strategies to prevent transmission of infectious disease among dogs at dog shows, sporting events, and other canine group settings. Stull J, Kasten J, Evason M, et al. Journal of the American Veterinary Medical Association: September 2016.

Clinical Nutrition: Alternative diets (raw and homemade): Risks, rewards and pet-owner decision making

Michelle Evason, BSc DVM, DACVIM
Michelle Evason Veterinary Internal Medicine & Nutrition Consulting
Atlantic Veterinary College; IDEXX UK
mevason@upei.ca

Nutrition is a basic component of an animal's survival needs and overall health. However, a disconnect between veterinary recommendations and client pet feeding practices can occur. These nutritional choices can impact both pet and human health through infectious disease risks related to handling and feeding raw meat-based foods. This session will summarize alternative diets and provide a view of pet-owner decision-making surrounding nutrition.

Definition of Alternative diets:

These diets are those that fall into one or more of the following categories: 1) vegetarian or vegan, 2) home-made (cooked or not), and 3) raw diets (may be homemade or commercial). Vegetarian diets typically don't include any types of meat, poultry or fish and may include eggs and dairy products. Vegan diets avoid all animal products, including flavourings, gelatin or supplements derived from animals. Homemade diets can vary widely, as can raw food diets; however, the latter are usually composed of raw meat, and may be partially or completely fed.

How common is alternative diet feeding?

Regional Canadian data[1] compiled on companion animal clients (dog and cat owners) visiting the Atlantic Veterinary College (UPEI, Canada) teaching hospital revealed that:

- ⇒ An unexpectedly high proportion of clients reported feeding homemade (37%) and raw meat-based diets and products (30%).
- ⇒ Of the 30% of clients reporting that they fed raw meat-based diets, about half of them fed raw meat exclusively and the other half reported that they partially fed raw meats.

Results from another recently published observational study of pet feeding practices[2] reported prevalence numbers also. This study demonstrated that, while conventional diets (e.g. kibble based) were the predominant method of pet feeding, more recent data indicates

that unconventional diets (e.g. homemade, raw meat based, vegan, vegetarian) are more prevalent today than ever before.[2] According to the study, 79% dogs and 90 % cats are offered conventional food; however, only 13 % of dogs and 32 % of cats are fed conventional foods exclusively. Approximately 2/3 of dogs and 50% of cats are also regularly offered homemade and raw food. The study stressed that due to the '*risk of nutrient insufficiency and associated conditions that have been attributed to unconventional feeding practices, veterinarians must be aware of pet feeding."*

What are pet owner motivations for alternative diet feeding?

Pet owner motivations for feeding alternative diets typically fall into one (or more) of the following categories:

- ⇒ Desire to feed pet according to personal beliefs, e.g., owner is vegetarian
- ⇒ Distrust of commercial pet foods and pet food industry
- ⇒ Desire to use or avoid certain ingredients, e.g., corn, byproducts, etc.
- ⇒ Belief that cooking (processing) food destroys vital nutrients, enzymes
- ⇒ Desire to feed an 'evolutionary diet', e.g., raw-meat based
- ⇒ Belief that diet is better for specific medical conditions, e.g. cancer, skin conditions
- ⇒ Belief that diet (and treats) provide health benefits over (or superior to) conventional feeding

Results from a recent study (2019)[3] that surveyed dog-owners' motivations, attitudes and practices specific to feeding raw meat-based diets (RMBD) are enlightening. This publication revealed that about 80% of pet-owners had completely abandoned commercial pet food and showed marked distrust to these, especially towards the lack of clarity on the ingredients used. The majority of owners believed RMBDs to be absolutely safe for dogs - and reported that feeding these diets led to a shinier coat, muscle mass gain, and cleaner teeth over feeding their pet conventional diets. Over half of this group of owners also reported that 'controlling the composition and quality of the ingredients provided to their animals was the main advantage of RMBDs'.

What are risks associated with home-made and/or RMBDs?

These concerns range from the physical, (i.e. fractured teeth, mouth or jaw injuries and obstructions (e.g., esophagus, trachea, stomach, small intestine, colon, rectum) and

complications of obstructions (e.g., sepsis, peritonitis, constipation)) to the infectious. The latter include, food safety issues, bacteriologic and/or parasitic contamination of diets, increasingly concerns surrounding MDR pathogens, and human health concerns.[4,6]

How to evaluate home-prepared diets?

Home prepared diets can be a wonderful thing IF prepared properly, consistently and under the direction of a veterinary nutritionist (vet boarded or PhD). For example, homemade diets may be useful for dogs with multiple health concerns as they offer the owner (and veterinarian) more control of the ingredients and greater nutrient customization to meet specific health concerns.

However, nutritional adequacy concerns are a key issue since owners may have incomplete nutritional information or the nutrient profile may not be appropriate for the pet. As well, the client might not follow the recipe instructions, and checking the nutritional adequacy of recipes is not a simple task.

To correctly assess the nutrient profile of a diet requires software, formulation skills, nutritional knowledge, clinical expertise and access to databases of available ingredients. Below are a few quick tips to help veterinarians evaluate a home-prepared diet.[5] The diet should:

- ⇒ Be a clear and specific recipe provided by a nutritionist (vet boarded or PhD)
- ⇒ Include the following:
    - o a protein source
    - o a fat source/essential fatty acid source
    - o a carbohydrate source (including fibers)
    - o a calcium source (e.g. calcium carbonate)
    - o a dog/cat specific vitamin/mineral supplement (i.e. premix)

After assessing the diet for the above, the veterinarian should discuss with the client if a nutrient source is missing and offer nutritionally adequate recipes formulated by a qualified veterinary nutritionist (or referral).

If a client wishes to feed their pet an alternative diet, it's important to offer guidance on complete and balanced diet options from nutritionists with advanced training. Remember that if the owners have questions that you are not comfortable answering, they have unusual

requests (e.g. wanting to feed their pet a vegetarian or vegan diet), or the patient has a complex medical condition, it's always an option to refer the patient to (or consult with) a veterinary boarded or PhD nutritionist for a homemade diet recommendation or diet formulation. I always try to remember that, as a veterinarian, at the end of the day, I am legally accountable for my recommendations, and my number one priority is animal and human health and safety.

Nutritional assessment and communication:

It's so important that veterinarians and their teams speak with every pet owner regarding their pet's diet and complete a nutritional assessment at every visit. Learn what foods your patient is being fed and perform a quick assessment (e.g. WSAVA Diet history questionnaire) in order to determine the nutritional adequacy of the diet. Ask open-ended questions so that the owner tells you everything that their pet eats throughout a typical day (or week), starting first thing in the morning right through to the end of the day. This also includes treats. As a wise nutritionist told me, 'Diet includes everything that goes into the pet's mouth'. At this time, you can also address any concerns with homemade diets or food safety, e.g. raw-meat based diets.

Educating clients about food safety

To keep everyone safe during food preparation for the pet, veterinarians should advise pet owners to follow kitchen hygiene, infection control and food safety practices. This includes common sense things like: washing hands immediately after handling pet food or feces and before preparing/eating food; keeping young children away from pet feeding areas; and storing food in a cool, dry place (away from where human food is stored/prepared).

Raw meat and eggs can be (and frequently are) contaminated with pathogenic bacteria and/or parasites. The following steps should always be taken if feeding a raw diet:

- Wash hands and surfaces thoroughly after handling
- Clean and disinfect all surfaces that the food touched
- Freeze until ready to use
- Keep away from other (human) food
- Don't let pet lick around your mouth and face after eating

- Be especially careful with young children (under 5 years of age) and immunocompromised individuals (e.g. human diabetics, etc.)

Do clients want us to talk about nutrition and exercise for pet health?

Yes. In terms of owner awareness and veterinarian communication about pet nutrition, the Atlantic Veterinary College study[1] revealed that the majority of pet owners are interested in veterinary directed discussions on nutrition and exercise. However, only approximately 1/3 of clients recalled always discussing nutrition with their veterinarian during veterinary visits. The remainder of respondents reported their recall of nutrition discussions with their vet as occurring intermittently.

Resources:

1. Nutrition and Exercise Knowledge, Attitudes and Practices of Clients at a Small Animal Teaching Hospital. Evason, et al. Canadian Veterinary Journal, May 2020.
2. An observational study of pet feeding practices and how these have changed between 2008 and 2018. Dodd, et al. Vet Record, June 2020.
3. Raw meat-based diets (RMBD) for dogs: survey of owners' motivations, attitudes and practices, Morelli et al. March 2019
4. Raw diets for dogs and cats: a review, with particular reference to microbiological hazards. JSAP April 2019.
5. Handling alternative dietary requests from pet owners. Parr JM, Remillard RL. Vet Clinics of North Am: Sm Anim Pract 2014.
6. Infectious Diseases of the Dog and Cat. Weese and Evason. CRC Press. July 2019.

Case Based Approach to Chronic Vomiting in Cats

Debra L. Zoran, DVM, PhD, DACVIM-SAIM
Texas A&M University, College Station, TX

Vomiting is a common and frequently complex problem in cats. Adult cats often have different and more chronic causes of vomiting than kittens, but the condition remains a common reason for cats to be presented to veterinarians for care. Vomiting can be caused by both primary gastrointestinal diseases (e.g. infectious, inflammatory, parasitic, anatomic (obstructive, trichobezoars), drug-related or nutritional) and by extra-gastrointestinal (GI) diseases, such as endocrinopathies (e.g. hyperthyroidism), metabolic disease (e.g. renal failure), inflammatory or other liver diseases, pancreatitis, and neoplasia (especially alimentary lymphoma). This wide spectrum of potential causes of vomiting in cats increases the difficulty for the practitioner in making a definitive diagnosis. Nevertheless, it is important to carefully consider each of the potential differentials to prevent the problem from progressing to create further clinical deterioration.

Primary Gastrointestinal Causes of Vomiting

Food Related Causes: Intolerance, Food sensitivity and Dysbiosis

The use of diet to assist in the management of vomiting is not a new concept. Nevertheless, the type of diet used to help manage the problem has become an increasingly complex issue. In many, if not most cases of uncomplicated vomiting or vomiting due to food type, the best approach is to feed a highly digestible diet or change the diet to one with fewer additives, flavorings, or other substances than may be associated with food intolerance. These types of diets are designed to provide food that is easy to digest (moderate to low fat, moderate protein, moderate carbohydrate), may have additives to improve intestinal health (soluble fibers, omega 3 fatty acids, increased anti-oxidant vitamins, etc), and contain no gluten, lactose, food coloring, preservatives, etc. There are many different brands available that fall under the category “highly digestible”, but, the key is to remember that they are not all alike. Thus, when one diet from this category not accepted by the cat, is ineffective, or seems to make the problem worse, you cannot assume that all diets in this category will be ineffective. The highly digestible diets from different pet food manufacturers have a wide variety of different formulations: different protein and carbohydrate sources, different levels of fat, and a variety of additives designed to promote intestinal health (FOS, MOS, omega 3 fatty acids, antioxidant vitamins, soluble fiber, etc). If one type of highly digestible diet has been fed for at least 2 weeks with minimal response, then is it entirely reasonable to either try another diet from a different source, or try an entirely different dietary strategy (e.g. high protein/low carb, novel antigen, hydrolyzed, etc). Another consideration is that the cat may improve by taking into account the amount or type of food fed. For example, feeding a canned food diet may improve gastric emptying – especially if the vomiting is occurring immediately after eating. Alternatively, if canned food is not an option, feeding smaller meals more frequently, to reduce vomiting that occurs in cats with altered gastric motility or reflux. The key is to remember that dietary management is a trial and error process – there is no single diet that will benefit all cats in all situations.

Food sensitivity and food intolerance are the most common adverse reactions to food in cats. Food allergy or hypersensitivity is an adverse reaction to a food or food additive with a proven immunologic basis. Food intolerance is a non-immunologic, abnormal physiologic response to a food or food additive. Both can be responsible for diarrhea and/or vomiting, but vomiting is a common presenting complaint. Food poisoning, food idiosyncrasy and pharmacologic reactions to foods also come under this category of adverse reactions to food. The specific food allergens that cause problems in cats have been poorly documented, with only 10 studies describing the clinical lesions associated with adverse food reactions. In these reports, the majority of reported cases were attributed to beef, dairy products or fish in cats.

The incidence of food allergy versus food intolerance in cats is unknown. However, in two recent studies of cats with non-specific diarrhea, 2/3rds of the cats improved with dietary therapy, suggesting that a large percentage of cats with diarrhea have some degree of food intolerance. The diagnosis of either food sensitivity or food intolerance is based upon a dietary elimination trial. The major difference between these two types of adverse food reactions is the length of time on the diet that is required to achieve a response (cats with food sensitivity require 6-12 weeks on the elimination diet before an improvement will be seen). Alternatively, in cats with food intolerance, resolution of signs usually occurs within days of the diet change (unless there is concurrent bacterial floral disruption or other factors influencing the response) – but within 10-14 days is a reasonable expectation. There are a variety of commercially available and homemade elimination diets, as well as diets formulated with hydrolyzed proteins, that may be used in cats with suspected food sensitivity or intolerance. The key is to select a diet that has a novel or hydrolyzed protein source (based on a careful dietary history), that is balanced and nutritionally adequate (commercial diets are best for this), however, homemade elimination diets may be needed to find an appropriate test diet. If a homemade diet must be used for long term therapy, a complete and balanced diet containing the necessary protein sources should be formulated by a nutritionist. In most cats with food sensitivity, avoiding the offending food is the most effective therapy and will result in complete resolution of signs. However, short term steroid therapy can be used to decrease the concurrent intestinal inflammation until the appropriate food sources can be identified.

Finally, some cats with vomiting due to food will respond to placing them on a high protein, low carbohydrate diet (canned growth or diabetic formula foods). The reason why kittens or cats respond to these diets is believed to be related to carbohydrate intolerance or to changes in the bacterial flora that result from maldigestion of high starch foods in the presence of abnormal GI wall function (inflammation) or dysbiosis (microbiome disruption) due to diet, antibiotic or other causes. There is increasing anecdotal evidence that in cats with signs of GI disease such as vomiting, feeding a diet containing either highly digestible, high protein and low carbohydrate content is beneficial. Obviously, dietary therapy is not the answer to effective control in all vomiting cats, but in many of these cats, dietary therapy is an important component of therapy that should be carefully considered and implemented and adjusted to meet the needs of the situation.

Inflammatory or Immune-mediated Causes of Vomiting

Inflammatory bowel disease (IBD) in cats is a commonly diagnosed condition of adult cats that represents multiple etiologies. IBD is characterized by persistent clinical signs (vomiting, diarrhea or weight loss) consistent with GI disease that occur in the absence of an identifiable cause but also have histologic evidence of mucosal inflammation and structural changes. There are a number of possible causes of intestinal inflammation that must be considered in the diagnostic process, including infectious, food sensitivity/intolerance, hyperthyroidism, neoplastic (lymphoma) or protozoal and parasitic. These should be investigated thoroughly, or empirical therapy instituted for an appropriate period, prior to settling on the diagnosis of idiopathic IBD. Food sensitivity can be particularly difficult to distinguish from IBD or other intestinal disorders. In a recent study, food sensitivity was reportedly responsible for at least 30% of all feline gastrointestinal problems. Thus, appropriate food trials are an extremely important component of both diagnosis and therapy of cats with GI disease or suspected IBD. In addition to food trials, the diagnostic plan for a cat with chronic vomiting should include: multiple fecal examinations or therapeutic deworming, assessment of thyroid and biochemical/metabolic status, assessment of pancreatic inflammation and function (GI panel/cobalmin/folate status), and potentially infectious diseases (e.g retroviral status, heartworm disease, etc). In addition, radiographs and/or ultrasound are important in assessment for the presence of infiltrative diseases such as FIP granulomas, histoplasmosis or

lymphosarcoma, pancreatic or hepatobiliary disease (triaditis or biaditis), and other proximate causes of vomiting and GI disease. But, ultimately, intestinal biopsies, either obtained endoscopically or at an exploratory surgery are necessary for the diagnosis of inflammatory infiltrates in the bowel wall, and for ruling out other specific causes (especially lymphoma or triad disease) of the clinical signs.

Recent studies indicate a strong association of development of IBD with a breakdown of normal tolerance mechanisms, host susceptibility and the enteric microflora. These same factors are likely very important in feline IBD, and in studies using experimental models of IBD, the resident microflora are essential cofactors in driving the inflammatory response. In addition, studies in cats with IBD assessing modulation of the enteric flora (using probiotics, prebiotics, or other specific therapy for cytokines) are in the early stages of study but are promising. At this time, therapy of IBD in cats continues to include inflammatory suppression, dietary therapy and in some cases, therapy to control nausea, gastritis or pancreatic pain (e.g. maropitant).

Extra-Gastrointestinal Causes of Vomiting

One of the first steps in evaluating a vomiting cat is to attempt to determine as quickly as possible, whether the vomiting is due to a primary gastrointestinal problem (e.g. gastritis, IBD, lymphoma, obstruction, etc), or caused by a disease outside of the gastrointestinal tract (e.g. hepatobiliary disease, renal disease, pancreatitis, endocrinopathies, heartworm, other systemic infectious diseases or cancer, etc). Diseases affecting the feline liver are a common cause of vomiting in cats and should be carefully considered. There are four major types of liver disease in cats: hepatic lipidosis (primary and secondary), cholangitis (acute or chronic forms), infectious hepatitis (e.g. FIP, toxo, histo, etc), and neoplastic liver disease (e.g. lymphoma). The first step is to assess liver enzyme activity – especially since the half life of these enzymes in cats are much shorter, and because cats don't have a steroid isoenzyme for alkaline phosphatase – any elevations of enzymes suggest either leakage (ALT, AST), or intrahepatic cholestasis or bililary tract inflammation (ALP, GGT, total bilirubin increases). In addition to enzyme assessment, liver function should be evaluated, either by function assays (if no changes in bilirubin are observed) or by careful review of albumin, cholesterol, glucose, BUN, and bilirubin as indicators of liver function. In many cases of vomiting, especially those due to primary GI disease, the diagnosis is made by imaging, evaluation of tests of GI function, or biopsy of the GI tract. However, in extra-GI causes of vomiting, however, abdominal ultrasound and or radiographs can be crucial in assessment of the hepatobiliary tree, pancreas, and in assessment of structural anomalies that may cause vomiting (e.g. uretral obstruction, masses of the spleen or liver, and pulmonary infiltrates due to HW disease, etc). And, despite the availability of incredible diagnostic tools, the best way to point the clinician toward the best diagnostic options is to start with the basics and make sure you have obtained a thorough history and physical assessment of the clinical signs.

KITTEN DIARRHEA

Debra L. Zoran, DVM, PhD, DACVIM-SAIM
College of Veterinary Medicine and Biomedical Sciences
Texas A&M University, College Station, TX

Diarrhea, Dysbiosis and Food

The use of diet to assist in the management of GI disease is not a new concept. Nevertheless, the type of diet used to help manage the problem has become an increasingly complex issue. In many, if not most cases of mild or persistent diarrhea in kittens, especially those cases without significant other co-morbidities (vomiting, infectious/parasitic causes, etc) or without significant weight loss, the best approach is to feed a highly digestible diet or change the diet to one without the substances that may be associated with food intolerance or dysbiosis (in kittens, that is most likely carbohydrates or other additives in lower quality foods). A highly digestible diet is not defined in a regulatory sense, but generally indicates a product with protein digestibility of > 87% (typical diets are 78-81%), and the digestibility of fat and CHO should be greater than 90% (typical OTC diets are 77-85% and 69-79%, respectively). Highly digestible diets (e.g. veterinary prescription diets for GI disease as an example) are designed to provide food that is moderate to somewhat lower fat (remember cats won't eat very low fat foods and need more fat than dogs in their natural diet to meet fatty acid/fat soluble vitamin requirements), moderate to increased protein (again remember, unlike diets for GI disease in dogs, cats need higher amounts of protein and to increase digestibility it must be higher quality), and moderate to no carbohydrate – again remember that cats do not need a source of carbohydrate for energy or any nutrient requirement). Commercially available highly digestible diets may have additives to improve intestinal health (soluble fibers, omega 3 fatty acids, increased anti-oxidant vitamins, etc), and most have removed ingredients that may cause issues in some pets: gluten, lactose, food coloring, preservatives, etc. There are many different brands available that fall under the category "highly digestible", but the key is to remember that they are not all alike – either in make up (protein, fat, or CHO percentages or type) or in what is added or removed as part of the formulation. One of the important differences to consider when trying to increase the digestibility and reduce intolerance/dysbiosis for feline diets, is to remember that the protein and fat contents are essential and working to decrease the number and amount of carbohydrates in the food is more important. For example, in most canine highly digestible diets specifically formulated for GI disease, the diets have greatly reduced the fat content because that is the one nutrient source for dogs that can result in dysbiosis, malabsorption and trigger intestinal upset, while in cats, this is not the primary problem, instead, carbohydrates present are often the greatest concern. Thus, diets containing no carbohydrate or only a single source carbohydrate are better for cats generally, and especially if they have disease of a chronic nature. Thus, If you have tried a "traditional" highly digestible diet from this category and it is not accepted or is ineffective, or seems to make the diarrhea worse, you cannot assume that all diets in this category will be ineffective, but instead look to find a highly digestible diet that has very low (or no) carbhohdrate. The highly digestible diets from different pet food manufacturers have a wide variety of different formulations: different protein and carbohydrate sources, different levels of fat, and a variety of additives designed to promote intestinal health (FOS, MOS, omega 3 fatty acids, antioxidant vitamins, soluble fiber, etc). If one type of highly digestible diet has been fed for at least 2 weeks with minimal response, then is it entirely reasonable to either try another diet from a different source, or try an entirely different dietary strategy (e.g. high protein/low carb, novel antigen, home made/whole food, etc).

Another consideration is that the diarrhea may be due to carbohydrate intolerance or bacterial changes resulting from diet changes or antibiotic use – a common problem in kittens from shelters. Recent data has shown that kittens consuming high protein, low carb diets have a completely different intestinal microbiome than kittens consuming diets with higher amounts of carbohydrates (dry foods) and these kittens have a completely different metabolemic profile – meaning that the diet may not only be influencing their microbiome but that flora is also influencing appetite, metabolism and a variety of other health factors. Further, in studies of preference kittens have been shown to actively prefer foods with protein (meat) greater than 50% ME and carbs less than 10%. Finally, there are a number of studies showing that the digestibility of whole foods and raw foods is much greater (based on fecal scores and fecal characteristics including left over nutrients) than dry processed foods – thus, kittens with chronic diarrhea may respond quickly to a homemade diet (high protein, no carb, no additives) when prescription diets have not worked. If you are using this approach, you need to get an appropriate recipe for feeding a complete and balanced diet – this can be achieved by contacting any veterinary nutritionist (a complete listing is on the ACVN website), or through websites like www.Balanceit.com. There is also strong and widely reported evidence that addition of probiotics with or without prebiotics to help influence the microflora are important therapeutic options in addition to finding the appropriate diet choice.

Many kittens that improve on a homemade diet can eventually go back to a commercial food, as long as the offending substance is not present in the diet – this may be carbohydrates, additives or any of a number of other things – so careful, slow reintroduction of foods is needed.

Gastrointestinal disease may decrease the availability of a number of micronutrients, such as vitamins and minerals, with important consequences for the pathogenesis, diagnosis and treatment of gastrointestinal disease. The diagnostic utility of measuring the serum concentrations of cobalamin and folate in cats with suspected intestinal disease has recently been established, and although the impact of deficiencies in cobalamin and folate are not completely known, the role of cobalamin in normal function of the GI tract and in many other aspects of metabolism is well documented. Further, because cats are obligate carnivores that consume much higher amounts of protein in their diet, the importance of cobalamin and other B vitamin in maintenance of protein metabolism cannot be overstated. Thus, evaluation of all cats and kittens with chronic GI disease, not just cats with severe diseases like IBD or lymphoma, is an important part not only of the diagnostic process, but in the management of these diseases as well.

Parasitic and Infectious Causes of Diarrhea

Trichomonosis

Trichomonosis, caused by *Tritrichomonas foetus*, has been recognized as a pathogen in kittens and adult cats. In cats, the organism infects the large intestinal mucosa and causes chronic large bowel diarrhea characterized by increased mucus, tenesmus, hematochezia and increased frequency of defecation. Most affected kittens are healthy, alert and active, and the only outward signs of illness are the presence of anal hyperemia or swelling, and painful defecation, as well as soft stool or diarrhea. The diarrhea can become explosive over time due to development of concurrent dysbiosis. Most infections are diagnosed in young kittens with chronic diarrhea (average age 9 mo), but infection can occur in cats of any age. Kittens that are exposed to the organism are highly likely to become infected, and infection is likely to be persistent. In a recent study, all 8 cats that were exposed to the trophozoites became infected – the organism was cultured from their feces throughout the 200 days of the study. However, infection with *T. foetus* does not necessarily correlate with the degree of clinical signs – especially in adult cats, as there are many cats that culture positive to the organism but are completely asymptomatic. The prevalence of this infection in the general population is unknown, but at a large international cat show, 31% of the cats (36/117 cats) were affected. In studies in Europe, similar percentages of positive cats were identified – whether they were in rescue groups or free living. The infection can be easily misdiagnosed as giardiasis unless the observer is trained to recognize the differences in the two species, and co-infection with *Giardia* spp. has been documented in 12% of cats. Infections are most commonly found in young cats from crowded housing conditions (shelters, catteries, rescue groups or cat "collectors") which may reflect an increased opportunity for exposure or, alternatively, due to environmental stress and immature immune function in young cats.

Diagnosis of this infection can be made by several different approaches. These are listed in order from relative ease of using the test and expense: 1) direct examination of the feces for the trophozoites (diagnostic if found, but often false negatives), 2) fecal cultures for the organism (using the InpouchTF kit, Biomed Diagnostics, White City, OR) (an option but not the easiest or the most definitive approach), 3) PCR of feces submitted to labs able to run fecal PCR testing for trichomonas (the most appropriate). The fecal smear test has very low sensitivity (14%), and errors in diagnosis can be made by inexperienced observers (confuse with giardia or other). However, it is still the easiest and potentially fastest way to make a definitive diagnosis. The InPouchTF kit is more sensitive than the fecal exam, but takes up to 12 days to grow the organism for diagnosis. The samples can be sent to lab for culture, or the culture kits can be obtained and used in the practice/shelter setting. Because other trichomonad species can grow in the pouch, PCR testing of the cultured organisms may be needed for confirmation. PCR testing is the most sensitive and specific method of diagnosis, but is also more expensive and may not be feasible in many shelter settings.

Therapy of cats with trichomonosis is difficult, as the most effective drug for the infection, ronidazole is not approved for use in cats, is difficult to administer –especially due to its powder form and taste, and has significant (although manageable, potential for toxicity). Metronidazole and other antibiotics have been used in both experimentally and naturally infected cats, but are completely ineffective in clearing the infection and metronidazole has been recently shown in studies of the microbiome to cause significant dysbiosis – so is NOT recommended. Ronidazole (powder-on feed antibiotic used in treatment of turkey cankor) at a dose of 30 mg/kg q24h po has been shown to clear the infection in both naturally infected and experimental cases of the disease (the disease is worldwide and responses to treatment in cats in Europe and Asia are similar to the US – 66-80% respond to treatment if given as directed above). This drug is potentially hepatotoxic and neurotoxic, especially when given BID, so once daily dosing is essential and is the only appropriate way to give the drug. This antibiotic should not be used empirically to treat cats with undiagnosed diarrhea. Recent studies using E.

faecium probiotic therapy have shown promise in reducing both diarrhea and remission as this bacterium helps reduce Trichomonas growth and adhesion. All kittens with protoazoal or coccidian parasitism likely benefit from both highly digestible (high protein/low carb canned food) along with probiotics containing this species. All other antibiotics and antiparasiticidals, including tylosin, enrofloxacin, azithromycin and fenbendazole, have not been shown to be effective against *T. foetus*, and in some cats may exacerbate diarrhea by altering the normal flora, or result in delay of clinical remission. Clinical remission of the diarrhea has been shown to occur in many infected cats, usually by 2 years of age, even if they are not treated. However, many cats that have this infection are not acceptable indoor pets due to the malodorous feces they produce. At this time there is no evidence that this organism is zoonotic, but it certainly is infectious to other cats, and thus appropriate infection prevention measures should be instituted.

Giardiasis

Giardia spp. is a frequent cause of diarrhea in cats and kittens, with a prevalence rate reported to be at 4 percent nationally, but the infection rate is much higher in shelters or catteries where it may be nearly 12%. In many adult cats, *Giardia* spp. infections are subclinical or transient, but in kittens, infection is classically associated with an acute onset of malodorous, pale, mucoid diarrhea. The diagnosis is relatively straightforward when the trophozoites or cysts are identified on fresh fecal smears or a flotation. However, because the cysts are shed intermittently, and they can be misidentified or confused with other fecal artifacts, the sensitivity of this approach is only about 50%. The sensitivity increases to 90% if zinc sulfate flotation is used to examine 3 separate fecal samples. Immunoassays and ELISA testing has also greatly improved the sensitivitiy and specificity of diagnosis of Giardia in cats and kittens, but in the shelter setting, these options may not be financially viable for all situations. Treatment of giardiasis in cats and kittens has not changed drastically for many years, and includes specific anti-protozoal therapy combined with environmental control. Metronidazole, at a dose of 25 mg/kg po q12h for 7 days, continues to be a highly effective therapy for the disease in affected cats, but has the potential to cause toxicity side effects and may also induce significant dysbiosis. Fenbendazole is reported to be effective in cats at a dose of 50 mg/kg po q24h. In some cases, combination therapy (metronidazole and fenbendazole) may be needed, but use of highly digestible diet, probiotics (with a product containing E. faecium) and careful environmental control for reinfection are key. Because re-infection is a major cause of persistence or recurrence of infection in a household, cattery, or shelter setting, institution of appropriate environment control measures is essential. These measures include environmental decontamination (cleaning of all floors, cages, litter pans and surfaces that have been in contact with feces with quaternary ammonium or Clorox containing disinfectants), coat cleaning (bathing or shaving the perineum of long haired cats), and isolation of affected animals during the diarrheic phase to prevent infection by co-grooming, etc. Because this organism is zoonotic, appropriate education about handling of infected cats and kittens is an important aspect of the environmental control procedures implemented.

Cryptosporidiosis

Cryptosporidium parvum is a coccidian parasite that infects the microvilli of the intestinal epithelium of kittens and immunosuppressed cats. The presence of the organism around the world is quite variable – with some regions having exceedingly low infection risk (Japan, New Zealand) vs others having more considerable infection risk (Eastern Europe). The organism was found in 8% of cat fecal samples in one study in the US using PCR methods. The disease caused by this infection can range from an asymptomatic carrier, to mild, transient illness, to prolonged, life-threatening malabsorption syndromes. In some adult cats, the organism can cause intestinal infiltrates similar to those observed with classic inflammatory bowel disease, and because the organisms are very small, unless special stains are used, may be not diagnosed. The organisms can be observed in feces at high power, but they are extremely small (< 4 um), and are easily missed unless special stains or assays using immunofluorescence and careful observation are employed. Further complicating the diagnosis, the organisms are shed intermittently, and thus, infected cats can often have negative fecal fluorescence exams. Because cryptosporidiosis is zoonotic, the person handling the needs to use appropriate precautions when handling the infected feces, such as wearing gloves and cleaning any utensils with disinfectants (bleach). Treatment of kittens infected with cryptosporidiosis is very difficult, as infection may be occurring due to concurrent infection with other organisms, due to poor diet or due to intestinal dysbiosis. Tylosin, metronidazole and other commonly used antimicrobials are ineffective in eradicating the organism. They may improve diarrhea if a bacterial component is active, but in most kittens they are not helpful and may increase the risk of dysbiosis. Azithromycin (dose 5-10 mg/kg po q12h) is relatively effective in humans, but has only been studied in a small number of kittens, however, the drug appears to be safe.

Other Infectious Causes of Diarrhea

Other causes of diarrhea that should be considered in kittens include several viral diseases: feline panleukopenia virus, hemorrhagic calcivirus, rotavirus, astrovirus, enteric coronaviruses (including the SARS-Co2) and the coronavirus causing feline infectious peritonitis. Feline panleukopenia is the most clinically

important intestinal virus of this group, and affects primarily unvaccinated kittens and cats causing fever, depression, anorexia, vomiting and diarrhea. Diagnosis is usually based on the clinical signs and history, but the canine parvovirus antigen test cross-reacts with the feline virus and thus can be used for confirmation. Testing for the SARS-Co2 virus is only indicated per CDC and AVMA guidelines if there are people in the household with signs of Covid19 and the cats/kittens are in close proximity with signs of illness. Cats and kittens may be infected with the virus, but their role in disease transmission is still believed to be a minor component of virus spread, compared to the high level of human-to-human transmission that remains the primary mode. Treatment of this and any viral enteritis in kittens is symptomatic and supportive. Because young kittens are prone to dehydration and hypoglycemia, fluid therapy and nutritional support are key aspects of the supportive care. Bacterial causes of diarrhea include salmonellosis, campylobacteriosis, clostridial infections, and occasionally yersiniosis, tyzzer's disease (Bacillus piliformis), and colibacillosis. Diagnosis of bacterial infections causing diarrhea in kittens or cats is very difficult, primarily because pathogens can be isolated in similar rates from both diarrheic and non-diarrheic feces. Other infectious causes of diarrhea include fungal diseases such as histoplasma, however, these are more likely to occur in adult cats. Most of the parasitic causes of diarrhea in kittens are typical, e.g. hookworms, coccidians, roundworms, and strongyloides, and be easily found on fecal flotation, however, therapeutic de-worming should still be performed in kittens that have diarrhea, even if the fecal is negative.

The Feline Pancreas: From Acute to Chronic Pancreatitis and Insufficiency

Debra L. Zoran, DVM, PhD, DACVIM-SAIM
Texas A&M University, College Station, TX

Feline pancreatitis is a disease complicated by its wide range of presentations and its complexity (association with triad disease). Diagnosis of pancreatitis can be relatively straightforward (in acute seriously ill cats) to exceedingly challenging in cats with the waxing waning illness of chronic pancreatitis, and for both conditions the treatment remains symptomatic and supportive. This talk will review the latest literature on the diagnosis of this disease in cats and review the treatment of cats with pancreatitis and one of the effects of chronic pancreatitis: exocrine pancreatic insufficiency.

Diagnosis

Pancreatitis is the inflammation of the exocrine pancreatic tissue secondary to multiple conditions and insults. Some investigators have separated acute pancreatitis (AP) and chronic pancreatitis (CP) forms, based on the severity and longevity of the clinical signs and on histopathologic findings. However, there is no consensus in the literature on whether diagnostic criteria of AP and CP are clinical or histologic. On histology, AP is characterized by neutrophilic inflammation and edema. This condition is different from CP, in which lymphoplasmacytic inflammation and/or fibrosis is a prominent histologic finding with permanent structural and functional impairment. Although AP and CP may have different clinical presentations, these titles may represent the same disease with different presentations occurring along its continuum. The acute disease that is frequently encountered in obese dogs fed a high fat diet, is not similarly reported in cats. Cats with AP are more likely to be underweight, and high fat diets do not appear to be an important predisposing factor. In CP, cats of all ages, sexes and breeds are affected, although Siamese cats are reported to have pancreatitis more frequently. Finally, the clinical signs of CP in cats are especially vague, with the most common signs being lethargy (reported in 100% of cats in one study), anorexia, dehydration and abnormal body temperature (either fever or hypothermia can be observed). This is especially true for cats with chronic disease. Vomiting and anterior abdominal pain, which are common clinical signs in dogs with acute pancreatitis, are reported to occur in only 35% and 25% of cats with CP, respectively, but are common in cats with AP. However, cranial abdominal pain may be more common than is reported, as detection of abdominal pain may be difficult in obese cats or cats with very focal disease. Cats with the most severe acute forms of pancreatitis, may be icteric or in shock, and the prognosis for these cats is significantly more guarded. Other conditions that often occur concurrently with pancreatitis in cats include hepatic lipidosis, cholangiohepatitis, inflammatory bowel disease, interstitial nephritis, diabetes mellitus or vitamin K responsive coagulopathy. In up to a third of cats with chronic pancreatitis, triaditis is present (cholangitis, pancreatitis and IBD), and in up to 60% of cats biaditis (two of the three conditions) is present. Thus, the clinical signs may be quite variable, and this must be taken into consideration with each patient. In addition, with increases in liver enzymes and bilirubin, the signs and abnormalities can easily be attributed to liver dysfunction, which further delays the diagnosis.

Routine evaluation of cats with suspected pancreatitis may include hematology, a serum biochemistry profile, urinalysis, abdominal radiography and/or ultrasound, and serum assays of pancreatic function (e.g. feline trypsin like immunoreactivity –fTLI), or inflammation (feline pancreatic lipase immunoreactivity – fPLI). Hematologic findings in cats with pancreatitis are nonspecific, but may include a nonregenerative anemia, leukocytosis or leukopenia (less common). In a recent study, cats with AP consistently had an elevated WBC (20,300 cell/uL) and mild decreases in platelets (mean = 180,000 platelets/ul). Reported changes in the serum chemistry profile include elevated serum alanine aminotransferase (ALT), elevated serum alkaline phosphatase (ALP), hyperbilirubinemia, hyper- or hypoholesterolemia, hyperglycemia,

azotemia, and hypokalemia. Liver enzyme elevations were more common in cats with mild pancreatitis (determined by surgical biopsy), and GGT ALP, and ALT were all moderately elevated in these cats (supportive of the fact that cholangitis is occurring concurrently). Serum lipase may be increased early in acute pancreatitis, but in a recent study amylase and lipase were found to be of little diagnostic value in distinguishing normal cats from those with pancreatitis. There are no changes in the urinalysis consistently observed or specific for pancreatitis in cats.

There are two available options for testing feline pancreatitis by the fPL – an in house table-top SNAP fPL assay and the lab based Spec fPL assay (considered the current standard). The SNAP fPL has recently been compared with the Spec fPL and shown that it has great correlation with the standard in confirming pancreatic inflammation when it is active (AP). However, the sensitivity of the test, with sensitivities greater than 65%. However, in low grade or CP, the sensitivity can be as low as 35%, and thus a large number of false negatives occur. In cats with chronic pancreatitis, enzyme levels can be quite variable, and thus, while the Spec fPL remains the gold standard for diagnosis, it is still necessary to evaluate the combined historical, physical exam, lab data and imaging information along with the enzyme assay when making a diagnosis.

The feline trypsin like immunoreactivity assay (fTLI) was developed years ago as the test of choice for diagnosis of exocrine pancreatic insufficiency, and the data and follow up have confirmed its utility for this condition. In addition to its importance as a pancreatic function test, in recent years, others have evaluated the fTLI as a diagnostic test for acute pancreatitis – working on the premise than an elevation in serum concentrations were consistent with pancreatic leakage or inflammation. While an increase in fTLI can be found in cats with acute pancreatitis, a normal fTLI does not rule out pancreatitis, and thus this test should not be used for diagnosis of acute or chronic pancreatitis. Nevertheless, the fTLI is still an important test for cats suspected of having CP, as they are at risk of losing exocrine (and endocrine) function – and may develop pancreatic insufficiency which can be detected by this test. Unlike dogs, in cats with EPI the signs are much more subtle, and weight loss is the most common sign (not diarrhea). So, measurement of TLI is important in cats with subtle signs, unexplained weight loss or GI signs not attributable to other causes. In addition to measurement of the TLI, concurrent measurement of cobalamin is also an important test of GI function that should be completed in cats with suspected pancreatic disease, as a majority of cats with insufficient pancreatic function have low (sometimes extremely so) levels of cobalamin – which by itself can lead to inappetence, diarrhea and intestinal dysbiosis/disease. In cats with low cobalamin, this is likely due to the lack of instrinsic factor (from the pancreas) and thus, will require life-long supplementation with cobalamin.

Imaging studies are frequently used to help identify cats with pancreatitis, however, the changes are not consistent and can be particularly subject to interpretation and operator expertise. While ultrasound may reveal a hypoechoic pancreas, hyperechoic mesentery, a mass effect, or may reveal a normal pancreas, these changes do not necessarily confirm pancreatitis as pancreatic cancer can have a similar appearance. Nevertheless, ultrasound can be very helpful in identifying other concurrent abnormalities such as bowel wall thickening, biliary track thickening, sludging or changes consistent with cholangitis, as well as other organ structural abnormalities. Studies have shown that mild (CP) pancreatitis was difficult to visualize or detect via abdominal ultrasound. However, studies also show that ultrasound has a 80% sensitivity and 88% specificity in cats with moderate to severe pancreatitis. However, because cats with CP can have a normal appearing ultrasound, it is important to use multiple diagnostic tools when making the diagnostic plan.

The gold standard for making an accurate diagnosis of pancreatic disease remains confirmation of inflammation on histopathology. This can be obtained through less invasive means (percutaneous pancreatic aspirate/biopsy and endoscopic biopsies) or through the

traditional exploratory. An exploratory laparotomy not only gives the opportunity to obtain biopsies of all tissues of concern: pancreas, liver, sampling of bile and biopsies of intestinal tissues and lymph nodes – all of which may be important in understanding the situation in a cat with triaditis. However, many owners refuse this option as surgery is expensive, can increase the risk of complications (from anesthesia/surgery), and depending on the patient, can result in challenging post-operative management. Thus, while biopsy is an important tool, it is not an option in all cases, and even if the biopsy reveals a normal pancreas – focal or chronic segmental pancreatitis cannot be ruled out.

Therapy of Chronic Pancreatitis

Therapy of chronic pancreatitis remains symptomatic and supportive, primarily because there are no evidence-based studies yet available reporting specific therapeutic approaches that are beneficial, and because cat may have multiple concurrent issues and in the absence of histopathologic or culture driven information it is difficult to make definitive statements of treatment. Because chronic pancreatitis may occur concurrently with either lymphoplasmacytic cholangitis or IBD, therapy for those diseases may be given regardless of what may be used for the pancreatic disease. Many have advocated the intermittent use of steroid therapy (methylprednisolone), and in some cats with chronic pancreatitis this may be reasonable, where LP inflammation is the primary problem causing clinical signs. However, in cats where fibrosis and pancreatic degeneration, not inflammation, is occurring, steroids would be expected to be counterproductive (particularly in causing insulin resistance and increasing the risk of diabetes). At this time, appetite stimulation (using mirtazapine) and pain control (buprenorphine or NK-1 antagonists like maropitant) are the most commonly recommended therapeutic approaches along with fluids as needed. If the cat is nauseous, maropitant may be quite helpful for both its antiemetic effect and pain relief. Finally, antioxidants (SAMe, fish oil) may be considered, but medicating cats with multiple things is often counterproductive, and there is no evidence that these added to the therapy changes outcome. Appropriate dietary and probiotic therapy (as for IBD or diabetes – high protein/low carb) are important – using a highly digestible diet is ideal, long with a diet that is low carb to reduce stress on beta cells, but there is no evidence to that feeding a low fat diet is helpful or indicated. Other immunosuppressive or anti-inflammatory therapy therapy should also be used cautiously and preferably with biopsy confirmation of inflammation. In most cats, symptomatic and supportive therapy can provide relief of signs but will not prevent relapses or recurrences – thus, client communication is essential for preparing them for the waxing/waning chronic nature of this disease.

Nutritional Therapy of Feline Pancreatitis

The diet chosen should be highly digestible and palatable, but the concept of low fat diet to reduce stimulation of pancreatic secretions is not recognized as an important aspect of therapy in cats (as it is in dogs) An important point about feeding cats during this period is to avoid force feeding – not only because it is very difficult to achieve the appropriate level of caloric intake by this method, but also because it can induce food aversion. In many cats with CP, a high protein/low carb diet will be beneficial to reduce the workload on the pancreatic beta cells from a high starch diet, and will also reduce carb malassimilation in dysbiosis and IBD. Alternatively, if the cat is already on a diet appropriate for a cat with IBD (a novel or highly digestible food) and is doing well, a change in diet is not indicated unles the cat is becoming diabetic. Lastly, supplementation of cobalamin, if indicated by measurement of levels, is also extremely important and will be necessary indefinitely.

Constipation in Cats: Prevention and Management Strategies

Debra L. Zoran, DVM, PhD, Diplomate ACVIM-SAIM
Texas A&M University

Colon Physiology and Nutrition Review

While the vast majority of digestion of food sources (protein, fat and carbohydrates) occurs in the small intestine of monogastric species, the colon has its own role in digestion and absorption of nutrients and is a major contributor to the balance of the gastrointestinal ecosystem through fermentation of dietary fibers and metabolism of ingesta incompletely digested in the upper GI tract. However, equally important to health is its role as an essential contributor to the maintenance of water and electrolyte balance (a familiar role of the distal nephron, but equally important here). Most carbohydrates in food are complex carbohydrate (not simple sugars like glucose) that are α-linked (meaning they are able to be acted upon by traditional enzymes that digest carbohydrates (CHO) (disaccharidases, amylases, etc) to break them down into their building blocks for easy digestion and absorption. However, diets also contain in much smaller, but important quantities, β-linked CHO which are found in the woody (e.g. cellulose) and meaty (e.g. guar gum) parts of plants and these make up the group of CHO called dietary fibers. The β-linked CHO present in dietary fiber are only made available when bacteria, with the enzymes necessary to break the β-linkage, digest them. For example, this occurs efficiently in the rumen of cattle or the hind gut of horses, but in dogs and cats, the time and number of bacteria needed to digest these fibers is only present in the colon, and these bacteria are only able to digest (ferment) a small portion of dietary fiber (mostly due to the short time these CHO are there).

Dietary fibers are a complex group of CHO that are generally divided into two separate groups: insoluble (poorly fermentable) and soluble (highly fermentable) fibers. Both soluble and insoluble fibers alter digestion and absorption of other nutrients in the small intestine. Soluble fibers (e.g. beet pulp or psyllium) tend to form gels that slow absorption of dietary nutrients. Alternatively, insoluble fibers increase the rate of passage of nutrients through the intestine and prevent digestive enzymes from having access to the nutrients in the small bowel. This can be helpful in obese animals when you are attempting to create a sense of fullness and reduce intake, but it is not at all a good idea in animals with small intestinal disease as this reduces digestibility of nutrients which can contribute to dysbiosis. In general, the amount or type of fiber that should be present in pet foods is widely debated and highly variable. This is especially true in cat diets, whose natural carnivorous diet would contain small amounts of fiber, typical of that found in the digestive tract of prey. However, commercial therapeutic diets containing fiber have widely varying amounts of insoluble, soluble or combinations of both. Other than its effects on digestion in the small bowel, fiber in the diet is essential for colon health. Soluble fibers (acting as a prebiotic) are primarily present to provide a food source for the colonic bacteria (the short chain fatty acids formed from digestion of soluble fibers are used by both bacteria and colonic cells for energy. Colonocytes are like small intestinal epithelial cells in that they receive the majority of their energy from luminal sources. However, they are different in that they preferentially utilize butyrate (a four carbon, short chain fatty acid produced by bacterial fermentation

of fiber, and other luminal CHO) for their metabolic energy needs. Because soluble fibers are more fermentable (digestible) than insoluble fibers, more butyrate is formed from these dietary fiber sources. Conversely, insoluble fiber (fibers that are not digested in dogs and cats) are essential components of normalizing colonic motility through increasing segmentation, and promoting propulsive (evacuation) activities; however these functions require that the dog or cat be adequately hydrated or the bulky insoluble fiber will increase the risk of constipation from the stool becoming harder and drier (and more difficult to pass).

The beneficial effects of fiber in colitis may be due to normalization of colonic segmental contractions, or its influence on colonic bacterial populations, or its ability to bind toxins or other luminal irritants (e.g. bile acids, fatty acids) and prevent further irritation. However, because constipation is a common problem in obese and older cats, and frequently diets high in insoluble fiber are recommended to increase fecal bulk and improve colonic contractions and motility. This can effective as long as the animal is well hydrated (dehydrated animals will pull even more fluid from the colon, making the feces even drier and harder, a common issue in cats), and as long as adequate colonic contractility remains (e.g. cats with megacolon do not respond to high fiber diets). Thus, no blanket recommendations exist: if clinical response is not achieved with the diet that you believe most appropriate, then an alternate diet approach should be attempted.

Prebiotics, Probiotics, Synbiotics

Prebiotics are dietary substances added to pet foods (soluble fiber sources or substances like FOS), or supplemented as a nutriceutical product to increase the beneficial bacterial flora and thus improve GI health. Criteria have been established for classifying food ingredients as prebiotics and these are: 1) they are resistant to gastric acidity, to hydrolysis by gut enzymes, and to absorption in the GI tract, 2) they must be fermentable by the GI flora, and 3) they are selective for stimulating growth of intestinal bacteria that contribute to overall health. By increasing the number and health of beneficial bacterial species, it is proposed that they will be able to out-compete pathogenic bacterial species, such as *Salmonella, E. coli, Clostridium*, or *Campylobacter* for space, food and other resources. Further, prebiotics also have other positive benefits that are more difficult to measure, including decreasing in fecal protein catabolites and causing positive changes in immune function. Examples of prebiotics that have been studies in cats include fructo-oligosaccharides (FOS), mannano-oligosaccharides (MOS), inulin, chicory, lactosucrose, and oligofructose.

Probiotics are supplements containing single or multiple species of beneficial bacteria aimed at colonizing the enteric ecosystem and helping to normalize the microfloral balance. There are many different probiotics on the market, and it is important to recognize the differences that may be found between OTC and veterinary nutriceuticals (e.g. Purina Fortiflora, Nutrimax Proviable, and many others) in product effectiveness and reliability. In addition, with increasing genomic work being done by the GI lab and others, there is increasing evidence that probiotic species have varying roles and functions in the GI microbiome – thus, the practitioner must familiarize themselves with these products to better understand their use based on field studies and published work.

Synbiotics are products containing both a pre- and a probiotic(s) in a single product formulation. In theory this approach would be ideal for providing the necessary food source for the probiotic bacteria present in the formulation, but the effectiveness of a specific prebiotic with a particular probiotic bacteria has not been extensively studied and dietary sources can be as effective for this purpose.

Colonic Diseases Causing Constipation

Infrequent or difficult evacuation of feces is termed constipation. Obstipation is intractable constipation that is refractory to control or cure and implies some degree of permanent loss of function. The causes of constipation in the cat are quite numerous, but often relate to inadequate intake of water or dehydration due to other causes resulting in dry feces. However, a number of other causes can be involved, including refusal to use the litter box (behavioral, structural), ingestion of feathers or bones resulting in abnormal or painful stool, an obstructive process in the colon (internal causes include masses or neoplasia, and external causes include pelvic fractures), inactivity or obesity – both decrease colonic regularity and function, or ingestion of a high fiber diet (typically dry food) followed by an episode of dehydration leading to development of a hard, dry, large fecal mass. In every case, an attempt should be made to determine the underlying cause. Either constipation or obstipation that are persistent and/or untreated, may culminate in the end-stage syndrome of megacolon, a process where the where the smooth muscles of the colon lose all ability to contract (neurologic dysfunction) and the colonic wall is flaccid and dilated, with loss of functional ability to contract. The presence of hardened feces in the colon, termed colonic impaction, is a consequence of prolonged constipation, obstipation or megacolon, but does not necessarily imply permanent loss of function. Diagnosis of constipation is relatively straightforward, however, functional evaluation of a dilated colon to assess the reversibility of the situation requires anorectal manometry, pelvic floor electromyography, motility assessments and intestinal transit time tests which are not routinely available. As a result, in most cases, the diagnosis is made on response to therapy and as with all organ dysfunction can be present in grades of severity. The key point is to aggressively treat and make every attempt to prevent recurrent constipation as once loss of smooth muscle function and development of neurologic dysfunction is irreversible.

Constipation, Obstipation and Megacolon

An acute episode of constipation is a problem that is usually easily managed in most cats, as long the cause(s) can be identified and corrected (e.g. dietary, life-style, structural issues). However, chronic, recurrent bouts of constipation or development of obstipation are more difficult problems for which the inciting cause must be identified and corrected to the greatest extent possible before treatment will be successful in preventing recurrences. The initial therapy of constipation is aimed at rehydration of the cat followed by gentle softening and removal of feces from the colon and rectum. In dehydrated animals, rehydration therapy is very important (even essential) to help soften the stool. Intravenous fluid therapy is preferred to oral rehydration in most severe cases, but SC fluids can also be helpful. In severely constipated/obstipated cats, general anesthesia followed by multiple enemas will be required to evacuate the

colon. Thus, appropriate preparation for the procedure (pre-anesthetic evaluation and hydration) are essential to preventing complications. The type of enema solution varies, but warm water should be used initially. Stool softeners such as MiraLax are often beneficial and may be added to the enema solution or given orally to help soften the fecal mass. Lactulose is another stool softener that can be used in the same way. Dioctyl sodium sulfosuccinate (DSS) is an emollient that can be added to warm water solutions to help soften the stool; however, it must be remembered that DSS is irritating to the colonic epithelium and in a chronically constipated cat this may be harmful to the colon wall. Enema solutions should be administered by gravity flow depending on the animal's size (in most cats 30-60 ml per dose). Solutions containing soaps or phosphate salts should be avoided due to their irritant or toxic effects – they will be absorbed quickly from the colon. Cats under anesthesia should be entubated to prevent aspiration should the enema procedure result in reflux or regurgitation of fluids in the GI tract anterior to the impaction site as the enema and colonic massage is performed.

Dietary management is an important long term management tool in cats with constipation or obstipation. Two of the MOST important aspects of improving the defecation ability of cats are maintaining hydration (canned food diet) and improving activity (weight loss, pain relief from OA). Many cats have pain or difficulty in passing stool because they have very dry hard feces – a problem created by eating only dry food (very low water content) that contains a very high fiber content (common in weight loss diets or hairball control diets). Fecal water content can increased by simply feeding a higher protein, lower carbohydrate food with added water (canned) – or if the cat will not eat canned food, reduce the fiber content of diet to less than 5% and use SC fluids or the new product Hydracare, to attempt to increase water intake/fecal water content. The standard treatment for many years has been to increasing fecal bulk with dietary fiber of moderate or poor fermentability to help stimulate the defecation reflex and shortens transit time – thus, to help move feces through the colon. However, this method is only helpful as long as the cat is **well hydrated**, and the stool is evacuated regularly (an active cat with normal, regular bowel movements and normal colon function. <u>However, in many cats (who tend to have too dry feces due to marginal dehydration) these diets will likely make the problem worse</u>. In these situations, either the cat must consume more water for the diet to be safe and effective to use, or these diets will increase constipation risk.

The current best approach for any cat that is prone to constipation is to feed a canned food (to increase water content) and add soluble fiber (metamucil or Miralax) to increase water and propulsion as needed. Both Metamucil and Miralax are tasteless, and can be added in very small amounts (1/4 tsp mixed in the canned food) and adjusted as needed to get the stool consistency you desire. The best approach for a cat that only eats dry food is to use a diet that contains mixed fiber – in other words, a small amount of insoluble fiber to "push" and the remainder of the fiber component is soluble (digestible) fiber that will actually "soften" stool and increase stool water content by fermentation. There are several commercially available veterinary prescription diets that have mixed fiber (both insoluble and soluble fiber sources – much like Metamucil – and these are far and away the best diets for most cats that will only eat dry food. However, it is still important to monitor stool firmness and consistency, as any high fiber

diet may make the stool too dry if the cat is not well hydrated or if the cat's colon function is not adequate. In all cats with severe colonic muscle dysfunction or failure (obstipation or megacolon), high fiber diets should be avoided completely – and only diets that are highly digestible (GI diets) that result in the maximal digestion of food and the minimal amount of feces should be used.

The emollient and lubricant laxatives, such as DSS and petroleum jelly, result in a softer stool either via increasing fecal water or by simply lubricating the fecal mass. These laxatives are good for short term management of mild constipation, and in softening a fecal mass that contains hair, bones, etc., but are not effective for therapy of chronic constipation. Osmotic laxatives, such as lactulose or polyethylene glycol-electrolyte (PEG, Miralax) solutions, increase intraluminal water content by their osmotic properties and are the most commonly used and familiar options. The PEG containing product Miralax remains unabsorbed in the feline colon and is very safe and effective in increasing fecal softness/water content. In addition, Miralax is odorless and easier to administer than other laxative products – as it can be added to food (canned or dry, but canned is easiest). Lactulose is fermented by the colonic microflora (like fermentable fiber), which increases fecal water content, so also provides SCFA, however, studies have shown that chronic lactulose use may lead to hypocalcemia and thus this option is not a good long-term therapy option. Lactulose is best used to help soften stool while deobstipation procedures are in process or with enema solutions. Stimulant laxatives, such as bisacodyl, increase the propulsive contractions of the colon and decrease colonic water absorption. They should never be used in a constipated cat, and are only used as a preventative to increase colonic activity – however, they are less effective than cisapride or other prokinetics in that family. Prokinetic therapy with cisapride or prucalamide will increase colonic smooth muscle contractions, and is especially useful with post operative ileus, constipation non-responsive to fiber or laxative supplementation, or in animals with some (minimal) loss of function. In severe cases of obstipation or megacolon that are unresponsive to dietary, Miralax and drug therapy, a surgical subtotal colectomy may be considered. However, there are many issues of long term management and potential complications associated with this procedure that lead to poor quality of life, and this surgery should only be used as a last resort with complete understanding of the owner of the likely outcome.

USDA – Module 5 – Vesicular Disease

Mel Stephens, DVM

(Notes)

USDA – Module 9
Interstate and International Health Certificates for Category 1 Animals

Mel Stephens, DVM

(Notes)

USDA – Module 21
Animals' Fitness To Travel

Mel Stephens, DVM

(Notes)

USDA – Module 29
Veterinary Feed Directives

Mel Stephens, DVM

(Notes)

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Cognition and Optimal Brain Function in Older Dogs

Maureen McMichael, DVM, M.Ed., DACVECC
Auburn University, Auburn, AL

Introduction

The number of geriatric pets has increased considerably in the last 10 years. In 1995 in the United States 24% of pet cats were over 6 years. Today it is estimated to be approximately 47%. In Europe between 1983 and 1995 the number of geriatric cats increased by over 100% while the number of geriatric dogs increased by approximately 50% in that time period. This growing subset of the pet population has received very little scientific research and publications on geriatric critical care are extremely sparse.

In humans, older age alone does not predict mortality. The main determinants of mortality are prior health status and severity of current disease process. Although we do not have studies of this nature in veterinary medicine it seems prudent to offer aggressive treatment to the older dog and cat once comorbid diseases, quality of life for the pet, and the owner's desires are taken into consideration.

This presentation will begin with a brief overview of geriatric pets, then delve into the main non-cognitive comorbid conditions that can mimic cognitive decline in older dogs, specifically dehydration, medications, nutrition, vision and hearing. Cognition, cognitive decline and the progression of it will be discussed next. We will conclude with actionable, practical options for older pets that have significant potential in our patient populations.

Definition of Geriatric in Veterinary Medicine

The term geriatric is difficult to define in veterinary medicine because it differs between dogs and cats and between different breeds (i.e., Great Dane has a much shorter

lifespan than a Chihuahua). In general, animals older than 7 year of age are considered to be geriatric. A more specific definition has recently been proposed. Giant and large breed dogs are senior at 6-8 years and geriatric at 9 years or older. Medium and small breed dogs are senior at 7-10 years of age and geriatric when 11 years or older. Cats can be considered senior at 12-14 years of age and geriatric when 15 years or older.

Fluid Therapy

Dehydration, common in older dogs, can have significant effects on cognition. Significant changes in multiple organ systems in geriatric animals should be taken into account when selecting the type, dosage, and rate of fluid choices in this age group.

Myocardial fibrosis, valvular malfunction, and myocardial fiber atrophy seems to increase with age in geriatric pets. The decrease in ventricular compliance limits the volume that the geriatric animal can tolerate while paradoxically increasing its dependency on volume. Geriatric animals are highly dependent on end-diastolic volume to increase cardiac output and therefore do not tolerate volume depletion very well during times of stress (i.e., illness, anesthesia, etc.).

Renal changes such as the decreased ability to concentrate or dilute urine, decreased renal blood flow, and the limited ability to conserve sodium all limit the geriatric animal's ability to handle either volume depletion or volume overload.

Balanced isotonic crystalloids (i.e., LRS, 0.9% NaCl) are ideal for the dehydrated geriatric patient. Both natural (i.e., fresh frozen plasma) and artificial colloids (i.e., Hetastarch®) are additional options for hypovolemia but should be administered at a slower rate in geriatric animals due to their propensity for volume overload. Supplements such as potassium chloride, vitamin B complex, and dextrose are added as needed.

A thorough search for any underlying or chronic disease processes (i.e., chronic valvular disease, renal failure) is essential when planning fluid therapy. It is imperative that fluid therapy in geriatric animals be monitored both diligently and frequently. Monitoring for optimal perfusion includes frequent checks of pulse quality, extremity temperature, venous lactate, urine output, body weight and mentation. Monitoring for fluid overload includes frequent checks of thoracic auscultation, body weight, urine output, central venous pressure, arterial blood gases or pulse oximetry, and thoracic radiographs.

Pharmacology

Medications, particularly in the very young or very old, can have major effects on cognition since optimal dosing is unknown and overdosing may occur more easily than in younger animals . Aging imposes several changes in the absorption, distribution, metabolism and elimination of many drugs. Oral absorption may be decreased due to decreased GI function as the animal ages. The loss of lean body mass can alter IM route absorption.

If fluid retention is present (such as with congestive heart failure, cirrhosis, or renal failure) drugs that are distributed to extracellular water (e.g., penicillins, NSAIDS, aminoglycosides) will be altered in their distribution. Albumin, the protein to which many drugs bind, also decreases with age.

Drug metabolism may change as the geriatric patient experiences a decline in hepatic function. The mass of the liver decreases with age and decreases hepatic function. This could cause increased plasma half-life of drugs that depend on hepatic excretion, metabolism, or conjugation. Decreased function of phase I metabolism reactions in the

liver appear to occur with age and cause decreases in oxidation, reduction, dealkylation, and hydroxylation reactions. Phase II reactions do not appear to be altered with age.

Drug elimination may be affected by a progressive decline in renal function with age. In geriatric people, there is a steady decline in renal function with approximately 40% of the nephrons becoming sclerotic by the age of 85 and renal blood flow and GFR decreasing by almost half. Due to the loss of lean body mass creatinine may remain normal (decreased production and decreased clearance). In dogs and cats approximately 15-20% are thought to suffer some degree of renal insufficiency as they enter the geriatric years.

In geriatric people, there is a progressive decline in the number of cardiac myocytes and in ventricular compliance. Autonomic tissue is replaced by fat and connective tissue and shows decreased responsiveness to autonomic drugs. It is likely that some decline of cardiac function occurs with age in animals and careful monitoring for specific endpoints is essential when prescribing cardiac drugs to geriatric animals.

Options for appropriate drug dosing in geriatric animals include measurement of renal function, therapeutic drug monitoring with frequent dosage adjustments, and dosage or interval reduction according to creatinine concentrations. The most practical and cost efficient of these options is dosage or interval reduction. Dosage and interval adjustments based on creatinine use the following formulas;

Adjusted dosage = Normal dosage X (Normal serum Cr/Patient's serum Cr) OR

Adjusted interval = Normal interval (1 ÷ {Normal Serum Cr/Patient's Serum Cr})

It is essential to take any co-morbid diseases (e.g., congestive heart failure, chronic renal failure, hepatic fibrosis) into account when considering dosage adjustments

for geriatric small animals. For example, if a dog with chronic renal insufficiency requires therapy with angiotensin-converting enzyme inhibitors the clinician must be aware of the significant likelihood of decreased renal clearance in this animal due to both its chronic renal disease and the older age.

Nutrition in Older Pets

Maintenance energy requirements (MERs) decrease with age in dogs but appear to increase after the age of 12 years in cats. There may also be a decrease in the ability to digest fat and protein as cats age. These changes can lead to either weight gain (i.e., if an older dog is fed food with the same caloric content as it ages) or weight loss (i.e., if an older cat is fed food with the same caloric content as it ages) in the older pet. The reduced ability to digest fats can lead to deficiencies in fat soluble vitamins (e.g., vitamin E) along with water soluble vitamins (e.g., B vitamins) and electrolytes. In older dogs with a limited ability to digest fats due to a diminished ability to secrete pancreatic lipase or bile acids medium chain triglycerides may be beneficial as a concentrated and highly absorbable energy source.

Adequate protein intake is essential for optimal immune function and is critical in geriatric animals. Protein requirements actually increase in older dogs and the old dogma of protein restriction for kidney protection has been discounted.

Antioxidants are essential to combat oxidative stress, which has been shown to increase with age in many species. The “Free Radical/Oxidative Stress Theory of Aging” suggests that levels of reactive oxygen species increase with age and amelioration of this increase can retard the aging process. Anti-oxidants can be administered exogenously and are thought to contribute to decreased levels of oxidative stress and perhaps to increased

quality of aging. Some specific antioxidants that can be easily added to the treatment regimen include Vitamin B complex added to IV fluids (@4 ml per liter when given at a maintenance rate), SAMe given orally (@ 20mg/kg PO q12h), and N-acetylcysteine given intravenously (@ 50mg/kg IV over 1 hour diluted 1:4 with 0.9% NaCl q 8h) or orally (@ 50mg/kg PO q8-12h). Oral N-acetylcysteine can be found in health food stores in the amino acid section.

Anorexia in the older critically ill patient is common and should be aggressively treated after a thorough search for underlying causes. Midazolam (cats), propofol (dogs) and probiotics (e.g., Fortiflora; dogs and cats) may improve appetite. Smell is an important appetite stimulant in both dogs and cats and clogged nasal passages (i.e., bilateral nasal catheters for delivery of oxygen) may cause a decreased appetite. Warming the food and placing a small amount on the tongue may help stimulate eating.

Vision Changes

Vision changes are common in older pets and may mimic cognitive decline. Nuclear sclerosis (lenticular sclerosis) due to increased density of the lens, although common, usually has no effect on vision. However, loss of night vision is common as well as decreased tear production with age. Cataracts are more common with age with a mean onset of ~7-8 years and corneal deposits (e.g., calcium, lipids, cholesterol) also increase with age. Retinal degeneration is also common with age with a decrease in cones and rods seen. With diminishing vision comes reluctance to venture out or the get lost in the year and this may mimic cognitive decline. Adding a vaporizer, especially, during cold weather, may help mitigate the dry eyes that are common in this group.

Hearing Changes

Hearing loss is common in older humans and cochlear degeneration is seen in older dogs. Hearing is extremely important in dogs and loss of hearing is often the straw that broke the camel's back. When hearing loss becomes apparent it usually unmasks vision abnormalities as well since the pet cannot compensate. They may appear to ignore commands, or respond inappropriately when hearing is compromised and this may mimic cognitive decline. N-acetylcysteine is used for hearing loss by the Navy when divers' hearing becomes damaged.

Cognition Dysfunction

Cognitive dysfunction syndrome (CDS) and Disorientation/Dysfunctions in Interaction, Sleep and Housetraining (DISH) are thought to occur in over 50% of dogs over 15 years of age. DISH resembles advanced dementia in humans and our patients may benefit from the new research in people. The recent discovery of the glymphatic (glial + lymphatic) system offers an exciting opportunity to optimize brain health in pets.

Why do we sleep?

Sleep is an extremely vulnerable state that can increase mortality so it must serve an important purpose. Recently, essential brain cleansing, has been documented to occur during certain phases of sleep. It has been shown that during deep sleep the brain "shrinks" up to ~60% in volume to allow for CSF "flushing" and clearance.

Glymphatic System

The glymphatic system uses an aquaporin (AQP4) dependent flow to clear debris (solutes) from the brain. Respiration moves CSF through the aqueduct and this occurs during the deep phase of sleep. Accumulation of these solutes is associated with

neurodegeneration in humans and laboratory animals. Elimination of the waste does not occur in daylight hours (the off switch is thought to be mediated by norepinephrine). Sleep disturbances are associated with dementia in humans and are common in aged animals. There was an 80% decline in glymphatic clearance in aged mice versus young mice and this is thought to be, partially, due to melatonin decreases with age. Sleep disturbances are also very common in hospitalized pets due to noise, stress/pain, loss of circadian rhythm, and lights. Blue light (high in LED and fluorescent lights) depress melatonin synthesis and should not be used after ~12 noon or so. In a traumatic brain injury model, CSF clearance was impaired for 28 days after injury.

Taurine, Not Just for the Heart!

Interestingly, one of the main determinants of success of CSF clearance is taurine, in the form of tauro-cholic acid. This bile acid is essential for the clearance of the solutes via conjugation and the limiting factor is taurine, an amino acid that we usually think of only for the heart.

Essential Strategies for Older Dogs

Vitamin B12, in the form of methylcobalamin or hydroxycobalamin, absorption declines with age in humans and is associated with cognitive decline. Providing subcutaneous injections may help to support cognitive health by providing this essential vitamin. Taurine supplementation, natural anti-inflammatory strategies (e.g., turmeric, low carbohydrate diet, etc.) and melatonin supplementation (e.g., 3-6 mg half an hour before bedtime) can all help to mitigate cognitive decline in older dogs and will be discussed in more detail during the presentation.

REFERENCES are available upon request

Microbiome Update: How it changes with age and how to optimize it for your older pet population

Maureen A. McMichael, DVM, M.Ed., Diplomate ACVECC
College of Veterinary Medicine
Auburn University

All Disease Begins in the Gut
Hippocrates 3rd Century BCE

Death begins in the colon
Elie Mechnikov (1845-1916)

Why the MB Matters

Changes in the MB are associated with cancer (colorectal, gastric, lung, etc.), Neurological (e.g., MS, epilepsy), Behavioral (e.g., anxiety, depression), Endocrine (e.g., diabetes, hepatitis), and Autoimmune conditions among others. In humans, 1 in 12 Americans, 1 in 9 women are affected with autoimmune conditions.

Autoimmune conditions are increasing in prevalence with 23-50 million Americans affected. There are approximately 120-140 different autoimmune conditions, many of these seen in veterinary medicine (ITP, IMHA, IBD, polyarthritis, etc.). What is driving this increase?

Chronic inflammation seems to be a central nidus and the systemic inflammation appears to stem from leaky gut. Leaky gut is driven by pathologic alterations in the microbiome, many of which occur with age.

What is the Microbiome?

Microbes in/on mammals include Bacteria, fungi, archaea, viruses, parasites Each niche has own MB. Skin, mouth, ears, lungs, eyes, etc. Importantly, these are functional! For instances, Atopic dermatitis affects 10-15% canine population. It has been shown that during flare ups there is a decreased diversity in skin bacteria leading to a decreased protective barrier (*Grice; J Invest Derm 2016)*

The Gut Microbiome contains approximately ~100 trillion microbes human gut, which is ~10X more than # of human cells, ~100X more genes than human genome, and ~90% of human illness attributed to MB to some degree This means that ~99% genetic material in humans is bacterial or viral and that we can change our DNA! Rapidly, 3-4 days

Quantifying the MB

Huge difference between SI, colon. Segments near epithelium vs stool, Inside vs outside stool, Inner vs outer end of stool. Clinical improvement matters most!

Formation of Microbiome

At birth neonate inoculated Microbes from vaginal canal
C-section microbes from skin. Very different microbiome with higher rates asthma, autism in humans. Animals?? Human birth centers “inoculate” C-Section baby with mother’s vaginal microbiome. Bulb syringe secretions are removed then the oral cavity is swabbed. During formation there are significant detrimental effects from antibiotics – especially oral! Humans

show higher incidence of asthma when exposed to antibiotics at birth. This critical window is when the MB directs formation of the Immune system and the gut epithelium. Newborns resemble microbiome of mother, siblings until weaning and then MB affected by diet. There is a high variation in MB due to diet, geography, and culture.

Structure of Microbiome

Adult Dogs and Cats have mostly Firmicutes, Bacteroidetes, Proteobacteria Fusobacteria, and Actinobacteria.

Mucous lines intestines, particularly the colon and this mucous layer is very important. It renews every hour and is critical to protect the gut lining. Layers of microbes are in the lumen as well as on the outer and inner mucus layer. The bilayered mucous layer allows the luminal layer to exit with feces. The mucous layer provides lubrication and escorts microbes out. Approximately 50% stool weight = bacteria!

Pathogenic bacteria are sensed by MB, which then increases peristalsis with the goal that the pathogenic bacteria are removed with the mucous. The mucous protects the epithelium of the colon as well. Colonic bacteria eat fiber, if they run out of fiber (low fiber diet) they begin to eat the mucous. When they run out of mucous they eat through the epithelial wall of the colon.

Fiber is essential for bacteria survival and for the formation of short chain fatty acids (SCFAs), which are anti-inflammatory compounds produced by the colonic bacteria. All carbohydrates are absorbed in the small intestine

and high carbohydrate diets starve the colon. Sugar loving bacteria communicate with the brain via the vagus and give the signal to eat more sugar!

Low fiber diets lead to degradation of the mucous (protective) layer by bacteria. After they eat the mucous, bacteria then eat through the epithelium. This is called bacterial translocation and because the entire GI tract, from the esophagus to anus, exists as a single layer of epithelium, extensive damage can occur in a short period of time.

Bacterial Translocation

Bacterial translocation is associate with chronic inflammation due to the inflammatory cascade effects of translocating bacteria. Since the bacteria cannot make SCFAs without enough fiber, there is a loss of that anti-inflammatory effect as well (e.g., SCFAs are anti-inflammatory). Short Chain Fatty Acids are an essential energy source for colonocytes. They also maintain epithelial tight junctions, and produce anti-inflammatory compounds. SCFA administration limits colitis in experimental studies, haven been shown to regulate sodium, water absorption, and increase mineral absorption. The lower pH in gut inhibits pathogens and PPIs are associated with increased pneumonia in humans.

The gut MB also has essential roles for immune regulation. There are tight junctions between epithelial cells that, in health, allow for an anti-inflammatory environment. The GALT, T&B cells, dendrites are all poised to initiate response depending on the microbe sensed. A signal tells a naïve T cell to become Pro-inflammatory or Anti-inflammatory (via T regulatory

cells;Tregs). The immune system also performs surveillance with dendritic cells sitting behind the epithelium and sending dendrites into the lumen to sample bacteria. Dendrites are the only cells that can capture live bacteria to determine the appropriate response (e.g., Pathogenic vs commensal). During pregnancy the dendrites transport beneficial microbes to breast milk.

There is a strong association between mental state and the MB via the Gut brain axis. The vagus nerve affects mentation via direct connections with the MB and the gut neurons are called the 2nd brain. Approximately, 80-90% of serotonin (e.g. the feel good chemical) is made in the gut.
The gut is also home to much vitamin production including cobalamin (B12), biotin, thiamine, and others.

Dysbiosis

What used to be called SIBO is now called dysbiosis and is thought to be due to loss of commensals, excessive growth of harmful organisms and reduction in overall diversity. Bacteria can induce chronic inflammation (from too little fiber) and pathogenic bacteria set off chronic inflammatory cascade that can induce cancer. Beneficial microbes are associated with decreased cancer, mainly due to decreased inflammation.

There are significant links between dysbiosis and autoimmune conditions in humans, such as RA, Asthma, Atopy, ITP, IMHA. Similar links occur with neurodevelopmental and neurodegenerative conditions such as Alzheimer's, and Parkinson's. It is unknown whether such links occur with cognitive dysfunction in animals.

There is also a strong correlation with dysbiosis and obesity, which is more prevalent as pets age. In mice, a MB transplant from obese mice to lean mice resulted in the lean mice becoming obese. The opposite occurred when lean mice MB was transplanted to obese mice. This suggests that the MB may be contributing the obesity in the aged pet. Proposed mechanisms include an increased dietary energy harvest as well as microbe induced changes in host glucose & lipid metabolism. Obesity is also associated with chronic low grade inflammation which can lead to insulin resistance via chronic LPS exposure. Probiotics (bifidobacteria) have been shown to lower LPS, improve glucose tolerance and reduce inflammation.

Dysbiosis in Older Pets

The top three causes of dysbiosis in dogs and cats are antibiotics, Proton Pump Inhibitors (& H2 blockers) and diet. Antibiotics are common and approximately 4/5 humans in US take at least one course of antibiotic every year. A single course of ciprofloxacin was shown to alter the MB for >1 year The number of days of antibiotic use is associated with increased risk breast cancer fatality (JAMA 2004, Lancet 2012).

Long term (over 7 days) use of Proton Pump Inhibitors (PPIs) is associated with Osteoporosis, C. Difficile, Pneumonia, B12 deficiency, AKI, Dementia, V fibrillation – magnesium deficiency in humans.

Diet, particularly a high carbohydrate, low fiber diet, is very detrimental to the MB. There are specific concerns as well to select ingredients such as glutenin and gliadin, the 2 main proteins associated with gluten. Gliaden triggers zonulin production, which results in break down of the gut tight

junctions resulting in leaky gut and chronic inflammation. Research at Harvard (Dr. Alessio Fasano) has shown that many autoimmune conditions show high levels of zonulin. When mice are exposed to zonulin they make get a leaky gut and make antibodies to beta cells.

Prebiotic fiber, non-digestible polysaccharide and oligosaccharide are fermented by colonic bacteria, generating SCFAs. Resulting in lower inflammation, more anti-inflammatory mediators, and a lower pH. Prebiotics (Bifidobacterium, Lactobaccili) protect the gut epithelium, increase the mucous layer, elongate the microvilli, and prevent adherence of pathogenic organisms. Prebiotic fiber acts like fertilizer, for every 100g consumed, 30 grams of bacteria produced. Food sources of prebiotic include Inulin – in chicory, garlic, onion, leaks, jicama, chicory, Jerusalem artichoke, raw dandelion greens, raw asparagus, green banana and potato (cook, then refrigerate, then warm to increase prebiotic fiber). In dogs, chicory root supplementation resulted in improved fecal scores in healthy dogs.

Probiotics have been documented since 1909, when they improved clinical signs in autoimmune arthritis. Supplementation with live cultures (Streptococcus lacticus, Bacillus bulgaricus) were used. Live organisms confer the best benefit, maintain tight junctions, up regulate tight junction proteins and increase mucin secretion by globlet cells. Probiotics increase defensins, prevent pathogen colonization, produce SCFAs, stimulate IgA secretion and decrease luminal pH. Even non-viable organisms may confer health benefits as they adhere to the mucous layer and stimulate immune function. In a study in shelter dogs a probiotic (*E.faecium* SF68; Fortiflora®) with metronidazole iImproved fecal scores versus metronidazole alone.

Foods rich in probiotics include all properly fermented foods such as kimchi, Sauerkraut, kefir, lassi, kombucha tea, tempeh, and pickles.

Other options for favorably altering the MB include giving a probiotic enema, which dates back to ancient Egypt and the Mayans. When making these it is important not to use chlorinated water and approximately 3-6 probiotic capsules are used. Ideally, probiotics with a high percent of Bifidobacteria, which are predominant in the colon, would be used.

Fecal microbial transplant (FMT)

Other options include FMT, which has been approved in US for recurrent *C.difficile*. There is >90% cure rate for this deadly condition after FMT. In Europe FMT is used for autoimmune conditions and there are multiple options for delivery including oral capsule, nasogastric tube, nasoduodenal, colonoscopic, or enema. Reports of clinical improvement with IBD, MS, myoclonus dystonia, Refractory ulcerative colitis, Autism, Parkinson's and Rheumatoid arthritis.

One of the most dramatic studies was in 17 children with Autism Spectrum Disorder. Researchers wiped out the endogenous flora with Vancomycin and then administered FMT. They reported a 80% decrease GI signs, 24% decrease core ASD symptoms, and even greater improvement at 2 years!

In dogs with eosinophilic IBD there were improvements after FMT with the dogs being symptom free for 3 months. Another example of 8 dogs with refractory C. perfringens that were given FMT showed the diarrhea resolving in all dogs afterward.

Conclusion

The microbiome is now known to be an essential player in health and disease. Microbes, chiefly from the gastrointestinal tract, are critical players in immune function, vitamin production, utilization of nutrients, detoxification, inflammatory and autoimmune disorders, and neurotransmission among others. In 2008 the NIH launched the human microbiome project and a plethora of new research is highlighting the importance of the maintenance of balance in the 100 trillion microbes residing on and in the mammalian body. Cutting edge treatment options include probiotic enemas, fecal capsules and fecal microbial transplants (FMT) and these are being used for infectious diarrhea (C difficile), autoimmune conditions (rheumatoid arthritis, multiple sclerosis, Crohn's disease) and obesity. The Harvard Medical School teamed up with MIT to create the OpenBiome Project to make FMT more widely available.

Hypercoagulable Changes in Older Pets
Mitigation Strategies
Maureen A. McMichael, DVM, M.Ed., DACVECC
College of Veterinary Medicine
Auburn University
Auburn, AL, USA

Introduction

The coagulation system becomes more prone to clotting (prothrombotic) with age in humans and, likely, pets. A combination of toxin exposure (e.g., pollution, pesticides, chlorinated water, etc.), stress, poor diet, and oxidative stress contribute. This talk with start with a brief review of physiologic hemostasis, including an update on the new models of coagulation. We will discuss the aging changes that contribute to prothrombosis, detrimental effects (including hidden and micro-thrombosis) and will conclude with practical strategies to mitigate prothrombotic states in our pet patients.

Endothelial Review

Our research into the endothelium has evolved from the belief that it was an inert tube that carried blood all the way to today where it is a living, breathing (internal mitochondrial respiration), organ that is extremely heterogeneous, highly intelligent (endothelial cells sense what other endothelial cells upstream or downstream are reacting to), and absolutely essential for the health of every cell in the body.

The endothelium in health is a powerful antithrombotic surface. First, the glycocalyx, a hairy like covering of the endothelium, has a negative charge that repels cells towards the center preventing platelets from rolling on the endothelium. There is also constitutive expression of numerous substances including thrombomodulin, endothelial protein C receptor, heparan sulfate proteoglycan, tissue factor pathway inhibitor, nitric oxide, prostacyclin and ADPases (enzymes that break down ADP). These are all antithrombotic. Lastly, the endogenous prothrombotic substances (e.g., collagen, von Willebrand Factor, tissue factor) are sequestered from the flowing blood being contained in the subendothelium (collagen, vWF) and adventita

(tissue factor).

Normal hemostasis is local – very important. We never want systemic clotting! During the process of physiologic hemostasis these three protections are removed – locally. For instance, when a needle penetrates the endothelium (e.g., phlebotomy) multiple mechanisms remove/inactivate thrombin diffusing away from a local clot. A breach in the endothelium does several things; it removes the negative charge, removes the anti-thrombotic substances, and exposes pro-thrombotic substances that are normally sequestered from flowing blood. This allows the creation of a local fibrin clot.

Healthy Endothelium

Prostacyclin & Prostaglandin

Prostacyclin (predominantly large vessels), and PGE_2 (predominantly small vessels) are constituitively expressed on the endothelium and are potent vasodilators. Importantly, these anti-thrombotics are inhibited via blocking of the COX enzyme system (e.g., NSAIDS), which is one way NSAIDS can lead to thrombosis.

Ectonucleotidases

Ectonucleotidases break down ADP (ADP; adenosine diphosphate) after it is released from platelets after platelet activation. ADP that diffuses away can lead to systemic platelet activation and these scavenge any ADP that diffuses away from a local clot.

HSPGs and HCII

Heparan sulfate proteoglycans repel platelets and are an integral part of the glycocalyx

HSPGs are markers of GLX degradation

Tissue Factor Pathway Inhibitor

Very important inhibitor of coagulation

Inhibits initial steps in coagulation

TFPI-F10a complex inhibits TF-7a

Antithrombin

Must bind to cofactor, Pentasaccharide sequence, On ~30% pharmaceutical grade heparin And on endothelial bound HSPG, AT inhibitory activity enhances >1000 fold By binding to heparin

Protein C pathway

Thrombomodulin expressed on endothelium and any that diffuses away from local clot becomes bound in TM. Complex of TM-Thrombin activates protein C and becomes an antithrombotic powerhouse. Activated protein C (APC) inactivates F5&8
Endothelial protein C receptor (EPCR) acts similar to APC but in different vessels.

Nitric Oxide Gas Diffusion

Because NO is a gas it has the ability to diffuse through the endothelial wall to the lumen, where it inhibits platelet reactivity and to the abluminal side, where it relaxes vascular smooth muscle and inhibits cell proliferation. This gas is crucial for blood vessel health! The precursor to NO is arginine and cats have a specific need for arginine in their diet. A study found that cats with thromboembolism were low in arginine compared with normal cats. (JVIM McMichael 2001).

Thrombosis

Thrombosis is defined as an unwanted clot (e.g., no breach in endothelium) that may be obstructive. Thromboembolism occurs after either dislodging of a local clot or the occurrence of systemic clotting (DIC – disseminated intravascular coagulation).

Hemostasis vs Thrombosis

The Inter-relationship between immunity and coagulation can be traced back to the beginning of evolution where one cell, the hemocyte, controlled immunity and hemostasis (invertebrates). This basic survival strategy walls off damaged, infected tissues in an attempt to limit infections. The coagulation system inhibits pathogens and the immune system sets off

clotting. Platelets wall off pathogens, neutrophils extrude DNA in nets to limit pathogens and platelets extrude NETS too! Polyphosphate (PolyP) in bacteria and viruses sets off the contact pathway to trigger systemic clotting.

Thrombosis and Immunity

There is a clear crosstalk between the coagulation and the immune system and there is a significant morbidity from thrombosis with infection. Newer treatments address both

Virchow's Triad

The components of clot formation occur due to changes in blood flow (stasis), blood constituents (retention of procoagulant factors), and the endothelial wall (stretching or damage).

Blood flow changes that are associated with prothrombotic states in veterinary patients include stasis in the Left Atrium (cats with HCM) which often leads to arterial thromboembolism (e.g., saddle thrombus). Blood changes that can lead to prothrombotic state include increased viscosity (e.g., Polycythemia, Hypergammaglobulinemia).

Alterations in Endothelium

Damage leading to down regulation and/or elimination of antithrombotics along with up regulation or exposure of prothrombotic substances. Inflammation, hyperglycemia, oxidative stress, LA stretching, physical damage (e.g., Tumor), Infections (e.g., ticks, bartonella) and Dilation (e.g., DCM, enlarged LA) all contribute.

Antithrombin

Inhibits F10a, F9a, F11a, thrombin (2a), only free thrombin, not bound in clot
Inhibits leukocyte activation –rolling and adhesion. Blocks expression of proinflammatory cytokines. Exogenous heparin eliminates anti-inflammatory effects of AT.

Once thrombin is made it sets off coagulation & inflammation. This system is dysregulated in sepsis.

Tissue Factor

TF is a key initiator of hemostasis in vivo. It is likened to a hemostatic envelope surrounding blood vessels & surfaces – fibroblasts, pericytes, keratinocytes. It is bound to F7a/F7 in dermal vasculature. High TF lungs, brain (astrocytes), pancreas, heart, uterus/placenta/testes
Influenza A increases TF expression in mouse lungs. Should we consider anti-thrombotics with pneumonia? TF is not thought to flow freely in blood because it is highly prothrombotic. Studies of stagnant blood – no free TF but Studies of flowing blood – free TF found
Flow may "activate" TF and this is an active area of research. TF in blood likely inhibited or encrypted and needs activation or key. Blood-borne TF may contribute to thrombosis not hemostasis.

What Do Platelets Do? Maintain tight junctions, especially post-capillary venules. When platelet numbers go down capillary endothelial junctions open.

Anti-Platelet Medications to be discussed include aspirin and clopidogrel.
Thromboxane A2 - aspirin
P2Y12 receptor - clopidogrel

Hemolytic Uremic Syndrome (Alabama Rot)
What Does von Willebrand Factor Do? Protection of Factor VIII
ADAMTS13 & Thrombotic Thrombocytopenia will be discussed in the context of the recent cases in the UK.
JA Kremer Hovinga, JN George. N Engl J Med 2019;381:1653-1662.

Conditions Associated with Thrombosis

IMHA – platelet reactivity, TF expression, MPs, inflammation, free heme scavenges NO

Cardiac – stasis, turbulence, endothelial injury, inflammation

PLN, PLE – loss of AT, endogenous anticoagulants, inflammation

Neoplasia – platelet reactivity, TF expression, inflammation, release TF + MV into circulation

Pancreatitis – inflammation, TF expression

Trauma – inflammation, TF expression

Thrombosis and Disease

Sepsis –activated monocytes major source of TF, Activates coagulation during endotoxemia

Surgery –triggers inflammatory response, increase monocyte TF

Pretreatment of monocytes with ubiquinol (reduced CoQ10) leads to decreases of TF expression and decreases oxidative stress

DIC - Definition

Disseminated intravascular coagulation

From a strong activator of coagulation. Could be PolyP, or Blood Borne TF. DIC is characterized by presence of widespread thrombi with deposition in the microvasculature. This occurs concurrently with a bleeding tendency.

DIC - Sequelae

Thromboses impede perfusion, may lead to multi-organ dysfunction syndrome (MODS).

Consumption of clotting factors & platelets leads to bleeding after the hypercoagulable phase.

DIC independent, powerful predictor of mortality but is always a complication of another disease.

DIC – Common Causes

Sepsis and systemic infections

Widespread inflammation

Cancer

Severe trauma – exposure of TF to multiple sites

Summary – Thrombosis associated with…

Inflammation, cytokines, increased platelet reactivity

Increased TF expression monocytes & ECs

Alterations in blood flow (turbulence)

Vessel wall changes

Endothelial damage, ROS, inflammation, hyperglycemia

Coagulation changes in blood leading to Increased prothrombotic substances, decreased antithrombotic substances.

Mitigation

Options for mitigation will be discussed in order of;

Lease likely to harm, low cost, may work

To More likely to harm, high cost, may work

Options to be discussed include arginine, methylcobalamin, N-acetylcysteine, dietary changes, microbiome enhancements, low dose aspirin, clopidogrel, factor X inhibitors, and others.

References available upon request

Oxidative Stress and Select Antioxidants in Aging Pets

Maureen McMichael, DVM, M.Ed., DACVECC
Auburn University

Objectives

This talk will focus on what you can do for your aging small animal patients to optimize health using anti-oxidants. Specifically, ubiquinol (heart failure, neuropathy, muscular degeneration, aging), N-acetylcysteine and SAMe (liver disease, detoxification, lung disease), curcumin (ingredient in turmeric; neurological diseases, aging, inflammation, brain health), alpha-lipoic acid (general health, cognitive decline), and DMSA (much less smell than DMSO; joint disease, arthritis) will be discussed and dosages given.

Measurement of Oxidative Stress is Problematic

Oxidative stress (OS) is a continually evolving state that is associated with numerous diseases and conditions in humans and animals. It is defined as reactive species (RS) in excess of antioxidant defense mechanisms. Antioxidants (AO) are defined as substances that can delay or prevent oxidation of a target molecule. OS can occur due to an excess of RS, a reduction in AO or both. Physiological levels of RS interact with the redox state and play essential roles in cell signaling and may be necessary to induce adaptive responses through antioxidant defense. Pathological levels of RS result in oxidative damage and activate cell death pathways. Elucidating the specific damage caused by RS and measuring the effect of treatment with exogenous substances is challenging. There is a significant drawback to *in vitro* testing in that cell culture is exposed to significantly more oxygen (environmentally) than most cells *in vivo. In vivo* testing has also been associated with significant issues including a lack of sensitive & specific, non-invasive, standardized tests to evaluate damage done by RS. Because of this lack of standardization, the clinical pharmacology of AO has not been effectively studied. In addition, much of the research on OS involves methodologies that are either not directly applicable or are not practical in clinical situations. Newer diagnostics that are more sensitive & specific are promising. Treatment of OS has not been rewarding and it is thought to be due to most studies using a single antioxidant. Newer treatment modalities include targeting the mitochondrial (MT) AO and using multiple AO that are synergistic. This review will cover new findings in OS in relation to specific disease states, the mitochondrial theory of aging, and normal antioxidant defense mechanisms.

Normal anti-oxidant defense mechanisms

In health, the major source of RS formation in cells is electron leakage from electron transport chains with ~90-95% of the oxygen converted to water and the remaining 5-10% is reduced, creating RS. The generation of RS is kept to a minimum by the high efficiency of electron transfer and sequestration of metal ions. Separate microenvironments exist for the MT, the lysosome, and the peroxisome; each contains a RS-generating system coupled to immediately adjacent antioxidant defense mechanisms. Three extra-MT sources of RS are the xanthine oxidase system, NADPH oxidase, and uncoupled nitric oxide. Once formed, RS can either react with another radical to form a covalent bond or, more commonly, react with a non-radical. When a free radical reacts with a non-radical, the non-radical loses an electron, transforming into a free radical. This is the essence of the chain reaction that propagates extensive damage to cell membranes. When the

radical combines with another radical the product can be more damaging than the original radical. An example is when nitric oxide (NO) combines with superoxide ($O^{\bullet -}$) creating peroxynitrite ($OONO^-$), which is 2,000 times more damaging than hydrogen peroxide (H_20_2). Alternatively, the reaction of two radicals can result in a termination of the cascade. The interaction of RS with lipids in the presence of free iron results in lipid peroxidation. Production of RS is balanced with endogenous AO defenses with the AOs controlling *levels* of RS, not eliminating them. RS appear to play essential roles in vivo including redox regulation of gene expression. Cells exposed to RS may undergo proliferation, senescence, apoptosis, or necrosis. The level of RS that causes cells to change from proliferation to any of theses appears to be cell type specific.

Types of Antioxidant Defense

In general, there are three lines of AO defense against damage caused by RS. AO proteins, such as albumin, haptoglobin, ferritin, and ceruloplasmin are abundant in plasma. Intracellular enzymatic AO include superoxide dismutase (SOD), catalase, and glutathione peroxidase. These are expressed in most mammalian cells and prevent the generation of RS. Small molecule AO are divided into water-soluble and lipid-soluble categories. Water-soluble AO include ascorbic acid (vitamin C), uric acid, bilirubin, glutathione (GSH), zinc and selenium. Lipid soluble AO include tocopherols (vitamin E), β-carotene, ubiquinol (co-enzyme Q; CoQ), and lycopene. Cell membranes contain tocopherols and β-carotene within their lipid layer and these can act to quench chain reactions of lipid peroxidation. Extracellular fluids contain molecules with AO properties (ascorbic acid, bilirubin, transferrin, haptoglobin, albumin, urate).

Glutathione peroxidase, synthesized in mammalian cells, is generally considered the first line of defense against RS formation. It is a sulfur-containing tripeptide (glycine, cysteine, glutamine) that reduces H_20_2 to water, using GSH as a substrate. OS has been shown to be associated with a depletion of GSH and this has been shown to induce apoptosis of hepatocytes. Vitamin E, the 2nd line of defense, inhabits the lipophilic interior of the cell membrane, where the PUFAs are located, and is a chain-breaking scavenger, halting lipid peroxidation. When a wave of lipid peroxidation reaches vitamin E it is oxidized to a free radical, sparing any adjacent PUFAs from oxidation. Vitamin C then combines with the E radical forming a poorly reactive, water-soluble, vitamin C radical, and regenerating vitamin E. Vitamin C is the most abundant water-soluble antioxidant and it can directly scavenge RS or regenerate vitamin E. Superoxide dismutase (SOD) is an oxido-reductase that contains copper, zinc or manganese at the active site. It catalyzes the dismutation of superoxide to oxygen and H_20_2. It is present in the cytosol (requires copper and zinc), the mitochondria (requires manganese), and on the extracellular surface (requires copper and zinc). Mitochondrial SOD is believed to play a major role in AO defense mechanisms. Catalase is a heme protein located in peroxisomes, which converts H_20_2 to water and oxygen. Catalase functions in conjunction with SOD; SOD converts superoxide to H_20_2 and catalase then converts the H_20_2 to water and oxygen

Mitochondrial Theory of Aging

The mitochondria (MT) play a central role in the generation of RS and OS has been shown to damage mitochondrial DNA (mtDNA). This may lead to lower numbers of mitochondria per cell with age or more dysfunctional MT. MT, the chief source of ATP, are considered the energy centers of the cell. MT damage is thought to contribute to the negative effects of

aging. Numerous experimental studies correlate increased MT AO with prolonged lifespan. In mice overexpression of MT catalase extended lifespan, a MT targeted AO (SkQ1) prolongs lifespan, and a mutation associated with decreased MT RS generation increased lifespan. A premature aging phenotype mouse model correlated aging with mtDNA deletions and MT respiratory chain failure occurred with high loads of mtDNA deletions. Age related conditions such as muscle and hearing loss have been associated with increased levels of MT OS. Mice with increased MT RS had accelerated hearing loss while mice with increased Mt AO had improved hearing compared with aged controls.

Oxidative Stress and the CNS

The CNS, due to its high oxygen demand, high level of polyunsaturated fatty acids, high levels of iron, and low level of endogenous AO, is quite vulnerable to OS. Increased MT OS is well documented in numerous CNS conditions including Alzheimer's, Parkinson's and Huntington diseases as well as multiple sclerosis. The amyloid plaques seen in Alzheimer's inhibit MT function by inhibition of the electron transport chain which leads to increased RS production. Overexpression of catalase in mice MT decreased amyloid toxicity and extended lifespan in a mouse model of Alzheimer's. There is substantial evidence for a central role of MT in the pathogenesis of Parkinson's, again with overexpression of MT catalase showing a protective role.

Oxidative Stress and Coagulation

There is strong evidence that OS leads to a hypercoagulable state in people and research animals. Nitric oxide (NO) is rapidly inactivated by RS, specifically superoxide anion. NO is essential for the maintenance of vascular flow (prevents vasoconstriction) and also prevents platelet aggregation. Preservation of NO may be one method that AO use to modulate coagulation and AO status is an important determinant of platelet function. Addition of specific AO leads to increased bleeding time in many studies. Alpha tocopherol is a platelet inhibitor and decreased platelet AO content in people is associated with platelet hyperactivity.

Oxidative Stress and Neoplasia

Mice lacking or low in specific AO systems show increased rate of hepatic carcinoma, lymphoma, adenocarcinoma, and pituitary adenomas later in life. Some malignant cells use RS to increase metastasis, angiogenesis, and proliferation and to suppress apoptosis. One mechanism that RS may use to promote angiogenesis is by increasing cell production of VEGF. RS have been shown to modulate integrin expression and suppress anoikis. Metastasis may be enhanced by RS via changes in intercellular communication, cell mobility, and increased vascular permeability. Matrix metalloproteinases (MMPs), which are involved in degradation of the extracellular matrix, can be activated by RS. Some RS (e.g., H202) have been shown to stimulate the proliferation and migration of human prostate cancer cells. In mice, non-metastatic cancer cells became invasive after depletion of AO and the effect was eliminated after instilling an AO rich preparation. Interestingly, RS appear to have a bi-phasic effect in cancer with significant excesses of OS impairing angiogenesis. The loss of MT cytochrome oxidase is associated with colonic dysplasia and MT catalase expression reduces tumor burden in mice with mammary carcinoma and hematopoietic tumors. Reducing MT RS in mice reduced their propensity to develop thymic lymphoma.

Quantifying Oxidative Stress

A common research method for studying OS is cell culture but this has significant drawbacks including the lack (or severe deficiency) of many AO (vitamins E, C, selenium) and the addition of free iron. Perhaps most importantly is the fact that most cells in culture are exposed to 95% air and 5% C02, which is equivalent to approximately 152 mmHg of oxygen, an extremely hyperoxic environment, compared to in vivo (in which most cells are exposed to less than 10 mmHg oxygen). There is likely increased RS formation due to the hyperoxia alone. Oxidation in cell culture can lead to false results in some studies. The excess exposure to RS in cell culture has been discussed as one reason why cellular proliferation is so common in laboratory settings.

In vivo testing of OS is problematic, time consuming, and less convenient than cell culture but has some inherent advantages. The most widely accepted in vivo method of testing for OS is the measurement of isoprostanes. When RS attack arachidonic acids on cell membranes isoprostanes are formed. They are produced *in vivo* independently of the cyclooxygenase enzyme by free radical-catalyzed peroxidation of arachidonic acid. The isoprostanes most commonly studied are the αF_2-isoprostanes. Much evidence now exists indicating that the F_2- isoprostanes are a reliable, non-invasive way to measure lipid peroxidation *in vivo* compared with other methods. Administration of AO has been shown to inhibit the formation of F_2-isoprostanes in both animal models of oxidant injury and in humans. Isoprostanes can be detected in all types of biological fluids and tissues; the free form can be measured in urine and plasma, esterified complexes can be measured in tissue, or metabolites can be measured in urine. Auto-oxidation can occur in lipid-containing samples during processing and storage which is why many researchers prefer urine. CNS specific isoprostanes appear to form with damage and some appear to be effective markers of damage. F2-dihomo-isoprostanes are derived from damage to myelin and may be a selective marker of white matter injury in vivo. Urine isoprostanes were 6 fold higher in humans with MS compared with controls and CSF levels of isoprostanes were significantly higher in another study. Urinary isoprostanes were higher in ALS patients compared with healthy adults.

Measurement of Endogenous Antioxidants

Measurement of specific AO in blood or tissues can be used as an indicator of OS if the levels of AO are low. Glutathione peroxidase is an essential part of the endogenous AO defense system. Glutathione, the substrate, exists in 2 forms; reduced glutathione (GSH) and the oxidized form, glutathione disulfide (GSSG). During OS, GSH is oxidized to GSSG. Measurement of the ratio of GSH to GSSG can be used to assess oxidative damage via depletion of GSH and has been reported in dogs and cats. This method is also susceptible to spontaneous oxidation *ex-vivo* and artificially elevated GSSG levels. In sled dogs α-tocopherol was shown to decrease significantly after an exercise run, suggesting that the endogenous AO capacity may not be adequate for the challenges of vigorous racing. In another study aimed at decreasing OS in racing sled dogs, Baskin, et al., reported that supplementation with α-tocopherol, β-carotene, and lutein increased plasma concentrations of these AO significantly in Alaskan sled dogs.

Treatment of oxidative stress

Most treatment of OS involves blocking the formation of RS, scavenging RS after they are formed or augmenting AO. Alpha lipoic acid is protective for diabetic neuropathy. Coenzyme Q (CoQ) reduced diastolic dysfunction in children

with cardiomyopathy. The reduced form of CoQ10 is called ubiquinol and is associated with greater OS reduction. Resveratrol prevented LV hypertrophy, diastolic dysfunction, and interstitial fibrosis in a mouse model of the metabolic syndrome. Quercetin reduced systolic BP and oxidized LDL in overweight humans and improved cardiac function in rats. It is likely that the best treatments will encompass a combination of drugs that target several steps in the OS injury cascade.

Blocking Formation of RS

Glutathione can act both as a chain breaking antioxidant, inhibiting lipid peroxidation, and as a metal chelator, preventing formation of the hydroxyl radical. It is synthesized in all mammalian cells with the rate of synthesis dependent upon cysteine stores in most organs except the liver. In the liver, GSH can be synthesized from either cysteine or methionine and the liver is the primary site for GSH synthesis, supplying up to 90% of circulating GSH. When GSH is given exogenously it cannot penetrate cell membranes. Cysteine is the rate-limiting amino acid in the formation of GSH and treatment with N-acetylcysteine (NAC) enables continued production of GSH. NAC is also a powerful scavenger of both the hydroxyl radical and hypochlorous acid. The protective effects of NAC are believed to be associated with the sulfhydryl groups trapping electrophilic intermediates by acting as a nucleophile. Treatment with NAC is protective against endotoxin challenge, radiation induced injury, and lung injury from toxic gas. In a rat model of IR injury, NAC blocked NFkB activity in addition to scavenging RS. It has attenuated IR injury during cardiac catheterization and has shown cardioprotective effects during ischemia. NAC has also shown some benefit in both sepsis and ARDS patients.

Vitamin E, composed of 4 tocopherols and 4 tocotrienols, is a lipid-soluble vitamin that antagonizes the peroxidative injury of membrane lipids and inhibits propagation of cell membrane destruction. It converts the alkylperoxyl radicals to hydroperoxides and then to tocopheroxyl radicals. The tocopheroxyl radicals are then reduced by vitamin C. Vitamin E, C and ubiquinol destroy RS involved in the "chain reaction" of lipid peroxidation**.** Vitamin C (ascorbic acid) is a water-soluble vitamin that allows regeneration of vitamin E for continued antioxidant effects. Ascorbic acid reduces the tocopheroxyl radical back to the antioxidant tocopherol. Vitamin C reduces ferric iron to ferrous iron, which under normal conditions improves absorption of iron from the GI tract. Under conditions of ischemia or increased availability of free iron, vitamin C can function as a pro-oxidant by providing more ferrous iron for the generation of hydroxyl radical (via the Haber-Weiss reaction). Several clinical trials reported disappointing results with supplementation of vitamin E. Unfortunately, most of these studies used either the synthetic form (no antioxidant properties) or only $1/8^{th}$ of the natural form. In patients with coronary artery disease, endothelial dysfunction was attenuated by administration of vitamin C and this appears to be due to superoxide scavenging by vitamin C. However, it appears that vitamin C must be given in very high concentrations to compete effectively with NO for superoxide. Ubiquinol (CoQ) appears to act as an antioxidant but the exact mechanisms are not clear. It appears to prevent both the initiation and propagation of lipid peroxidation.

Scavenging RS

SOD exists on the extracellular surface, in cytosol and MT. It scavenges superoxide anion and converts it to H_20_2. If there is not sufficient catalase available to convert the H_20_2 to water then H_20_2 will accumulate and contribute to the formation of the hydroxyl radical. In this case, SOD can be considered a pro-oxidant. Exogenous SOD has been shown to be protective in many models of IR injury. Its short half-life may be a factor in the studies that showed no

improvement. In renal transplants, it has been shown to decrease acute rejection and improve 4 year graft survival. Catalase converts H_20_2 to water and oxygen. It is essential that catalase be present along with SOD to convert the H_20_2 produced by SOD to water and oxygen. The paired administration of SOD and catalase conjugate has been shown to be effective in attenuating OS in several models.

Since free iron is central to the formation of the hydroxyl radical many treatment strategies attempt to block iron. However, iron is essential to many biological processes and iron chelation therapy can have potentially toxic side effects when they interfere with normal iron metabolism. Most strong chelating agents remove ferric iron from proteins (i.e. transferrin) and can interfere with iron incorporation into hemoglobin. Deferoxamine chelates ferrous iron and has been shown to reduce RS injury in several models. Several studies evaluating deferoxamine have been unrewarding most likely due to the toxic side effects and its short half-life in circulation in humans (~5minutes). DMSO scavenges the hydroxyl radical and the metabolite that is formed traps other RS. It permeates cell membranes to get to intracellular sites of RS formation and is also thought to inhibit platelet aggregation and increase vasodilation. It can lead to the formation of the methyl radical, which can then react with PUFA's to form methane gas or can react with oxygen to form methyl peroxyradicals. It is believed that the levels of DMSO needed to scavenge the hydroxyl radical may be high enough to cause damage to healthy cells. During the breakdown of ATP during ischemia, there is a buildup of adenosine. Adenosine is also released by neutrophils, endothelial cells, and myocytes. Interestingly, adenosine, in high concentrations, is believed to be responsible for the benefit seen with ischemic pre-conditioning. In addition to stimulating A1, A3, and potassium ATP channels, adenosine may inhibit conversion of XD to XO during ischemic periods. If this is true, then the accumulation of adenosine during ischemia would result in decreased RS formation. Adenosine is believed to decrease release of superoxide radical by neutrophils, to decrease leukocyte adhesion, and to increase the synthesis of NO via A2 receptor binding. Adenosine can cause hypotension and AV block when given IV.

Simultaneously administering several AO that work synergistically has shown promising results. A human product, Protandim®, combines 5 active ingredients: Silybum marianum (milk thistle), Bacopa monnieri, Withania somnifera (ashwaganda), Camellia sinensis (green tea), and Curcuma longa (turmeric extract). A new veterinary product, Canine Health®, by the makers of Protandim, adds joint protection and comes in a chewable tablet. This combination has been shown to work synergistically to increase SOD and catalase and to induce heme oxygenase-1 (an enzyme that breaks down heme) in humans.

Curcumin, the active ingredient in turmeric, is a potent anti-inflammatory agent with over 200 published studies and will be discussed in more detail in the talk.

DMSA has potent anti-inflammatory properties and is also inexpensive and less offensive (to the olfactory system) than DMSO.

References available upon request

Update on Anti-Aging Research
Applications for the Geriatric Pet

Maureen A. McMichael, DVM, M.Ed., DACVECC
College of Veterinary Medicine
Auburn University
Auburn, AL

Introduction

There has been a plethora of anti-aging research in the last few years, some of which is applicable to our veterinary patients. The main benefits of these strategies are their ability to halt the negative effects associated with aging such as arthritis, cognitive decline, frailty, sensory loss (e.g., hearing, vision loss), and energy level. The area that has received the most attention is caloric restriction and this area has very promising results.

Review of Essential Repair Mechanisms

Autophagy

This is the body's ability to clear damaged organelles and to clean up damaged DNA by clearing excised genomic fragments. When an organelle is targeted for recycling (e.g., mitochondria) the organelle is isolated within a vesicle called an autophagosome which subsequently fuses with a lysosome. The new, autolysosome, is then degraded and the organelle parts are recycled. This essential mechanism allows the body to maintain healthy cells. Autophagy is inhibited in early stage cancer, allowing tumor formation and growth. Autophagy occurs during periods of low to no energy input (e.g., fasting, low protein ingestion).

Sirtuin Signaling

Sirtuins (SIRTs) are a family of NAD+ dependent deacylases that prevent disease and may reverse some aspects of aging. Sirtuins delay cellular senescence and extend lifespan through age related telomere attrition and promoting DNA damage repair. Sirtuins regulate DNA repair, fat differentiation, glucose output, insulin sensitivity, fatty acid oxidation, inflammation and aging. There are different SIRTs in the cytoplasm, mitochondria, nucleus of each cell. There is an

association between loss of SIRTs in cancer cells and accumulation of mutations and genomic instability.

mTOR

The mammalian (or mechanistic) target of rapamycin (mTOR) are kinases that play important roles in autophagy, protein synthesis, mitochondrial biogenesis, cell growth & proliferation, cell survival & motility, and transcription. Decreased mTOR is associated with increased life span in mice and worms. A simplified description is that when mTOR is turned on mammals are in growth mode (e.g., cells divide, all energy goes towards growing) and when mTOR is turned off we are in preservation and clean up mode (e.g., autophagy). Amino acids, particularly methionine and leucine, are potent activators of mTOR and this "abundant energy" signal tells the body to grow. When mTOR is off (no energy or amino acids coming in) it tells the body to switch to quiescent mode. In quiescent mode the body switches to autophagy and the clean up begins.

Caloric restriction

Calorie restriction acts, in part, by inhibition of mTOR. When no protein is coming in, mTOR shuts off, and autophagy (clean up, recycle) begins. This allows removal of damaged organelles, cleaning up debris, and removal of damaged DNA. Calorie restriction (fasting) reverses chemotherapy induced DNA damage. Cycles of fasting delay progression of melanoma, glioma, breast cancer and improve effectiveness of chemotherapy.

In 2006 researchers published findings from a study in dogs. There were 48 Labrador Retriever puppies from 7 litters that were split into pairs (matched for sex and weight). All were fed the exact same diet but the calorie restricted group were fed 25% less than the control group starting at 8 weeks of age. The calorie restricted group lived 1.8 years longer, and, importantly had a significant delay in age related diseases such as osteoarthritis. Newer research into this study suggests that signals from the microbiome of the calorie restricted group may be involved in the longevity and health of the dogs.

https://pubmed.ncbi.nlm.nih.gov/18062831/

Studies in humans are a bit more difficult and the CALERIE study attempted to mimic the dog study. The humans, however only cut down their intake by 11.9% in the calorie restriction group. Despite this, after 2 years there was a decrease in cholesterol, systolic and diastolic blood pressure, insulin sensitivity, metabolic syndrome score and C-reactive protein.

Promising Therapies

There is so much new research on anti-aging. Some are not practical in pets (e.g., significant decrease in all cause mortality from sauna use) while others are (e.g., cold therapy –jumping into a cold lake or ocean has been shown to improve immune function. Here we will discuss some of the more promising ones.

Diet

First, caloric restriction has the most solid research behind it. Cutting a dog's calories by slowly decreasing the amount fed over time to ~20% less calories is likely to be very helpful over the course of the dog's life.

As we discussed in the microbiome talk, the gut is the workhorse of the body, and likely where health or disease begins. Inflammation that begins in the gut spreads to all areas of the body wreaking havoc, pain, and dysfunction. There is a delicate balance between health producing microbes and disease producing microbes that is easily disrupted leading to disease. All of these conditions are significantly worsened as dogs age and many of them can be addressed with simple and safe strategies.

Essentials of a healthy microbiome start with high quality fiber. The best sources of this good fiber are often lacking in many K9 diets. These include asparagus, Jerusalem artichoke, dandelion greens, banana, leeks and garlic and onions. These last two (garlic and onions) can cause problems in dogs if ingested in large amounts so it is best to limit these to small amounts

or skip them. Intestinal gas (flatulence) can occur if the fiber is increased too quickly. It is best to go slow and build up.

There are changes to the microbes in the gut as canines age. Depending on the state of health there can be loss of balance with a higher percentage of more harmful microbes that can induce inflammation. This inflammation is not limited to the gut but affects the entire body. That means the joints, the brain, the heart, these all feel the effects of inflammation. Fermented foods such as pickled veggies, miso, sauerkraut, and kombucha are very helpful in replacing some of the good microbes that may have been lost due to antibiotic administration, illness, or even from chlorinated water. Chlorinated water, which we all drink, is one reason to continue to supplement the diet with fermented food. Many dogs love fermented veggies and these make a wonderful snack.

The absorption of fats, fat soluble vitamins, and some B vitamins becomes harder and harder as dogs get older**.** Supplementation with select vitamins that are very difficult for the gut to absorb in older dogs (e.g., B12) should be considered. Additionally, small amounts of healthy fats (e.g., Medium chain triglyceride oil, coconut oil, grass fed pastured butter, omega 3 fatty acid supplements) may be helpful. Vitamin B12 is essential for gut health and, at the same time, are harder for the gut to absorb with age. Absorption of B12 is quite complicated as first it has to be present in the food, then get bound up to intrinsic factor (a carrier) in the stomach, then travel all the way down the intestinal tract and then get absorbed much lower down in the intestines by specialized receptors. As dog's age, all of the systems that help absorb B12 can become less efficient, making it harder and harder to absorb. Not having enough B12 can lead to decreased absorption of nutrients from food, low RBC counts (and low energy), difficulty fighting off infections (e.g., immune function compromise), mental decline (e.g., forgetting or not responding to commands, acting confused), decreased energy, balance problems (seem wobbly or unstable), difficulty sleeping at night, and many other issues. In order to get around the difficulty of absorbing this orally in older dogs many veterinarians prescribe injections of

B12. The Texas A&M GI Lab has published dosages for dogs on their website. The website can be found here. https://vetmed.tamu.edu/gilab/research/cobalamin-information/#dosing

Gastric Acid

The gastric acid and prostaglandins in the stomach decrease with age. The combination of these make digesting food take longer and will increase the chance of ulcers in the stomach if the dog is on any anti-inflammatory medications (e.g., NSAIDs, Rimadyl®, Ibuprofen, etc.). Alternative options for inflammation include turmeric, ginger, Epsom salt soaks, cold laser therapy, and acupuncture.

In older humans (and likely dogs and cats) the taste buds decrease. It is likely that this contributes to a diminished appetite. The combination of nasal changes (decrease in olfactory receptors, drying out of nasal mucous) and decreased taste buds likely contributes to appetite changes. A simple vaporizer may help improve both taste and smell functions a bit.

There is decreased movement of food through the GI Tract due to lower gastric acid, lower numbers of neurons and decreased blood flow to the gut. This slows the movement of the intestines so that it takes food longer to move through the GI tract. Allowing older dogs more time to digest (e.g., before vigorous exercise) may be helpful.

Constipation if common. Constipation should be considered a serious condition as it is related to cognitive decline in humans (and likely dogs). There is a clear association between constipation and onset of Parkinson's in humans. Interestingly, the only substance that is associated with absence of Parkinson's is coffee consumption, which has a beneficial effect on constipation. Decreased movement of the GI tract leads to constipation, which is quite common in older dogs. Two issues that can worsen constipation are dehydration and lack of fiber. Older dogs have less efficient kidneys (they cannot optimally dilute or concentrate urine) and are predisposed to dehydration. In addition, the thirst response is diminished in older dogs so they will often become dehydrated *before* they become thirsty and have a hard time catching up. As

dogs age, it is essential that they have access to water more frequently to prevent dehydration. Healthy fiber options include raw carrots, raw beets, sweet red peppers, pumpkin, squash, sugar snap peas, green beans, cauliflower, broccoli. Trying multiple fresh veggies is worth it as many dogs will happily chew on a sweet red pepper or a sugar snap pea but refuse other options.

Gastrointestinal Bleeding is Common. Anemia is very common in older humans and leads to lots of complications and poor outcomes after illness or injury. It is likely common in our geriatric patients as well. It is not completely clear why older humans are anemic but it is likely from loss of blood in the gut. Chronic inflammation of the gut from lack of high quality nutrition, significant lack of healthy fiber, and ingestion of toxins in the foods (e.g., pesticides) contribute to the chronic inflammation. Optimizing the diet with high quality fiber (e.g., fresh veggies), fermented foods, organic bone broth (source of collagen), and high quality fat in small quantities to start (e.g., coconut oil, grass fed butter, cod liver oil, etc.) can lead to gut healing.

Immune Function

There is decreased immune function with age. The largest part of the immune system is housed in the gut. As dogs age, there is a decrease in immune function that can lead to a greater number of infections. The immune system is also less responsive with age so it is slower to respond to an infectious challenge. Keeping the immune system in top shape can significantly increase the healthspan (healthy lifespan) in dogs. Keeping the gut healthy is essential in building healthy immunity. Gut healing options include anti-inflammatories (e.g., turmeric, ginger), supplementation of gut healing vitamins (e.g., B12), and replacement of healthy collagen to heal the gut lining (e.g., organic bone broth).

Energy Requirements

In dogs a 20% decrease in energy requirements was documented to occur after the age of 7 years. It is likely that in pet dogs the decreased activity (e.g., partially from arthritis) contributes to lack of exercise and this contributes to decreased food requirements. Aging is associated

with decreased muscle mass (sarcopenia). The loss in muscle leads to a host of problems including instability in the joints (progressing to arthritis), weakness, and lack of desire for exercise. The vicious cycle then leads to more muscle loss (from lack of activity). High quality protein is essential for muscle strength.

Melatonin

Melatonin, a hormone that is essential for sleep, is produced in the gut and the brain (80% is made in the gut). There is a documented decrease in melatonin with age in people and it is likely that at least some of this comes from a dysfunctional gut. Keeping the gut healthy may help keep melatonin levels normal but supplementing with melatonin (3-6 mg/dog given 30 minutes before bed) can help older dogs sleep. Since the detrimental solutes in the brain are cleared by the glymphatic system during the deep phase of sleep, melatonin may help prevent cognitive decline.

Promising Strategies to be discussed

Fasting Mimicking Diet

This diet has been shown to mimic a fast due to the very low protein nature of the diet. There is significant research on this diet in humans with cancer and the diet will be discussed in the context of preventing aging.

Curcumin

There are over 200 studies documenting the powerful anti-inflammatory effects of curcumin, the active ingredient in turmeric. It is best consumed with a little bit of fat and black pepper. We will discuss the options for curcumin in aging pets.

Ubiquinol (COQ10)

Ubiquinol, the reduced form of CoQ10, is essential during several points in the electron transport chain in the mitochondria. Ubiquinol decreases with age and there is a large body of research on supplementation for cardiac health.

Nicotinamide riboside (NR) and nicotinamide mononucleotide (NMN)

These two precursors to NAD+ are very important for functioning of the cell, particularly the mitochondria. Research on supplementation will be discussed along with options for treatment.

Crocin

This is the active ingredient in Saffron, the deep yellow spice. Current research suggests it is anti-inflammatory, anti-cancer, anti-anxiety, and anti-diabetic. It has been shown to inhibit reactive oxygen species (e.g., free radicals) and advanced glycation end products (AGEs).

And more….

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Where Do I Start With This Pruritic Patient?

Millie Rosales, DVM, DACVD
Email: miamivetderm@gmail.com

It has been reported that approximately 75% of the cases you will see in general practice have a dermatological condition. If you google "top medical conditions in pets", skin and ear problems are at the top of the list. Therefore, it makes sense to have a good understanding of dermatology and know how to work up the most common patient you will see: the pruritic pet.

When a pet is itchy it can have an effect on the bond between the pet and its owner. There is frustration on the client's side when they see their pet scratching, chewing and excoriating themselves. Since many pruritic pets have odorous, lesioned skin, it prevents the owner from wanting to touch their pet, further affecting the human animal bond. Therefore, the goal in working up the pruritic patient is not only to find the right medications to provide the pet relief, but also to find the underlying cause of the skin condition so the pruritus doesn't become chronic and continue to erode the human animal bond.

What is this pet's story? History taking

It would be easy if a pet could talk and tell us what ails them. Since they don't, we rely on getting this information from the pet owner. The history is a crucial part of identifying what is making the pet itchy. This takes practice, and it takes time. Dermatology cases are not 15-minute appointments. Many of these cases require much more time on initial presentation and this should be scheduled appropriately into your day.

The key information to obtain from the client includes: signalment (age, breed, sex), a complete medical history of past and present problems, and a dermatological history. For efficiency purposes, a questionnaire with all the pertinent medical and dermatological questions can be given to the client to complete. Some important data to obtain are the original presentation and progression of the skin problem, the degree and distribution of pruritus, presence of seasonality or not, previous treatments and the pet's response to those treatments, and if other pets are affected. Be a detective and gather as much information as possible.

Dermatological exam

Examination of the skin, coat, nails, and mucocutaneous areas should be checked in all pets that present with pruritus. Don't just focus on the particular itchy area; be sure to perform a thorough exam of the entire body. On exam, look for primary and secondary lesions and note the distribution and pattern to the lesions, check for the presence of ectoparasites, and examine the oral cavity. An otoscopic exam of the ears is important, even if the pet does not appear to have an otitis problem. Many conditions that affect the skin will also affect the ears.

Differential diagnosis/Client education

With the history and examination findings, make a list of potential causes for the pet's pruritus. This differential list should be explained to the pet owner. This will help the client to understand why certain diagnostics are necessary. Good client education on the different causes is an important part of the consultation, and it is vital in creating that bond between the practitioner and client.

Dermatology diagnostics

The minimum procedures for most dermatological cases are skin scrape, skin cytology, ear cytology (if otitis is present), and a dermatophyte culture. These tests can provide a wealth of information and should be done routinely for all skin patients. These are inexpensive, simple diagnostic tools that can be performed in-house. A Wood's lamp is another good diagnostic tool when used appropriately and is discussed below. If the pet's skin condition is more complicated, then skin and ear cultures and skin biopsies may be necessary.

The one diagnostic that should not be ignored in any pruritic (and even non pruritic) patient is the skin scrape. A deep skin scrape (must get capillary bleeding) to look for demodicosis is essential. If the case looks suspicious for *Sarcoptes* (i.e., lesions on the pinna, elbow, hock lesions, and extreme pruritus), then superficial skin scrapes may be necessary.

Skin cytologies are important to discern if there is secondary bacterial and *Malassezia* infections, which are often significant contributors to pruritus. There are many different methodologies of performing skin cytologies: direct smears, impression smears, swab smears, acetate tape strips, and scrapings. Become familiar with one of these and perform them routinely on your dermatological cases.

If the pruritic patient also has otitis, then ear cytology is essential to assess if bacteria and/or yeast organisms are causing the infection. This is discussed further in the otitis lecture.

Quite often a staphylococcal infection is mistaken for dermatophytes. Cytology of the lesions and a fungal culture will help rule in or rule out dermatophytosis as a cause. For those who have a Wood's lamp in their clinic, this is best used as screening tool for dermatophytosis, not for diagnosis. Not all strains of *Microsporum canis* (the most common dermatophyte of concern in pets) will fluoresce. Certain bacteria, greasy scales or sebum, and even topical medications can fluoresce on Wood's lamp, potentially giving a false positive if a culture is not performed. Dr. Moriello, one of the leading veterinary dermatologists in dermatophyte research, recommends the following steps when using a Wood's lamp: 1) use a magnifying electric lamp for more reliable results because the handheld, battery operated lamps have a chance for false negatives; 2) darken the room and let the observer's eyes adapt to the light for 2-3 min; 3) the lamp needs to be held close to the skin (approximately 4-10 cm); and 4) observe for apple-green or blue-green hair shaft fluorescence. If fluorescent hairs are present, then these should be sampled for culture.

For dermatophyte cultures, culture plates are recommended over vials. The plates are easier to inoculate with the hair samples and easier to sample fungal colonies for microscopic analysis. Dermatophyte test medium (DTM) contains Sabourarud's dextrose agar with cycloheximide, gentamicin, and chlortetracycline. These latter three ingredients inhibit contaminant bacterial growth. DTM also contains phenol red which is a pH indicator that will turn the medium red when there is fungal growth.

Daily observation of the culture plate is essential to catch the red color change and fungal colony growth when they first appear. A common mistake is to interpret color change and fungal growth alone to mean the patient is positive for dermatophyte, however, the fungal growth on the culture plate must be examined microscopically because other fungal organisms can mimic dermatophytes. To do this, carefully sample the colony with clear acetate tape, apply the tape to a glass slide over a drop of lactophenol cotton blue stain, and then examine it under 100x-400x magnification for macroconidia.

Macroconidia look different for each species of dermatophyte. Please refer to appropriate dermatology or microbiology literature for the appearance of these macroconidia.

Often pets are incorrectly diagnosed with dermatophytosis due to in-house cultures not prepared or read correctly. Since daily observation of the culture plate may not be conducive for a busy clinic and because examination of macroconidia requires practice, the best recommendation is to send culture plates to a veterinary reference lab.

Cultures of the skin and ears are also important diagnostic tools in dermatology. These diagnostics are not typically necessary at the initial visit but should be considered if the patient is not responding to therapy. If a pet has had repeated exposure to antibiotics without resolution of the skin infection, a skin culture is recommended. Skin cultures are collected through sterile sampling of a primary lesion (i.e., papule or pustule) using a sterile needle. Puncture a papule or pustule and sample the material inside of it with a Culturette swab. Alternatively, collect a sterile punch biopsy of the primary lesion in a sterile tube. If there are no primary lesions present, lift the crust of an epidermal collarette with a sterile needle and sample the area under the crust with a Culturette swab or perform a sterile punch biopsy of the epidermal collarette lesion. For deep pyoderma/draining tract lesions, a punch biopsy of the lesion is always necessary. Swabs of the fluid in the draining tract in these types of lesions may not contain enough organism to give a conclusive culture.

For recurring ear infections that are not responding to appropriate topical therapy or where you see rods on cytology, an otic culture is recommended. *Pseudomonas* bacteria needs to be ruled out, as this organism typically affects the middle ear and requires aggressive treatment. Cultures of the ear are for systemic therapy only and not to guide topical therapy. Topical otic medications will achieve higher concentrations than what is measured in vitro, and therefore, should always work for an ear infection when applied properly.

Biopsy of the skin for pathology is another important diagnostic tool in a dermatology work up. There is never any harm in obtaining a tissue sample for pathology and is warranted for any dermatosis that is not responding to appropriate therapy. A pathology report can help identify the underlying cause of the skin condition, however, it is crucial to obtain samples correctly. Take punch biopsies of multiple sites and a variety of lesions. The best samples are primary lesions (i.e., papules, pustules, vesicles). If primary lesions are not present, then secondary lesions can be used. Biopsies should not include a significant amount of normal skin margin. Therefore, do not biopsy a lesion where half is abnormal skin, and the other part is normal. Depending on how the tissue sample is processed, the abnormal portion may not be sectioned, and the pathologist may only read the normal skin. A 6 mm punch biopsy is recommended as it will provide adequate tissue to analyze. Smaller punch biopsies are best reserved for difficult sites like the pinna, nasal planum, areas around the eye, or foot pads of small patients.

Narrow your list of differentials - what is making this pet itchy?

Use the results of your diagnostics to narrow the list of differentials and generate a treatment plan. Again, client education is important here.

Treatment plan

The treatment plan for the pruritic pet should include medications that will relieve the patient's pruritus, resolve secondary infections (i.e., use of antimicrobials), and if possible, resolve the underlying cause. Depending on the case, the patient will likely require a recheck in a few weeks to ensure the treatment plan is working. Client education of the medications prescribed and patient's prognosis is critical.

Summary

Repetition of this systematic approach to the pruritic pet not only helps diagnose the patient's skin condition, but with practice, it also makes dermatological case work ups flow easier. With constant feedback to the client, this approach also helps create a trusting bond between practitioner and the pet owner.

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The New and Not So New Drugs and Therapies for Atopic Dermatitis in Dogs

Millie Rosales, DVM, DACVD
Email: miamivetderm@gmail.com

Atopic dermatitis (AD) in dogs is currently defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features and is most commonly associated with the formation of IgE antibodies against environmental allergens. The key to this definition of AD is that there are characteristic clinical features to this condition, as there are cases of AD with classic signs but where IgE is not associated with the disease. The term atopic like dermatitis (ALD) has now been introduced to describe patients with clinical signs of AD and no detectable IgE. The latter indicates that for some patients, there is an alternative mechanism of disease and that IgE is not necessary to establish disease. As such, the current definition of AD takes into account that although most commonly IgE antibodies are formed to environmental allergens, there are cases with other triggers to AD, like food. Therefore, there are individuals where food can be a flare up factor, and this blurs the distinction between food allergy and atopic dermatitis in dogs.

Atopic dermatitis is diagnosed based on patient's signalment, clinical signs, disease history and elimination of other pruritic conditions. AD is a diagnosis of exclusion. Most atopic dogs begin showing signs between the ages of 6 months and 3 years. There is no gender predilection. The hallmark clinical sign is pruritus. Recurrent skin infections are also common. Some cases of recurrent skin infections are not pruritic. Otitis externa as the only clinical feature, or in combination with skin lesions, can occur with AD. Ocular conjunctivitis, epiphora, and rhinitis are uncommon signs. The pet's clinical signs may be seasonal to non-seasonal with or without seasonal exacerbation, depending on the patient's environment.

Primary clinical lesions associated with AD are erythematous macules, patches, and papules, which are less often seen. Due to excess trauma from pruritus, secondary lesions like alopecia, lichenification, hyperpigmentation, and excoriations are common. Areas of the body affected are the face, concave aspect of pinnae, dorsal and ventral aspect of the paws, ventral neck, axillary, groin/abdomen, flexor surface of forelegs, medial aspect of the extremities, and perineum/ventral tail.

Other skin conditions can mimic or occur concurrently with AD and must be ruled out. This includes parasitic conditions, like scabies and demodex, and other allergies, like food allergy. It is important to assess for skin infections like *Staphylococcus* and *Malassezia*, which are frequently seen with AD.

When managing the treatment of an AD patient, two concepts should be kept in mind. One is the "pruritic threshold" and the other is the "principle of summation of effects". In the pruritic threshold theory, an individual pet can tolerate a certain level of pruritic stimulus without becoming clinical. Once that threshold is reached, itching will ensue. A heavy pollen allergen load, secondary infections, or even flea bites can push the AD patient over its threshold and lead to flares. The principle of summation of effects underscores that the management of AD is multimodal. Control of the factors that may be contributing to the pruritus along with therapies that will decrease inflammation of the skin is key in the success of managing AD. Each therapy by itself may not be enough to resolve the pruritus but, in conjunction with other agents, helps make a difference. Below is a discussion on the different medications and therapies to manage a dog's atopic condition.

Use of anti-itch medications: Apoquel (oclacitinib), Cytopoint (lokivetmab), Atopica (cyclosporine), and glucocorticoids

The last 8 years have been a revolutionary time for dogs with AD, due to the advent of Apoquel and Cytopoint made by Zoetis. These medications have become the typical go to agents for managing AD. Apoquel and Cytopoint are a unique set of drugs that work differently from steroids and cyclosporine, with a more targeted approach to managing pruritus associated with AD.

Apoquel is the brand name for oclacitinib, the first Janus kinase (JAK) inhibitor approved by the FDA for use in dogs with allergic skin disease. Oclacitinib inhibits primarily JAK-1 and JAK-3 dependent cytokines and other pruritogenic and proinflammatory cytokines important in allergic disease in dogs. One of the cytokines it acts upon is interleukin 31 (IL-31), the key cytokine in causing pruritus in dogs. Apoquel's speed of action is comparable to prednisone and faster than cyclosporine, with a study showing Apoquel's control of pruritus begins within 4 hours. Its quick onset of action with minimal side effects has made it an excellent choice over glucocorticoids. Its safety is supported by Zoetis pharmacovigilance studies of over 5 years that have shown adverse reactions (i.e., vomiting, diarrhea, lethargy, anorexia, and bloodwork changes) associated with Apoquel are rare. Apoquel is dosed at 0.4 mg/kg-0.6 mg/kg twice a day for the first 2 weeks then tapered to once a day. Long term use of twice daily dosing is not recommended due to immunosuppressive concerns. This medication is not approved for dogs less than 12 months of age since during the initial studies puppies given Apoquel at 3-5x the dose developed demodicosis. Skin scraping is recommended of all dogs before starting on Apoquel. Apoquel should be avoided in patients with pre-existing cancers. Apoquel can be administered with or without food, discontinued without tapering, and combined with many medications that are typically given to AD patients (i.e., antimicrobials), except for glucocorticoids and cyclosporine.

Cytopoint, or lokivetmab, is a USDA approved caninized monoclonal antibody for canine allergic skin disease and AD. After subcutaneous injection it remains in circulation for several weeks and can control signs of pruritus from 4-8 weeks. Cytopoint works by binding to and neutralizing soluble IL-31, making it unavailable to bind to cell receptors. The antibody-antigen complexes formed are eliminated by normal protein degradation pathways. The fact that this is a biological medication and not a chemical drug per se is appealing to some clients. Cytopoint comes in 1 ml vials in four dosing concentrations. Since there are no preservatives in the vials, it is a one-time use only. The average dosing for Cytopoint is 2 mg/kg. Zoetis provides a convenient dosing table as a guideline. Effects can be seen within one day with full results by day 3. Adverse reactions are rare but may include vomiting, diarrhea, and lethargy. The effects of Cytopoint gradually wear off, which alerts the client that the patient needs another dose. Cytopoint is safe for dogs of all ages. One of the best features of Cytopoint is that it has no known drug interactions and can be combined with various medications used for atopy. Cytopoint is a great option for: clients that may have difficulty administering medications, dogs under year of age with atopy, flexible dosing for toy dogs, patients with preexisting cancer, and for clients who are looking for non-drug options.

For patients with seasonal allergies where their symptoms last a few months per year, Apoquel and Cytopoint are good options because their onset of action is rapid. Apoquel can be started when the first clinical signs appear and used daily for the duration of the allergy season. Cytopoint can be used in a similar fashion. For AD patients with year-round signs, daily administration of Apoquel is needed as its antipruritic effects wear off within 24 hours. Cytopoint can be used as a sole treatment for chronic cases of AD at every 4-8 weeks. The ability to combine Apoquel and Cytopoint makes these agents excellent

options when managing an atopic pet. The severely atopic patients may need to use Apoquel and Cytopoint concurrently long term or only during points of flare ups.

Cyclosporine was the main therapy available for AD before Apoquel and Cytopoint arrived to the market. It has been in the market for over 15 years, and it is still a useful medication in those AD patients where Apoquel and Cytopoint have failed and where the pet cannot tolerate glucocorticoids. Cyclosporine works well for the unique set of atopic dogs that present with pododermatitis and interdigital nodules/cysts. Cyclosporine is a calcineurin inhibitor that works by suppressing T cells and IL-2, which eventually leads to reduction in pruritus. Cyclosporine microemulsion formulation is the recommended form to use for AD cases. There is question on the bioavailability of generic formulations of cyclosporine, therefore it is recommended to start with the Atopica cyclosporine brand. If generic cyclosporine has to be used, it should be the modified microemulsion formulation. The slow onset of action of cyclosporine makes this product unsuitable for the AD patient exhibiting an acute flare. However, cyclosporine is an option to manage the patient with chronic or year-round AD. It can be combined with oral prednisone for the first few weeks to alleviate clinical signs, this buys time for the cyclosporine to work. Oral cyclosporine should be administered at 5 mg/kg once daily until clinical signs are controlled, this can typically take from 4-8 weeks. Once signs are in remission, then the dose is adjusted by either decreasing the frequency (i.e., every other day) or decreasing daily dose. Food may affect absorption of cyclosporine, and in dogs, it is recommended to administer it on an empty stomach. However, a fatty meal may help increase absorption in some dogs, and anecdotally, a full meal may help reduce the side effects of vomiting and diarrhea that sometimes occur. Caution with the concurrent use of drugs that inhibit the cytochrome P450 microsomal enzymes (i.e., azoles) as these can cause increased cyclosporine levels and toxicity. Gingival hyperplasia, skin growths, and hirsutism are uncommon side effects of cyclosporine. Although newer allergy drugs have replaced cyclosporine as the first choice for AD, this therapy should not be overlooked.

Glucocorticoids (GC) or corticosteroids have been around for decades and withstood the test of time. Glucocorticoids mechanism of action is non-specific in comparison to Apoquel and Cytopoint's targeted action. If used correctly GC can be helpful in the management of an AD patient. Short acting GC can be used to manage acute cases of AD. Prednisone or prednisolone at 0.5 mg/kg twice daily tapered to once daily then to every other day over a few days to weeks can lead to a considerable improvement in a dog's pruritus and alleviate the acute flare up. Long term use of oral GC to manage AD is not recommended unless other therapies have been attempted and failed. If the latter is the case, then a short- acting oral GC can be used and tapered to the lowest dose and frequency that will keep the patient comfortable and minimize side effects. Keep in mind that, over time, tachyphylaxis may occur with GC, necessitating higher doses. There is a predisposition for secondary skin and urinary tract infections with long term use. Biochemistry abnormalities such as elevated alkaline phosphatase and elevated blood glucose levels can occur. Long term steroids can lead to atrophy of the skin and, in rare cases, calcinosis cutis. Frequent monitoring through follow up exams and bloodwork, and urinalysis should be the standard of care for AD patients on long term GC. The use of long-acting injectable GC should be avoided in an AD patient, as it is impossible to take back any potential side effect it may cause and impossible to taper the dose. If improvement of atopic signs is not seen with oral steroids, it is important to look for other causes of pruritus, like secondary infections, ectoparasites or food allergy.

It is not uncommon that some AD patients need a combination of the above allergy drugs to manage their clinical signs. Strive to combine these medications for a short period or when there is an acute

episode or flare. Cytopoint is excellent in that it can be combined with any of these agents. Apoquel should not be combined with GC or cyclosporine due to concerns of immunosuppression.

There is no standard recommendation for laboratory monitoring (i.e., hematology, serum biochemistry, and urinalysis) for long term use of Apoquel, Cytopoint, or cyclosporine administration. The author does recommend yearly to twice yearly lab monitoring with or without urinalyses and urine cultures.

Use of antihistamines

Histamine is not the only inflammatory mediator of AD. This is why antihistamines provide a mild reduction of pruritus for most AD patients. However, they may be of benefit as an adjunctive therapy with other allergy medications. Antihistamines work best before the allergy flare up has started to block the effects of histamine. Common first-generation antihistamines include diphenhydramine (Benadryl), chlorphenarimine, and hydroxyzine (Atarax). These types of antihistamines can cross the blood brain barrier and, therefore, have sedative effects, which may explain why it appears to work in some dogs. These antihistamines must be used more often to see results. Second generation antihistamines are less likely to cross the blood brain barrier so there is minimal sedative effect. These are also longer acting antihistamines and include loratadine (Claritin) and cetirizine (Zyrtec). Antihistamines are not effective for moderate to severe AD patients, and therefore, are best reserved for mild AD cases. This class of drugs appear to work better when given on a continuous daily basis as a preventative. Individual responses to the different antihistamines can be vary, so a trial with each type may be necessary. See Table 1 for doses. Since antihistamines are inexpensive and have minimal side effects, clients often ask about their use. These can be combined with other allergy medications, but it is important their use is consistent.

Use of oral essential fatty acids (EFAs)

Essential fatty acids (EFA) only provide a small benefit in reducing clinical signs of AD. They are not enough to be used as a single therapy. Their benefit can come from improvement of the quality of the coat and may affect superficial skin lipids, therefore improving the skin barrier. Oral EFA provide no relief for acute episodes of pruritus, since it can take several weeks for it to incorporate itself on the skin and show any benefit. EFA can be found as oral supplements or in enriched diets, the latter of which provides higher amounts of EFA. Oral EFAs can normalize the stratum corneum lipid similarly to topical EFA, therefore there is no benefit in doing both. EFA and antihistamines work synergistically together and can be used as a long-term therapy for AD. There is no good information on the specific omega 3 and omega 6 combination, dosage, ratio or formulation that will lead to pruritus reduction and improvement of the coat and skin barrier. The rule of thumb for EFA use has been 180 mg EPA per 10 lbs.

Managing bacterial and yeast infections

Dogs with AD are prone to secondary bacterial and yeast infections on their skin and ears. Studies have shown that dogs with AD have higher number of staphylococcal and *Malassezia* organisms on their skin in comparison to normal dogs. Research also has shown that some of these AD patients have a hypersensitivity to these organisms, initiating and perpetuating clinical lesions and pruritus in these pets. It is especially important in these AD patients to manage the secondary infections with the

appropriate systemic and/or topical medications. Many times, treating the secondary infection is enough to reduce the flare and bring the patient back into remission.

Topical therapy: Good hygiene of the skin

Topical therapy of the skin is important and essential in the management of AD. The role of topicals in the management of AD is removing allergens and organisms from the skin, potentially restoring the skin lipid barrier, and providing pruritus relief. There are a variety of topicals available. Many topicals contain antimicrobial ingredients (i.e., chlorhexidine, azoles) that will remove microorganisms. Topicals that contain ingredients like lipids (i.e., ceramides, cholesterol, and EFAs) and phytosphingosine can aid in repairing the skin barrier. The use of a low potent steroid topical can also help reduce pruritus and lesions. Care must be taken to avoid causing steroid-induced skin atrophy especially if the topical is repeatedly applied over the same skin area. The more often bathing is performed properly, the greater the effect on reducing pruritus. The benefit of topical therapy is even higher in dogs with mild signs of atopy, where in this may be enough to control clinical signs without the need of any systemic medications. All AD dogs should be bathed once to twice a week regularly.

Intradermal skin testing and allergy serology testing for immunotherapy

Intradermal skin testing (IDST) and serology testing will identify IgE hypersensitivity to particular environmental allergens. It is important to keep in mind that these tests will also show positive reactions in dogs not having signs of atopy and should not be used as a diagnostic tool. Therefore, these tests cannot be used to diagnose a dog with atopy or be used to differentiate from other pruritic conditions, like food allergy. IDST measures cutaneous IgE to the allergen and serology measures circulating IgE. The purpose of these tests is for formulation of allergen specific immunotherapy (ASIT). If the client is not interested in immunotherapy, then these tests are of no value since complete avoidance of the offensive allergens is not possible and ineffective at reducing clinical signs. There is debate over which allergy test is best. IDST is considered the gold standard because it tests the affected organ - the skin. Since IDST is mainly performed by veterinary dermatologist, referral will be necessary. If referral is not possible, then a reliable and reputable serology lab should be chosen. Recent studies have shown that results of serology tests can vary between laboratories which impacts the ASIT. A good test result should match the patient's environment and clinical signs, especially if the pet exhibits a seasonality. For example, if the test shows that a pet is allergic to mango trees and there are no mango trees in the area, then that result makes no sense and should not be included in the immunotherapy. As a result, one important factor in success with immunotherapy is interpreting the results of the test in relation to the patient and choosing the right allergens. ASIT can take months to over a year to impact clinical signs. The client must be properly informed of this and be prepared to dedicate the time. Due to the delay in onset, the patient will need an allergy medication or therapies to alleviate AD signs and keep it comfortable until immunotherapy is effective. It is important the client understands this and does not abandon immunotherapy too soon. Apoquel, Cytopoint, and cyclosporine are good options to initiate with immunotherapy. Traditionally immunotherapy is performed via subcutaneous injections, but sublingual immunotherapy is now also available. The ease of sublingual administration is counteracted by the fact that the client must remember to administer it much more frequently – up to twice daily. There is no particular immunotherapy protocol for subcutaneous injection that has proven to be most effective. The literature has information on traditional versus rush (sped up) versus low dose procedures. Regardless of the protocol followed, the second most important factor for success with

immunotherapy is feeling comfortable making adjustments to the frequency of injections and volume injected depending on how the pet is responding or if there are adverse reactions. Consultation with a local dermatologist or laboratory where the immunotherapy was developed may be necessary. There is no information if ASIT should be continued for the pet's entire life. Once a patient has had prolonged remission of AD signs, immunotherapy injections can be reduced to once a month. The ease in frequency of administration is often a welcome change for the client. A retrospective study of pet owners with atopic dogs that had their pets on immunotherapy over a year showed that two thirds had satisfactory to excellent response. The average improvement on immunotherapy is from 60-70%. This effectiveness may be enough to keep the pet off allergy medications or lessen the frequency of their use.

If the practitioner feels comfortable managing an atopic pet on immunotherapy, and the client is willing to take on the responsibility, then this is definitely a worthwhile therapy for an atopic pet.

In conclusion, AD is a life-long condition that will require maintenance therapies. There is now an arsenal of drugs and therapies available, and a combination of these is often necessary for the best outcome. The concurrent use of antihistamines, EFA, emollient and antimicrobial shampoos, and ASIT may allow reduction in the dose and/or frequency of oral glucocorticoids, cyclosporine, Cytopoint, and Apoquel required to maintain remission of clinical signs of AD.

TABLE 1:

Antihistamine	**Dog Dose**
Diphenhydramine	2.2 mg/kg every 8-12 hours
Chlorphenarimine	0.4 mg/kg every 8-12 hours
Hydroxyzine	2.2 mg/kg every 8-12 hours
Cetirizine	0.5 mg/kg- 1 mg/kg every 24 hours
Loratidine	1 mg/kg every 12 hours

Resources:

Mueller and Kirk's Small Animal Dermatology. Miller, W.H., Griffin, C.E., Campbell, K.L. 2013. 135-152, 365-388.

Treatment of Canine Atopic Dermatitis: 2015 updated guidelines from the International Committee on Allergic Diseases of Animals. Olivry,T., DeBoer, D., Favrot, C., Jackson, H., Mueller, R., Nuttall, T.,Pascal, P. BMC Veterinary Research (2015) 11:210

Zoetis website on Apoquel and Cytopoint

Otitis in Dogs

Millie Rosales, DVM, DACVD
Email: miamivetderm@gmail.com

Otitis externa is a common clinical problem encountered in general practice. Recurrent otitis can be a challenge to treat and manage. It is important to understand how to prevent an ear infection from becoming chronic. This lecture focuses on the diagnostic approach and management to otitis in the canine patient.

The anatomy of the ear and the otic exam

The pinna and external ear canal collect sound waves and transmit them to the tympanum membrane and auditory ossicles. The external ear canal is divided into the vertical and horizontal parts. The auricular projection is a fold of cartilage in the vertical ear canal that blocks access to the horizontal portion of the ear canal. During an otoscopic exam the auricular projection is moved by pulling on the pinna dorsally then laterally.

The skin that lines the ear canal is normally a smooth surface with a thin epidermis and dermis that contains adnexa (i.e., hair follicles, sebaceous and ceruminous glands). The vertical ear canal has more adnexa than the horizontal ear canal. There is breed and individual difference in the amount and density of glands and hair. Ceruminous gland density has been associated with the development of otitis externa.

The tympanum separates the external ear canal from the middle ear cavity. A normal tympanum is a translucent membrane with a white C shaped area in the dorsal part that corresponds to the manubrium of the malleus bone. The tympanum is divided into two parts: the pars flaccida and pars tensa. The pars flaccida is the smaller portion that is next to the manubrium. The pars flaccida can be dilated and distended and is sometimes confused with a mass effect. The pars tensa is the ventral portion of the tympanum.

The middle ear consists of the tympanic cavity, medial wall of the tympanic membrane, the auditory ossicles, and auditory tube. The tympanic cavity is divided into three parts: dorsal, middle, and ventral. The latter part is the largest portion and is the site that traps debris when otitis media is present.

It is good practice to examine the ears of all dogs to understand the variants of normal for different breeds. When performing an otoscopic exam, the key things to look for and note in the medical record are: 1) what type of discharge is present in the ear (i.e., ceruminous or purulent); 2) description of the otic canals (i.e., stenosis, hyperplasia); 3) tympanum presence or not and tympanum appearance (i.e., translucent, cloudy, or bulging); and 4) presence of growths in the ear canal.

In addition to the otic exam, a good history is necessary to understand the progression of the ear problem in the pet. Since most chronic otitis cases also have concurrent skin lesions, a dermatological exam of the entire body and a history of any concurrent skin problem(s) are also important to arrive at the primary cause for the otitis externa.

Many dogs with otitis are painful, and it is difficult to do a thorough otic exam. If this is the case, sedating the pet for the exam may be necessary, or, if it is not contraindicated, sending the pet home

with oral steroids to decrease the inflammation in the ear and reevaluating the pet in a few days to a week.

Otitis diagnostics

All otitis cases should have ear cytology performed upon presentation. Ear cytology is simple to do in-house. Cytological examination does not establish the primary diagnosis, but it identifies secondary infectious agents. Cytology is repeated after the course of therapy to evaluate treatment progress and to decide if treatment can be discontinued. *Malassezia*, cocci bacteria, such as *Staphylococcus* and *Streptococcus*, and rod bacteria, such as *Pseudomonas* and *Proteus*, are commonly seen on ear cytology. The presence of numerous bacteria in the absence of an inflammatory response in the ear usually indicates colonization and not clinical infection.

Ear cultures are not necessary for first-time otitis cases. Culture and susceptibility testing are recommended in dogs that are not responding to appropriate topical therapy, when rods are seen on cytology (to rule out *Pseudomonas*), and for otitis media cases. With the increase in methicillin resistant staphylococcal infections, cultures should also be performed if the clinician suspects this organism. Cultures guide treatment for systemic or oral therapy. Cultures should not be used to guide the choice of topical therapy since topicals will achieve higher concentrations in ears than what is measured in vitro through cultures. If the topical therapy is applied and dosed correctly, it should treat the otitis.

An ear smear is another diagnostic tool which is done if there is suspicion of *Otodectes* mites.

The three P's in otitis

Primary causes for otitis externa

To prevent recurring and chronic ear infections, it is important to investigate for the primary cause(s) of the otitis externa. Primary causes create ear disease in a normal ear without any other cause or factor. The primary cause may not be noticed by the pet owner until secondary causes occur. Once the primary cause changes the ear microenvironment, secondary infections develop. The most common primary cause for otitis externa is allergies. Allergies to food and atopy are by far the most common reasons a pet develops an ear infection. Allergies can cause both unilateral and bilateral otitis, and it may be the only presentation, without signs of skin problems. Mites like *Otodectes*, or even *Demodex and Sarcoptes*, are other primary causes of otitis. Endocrine disorders, like hypothyroidism and hyperadrenocorticism, can also make a dog prone to ear infections and should be on the differential list in any older dog that presents with no prior history of ear issues. For a patient with unilateral otitis externa, growths in the ear canal or a polyp should be ruled out. Finally, autoimmune disorders, keratinization disorders, and foreign bodies are other primary causes. Examples of foreign bodies include plant awns, sand, or dried out ear medications. These foreign materials can cause inflammation of the ear canal and subsequent infection. Counseling the client about these primary causes is key when working up an otitis case. It is important to find the underlying cause and resolve it or manage it to prevent repeated ear infections and further damage to the ear canal.

Secondary causes and perpetuating factors

Perpetuating factors and secondary causes of otitis externa are considered separate entities; the terminology is not interchangeable. The reason for this is the treatment and prognosis of secondary

causes is different from perpetuating factors. Secondary infections are easier to treat and resolve more quickly than perpetuating factors.

Secondary causes create further disease in an already abnormal ear. Secondary causes are infections caused by *Malassezia* or bacteria, like *Staphylococcus pseudintermedius*, or the gram-negative organisms *Pseudomonas, Proteus, E. coli*, and *Klebsiella*. *Pseudomonas* is most commonly seen in chronic otitis cases. With the exception of *Pseudomonas*, these infections can be eliminated with proper treatment. If the primary cause or perpetuating factors are not addressed, these infections become chronic or recurrent and difficult to resolve. Don't focus entirely on treating these infections and forget to investigate for primary causes.

Perpetuating factors occur after the primary cause and secondary infections appear. They are the result of otic inflammation that changes the anatomy and physiology of the ear canal, ear drum, and middle ear cavity. Over time, these factors lead to chronic changes to the ear (i.e., hyperplasia and stenosis) which prevents adequate response to medical treatment of infections. These factors can lead to relapse of ear disease even when the primary cause is addressed.

Otitis media, extension of infection from the external ear through the tympanic membrane, is another perpetuating factor. Otitis media is a common reason for recurrent otitis externa, as it is a source of infection that reinfects the external ear.

Predisposing factors

These are factors that are present prior to ear disease formation, and they increase the risk for development of otitis externa. It is important to keep these in mind, and if possible controlled, in otitis externa cases.

Pendulous ears and hairy ear canals can be associated with an increased incidence of otitis externa. It is suggested that there is an increase in relative humidity of the ear due to these factors.

Narrowed ear canals, notoriously seen in some brachycephalic dogs, is a predisposing factor. This can be a compounding and complicating factor in dogs that are already prone to allergies and whose ears become inflamed and more narrowed with an allergy flare up.

Excessive moisture in the ear from swimming can be as factor precipitating ear infections. One concern is that the water getting into the ears is contaminated with bacteria which leads to secondary infections. Detergents from bathing with a shampoo may get into the ear, irritate the ear canal, and lead to secondary infections. Typically, however, when a dog's ear flares up after a bath or swimming, it is due to another problem, such as atopy.

Otitis Media

A common reason why otitis externa becomes chronic is due to otitis media. Otitis media should be considered as a differential when there is: recurrent otitis externa of greater than 3-6 months duration, loss of an intact tympanum or abnormal appearance to the tympanum, *Pseudomonas* bacteria on culture, head tilt, or neurological signs (i.e., vestibular signs).

Repeated infection in the external ear canal can eventually lead to a ruptured tympanum, which allows bacteria and exudate to migrate into the middle ear. The ruptured tympanum regrows and closes,

trapping infection behind the ear drum. In these instances, the tympanum may appear cloudy or bulging. Exudate trapped in the middle ear cavity cannot be easily removed through routine at-home cleaning or sedated external ear flush. A middle ear flush with or without myringotomy is necessary in these cases. Video otoscopy devices or a syringe attached to red rubber catheter that can reach the tympanic cavity are used to remove infections and exudates from the middle ear.

Treatment of otitis media involves 8-12 weeks of systemic antibiotics based on culture of the middle ear, topical antibacterial therapy, and frequent cleaning. Corticosteroids, both topical and oral, are often necessary in these cases to reduce inflammation in the ear. Pain medication may also be necessary for a few days after the middle ear flush procedure. It is important to recheck every 2 weeks to evaluate for tympanum regrowth and assess that the treatment plan is working. An ear cytology should be performed at each visit.

Resistant infections

Pseudomonas otitis infections are challenging otitis cases. Clinically these cases present with purulent to black tarry exudate and erosive to ulcerative otic canals of one or both ears. The intrinsic multiresistant genes of *Pseudomonas* bacteria plus their gram-negative cell wall, efflux pumps, and biofilm production contribute to their natural resistance to antibiotics. Their destructive enzymes (i.e., proteases and collagenases) cause significant epithelial damage to the ear canal and can rupture the tympanum. It is for this reason that most *Pseudomonas* bacteria cause otitis media. The majority of these cases need to be treated as middle ear disease.

End Stage Ears

End stage ear disease occurs when there is severe stenosis of the ear canal due to hyperplasia, proliferation, fibrosis, and in some cases, mineralization of the auricular cartilage. These chronic changes to the ear prevent proper drainage of otic exudate, making it difficult to properly clean the ear and apply medications. Dogs with end stage ears can no longer be treated medically. It is only through surgical removal of the ear canal via a total ear canal ablation procedure that the patient can find resolution and relief of the ear infection. It is important to council the pet owner of the importance of proper diagnostics and management to prevent the end stage ear.

Treatment

Ear cleaning

The ear has a self-cleaning mechanism through a process of epithelial migration that starts from the tympanic membrane outward and upward to the external auditory meatus. When the ear is diseased, this cleaning mechanism can fail, leading to the accumulation of toxins, microbes, and debris. This can inactivate topical medications. Sometimes the primary and secondary causes are addressed and yet the ear relapses with an infection within a few months. In these cases, the epithelial migration has not returned to normal, and cerumen and debris build up again, causing changes in the ear canal environment leading to a recurrent infection. These relapses can be avoided by cleaning the ear routinely until normal epithelia migration returns, which can take years or it may never return to normal. This is why ear cleaning is an essential part of successfully treating otitis externa.

Cleaners are divided into ceruminolytic cleansers, milder cleansers, and antiseptic/drying agents. Ceruminolytic cleansers contain agents that are potent surfactants and detergents that break down waxes and lipids. These agents are irritating if left in the ear long term and, therefore, need to be flushed out. These products can also be ototoxic if left in the middle ear. Milder cleansers can be left in the ear. These have mild ceruminolytic effects and are best used for the normal to slightly dirty ear. Antiseptic/drying agents help to dry ears and control microbes in the ear. There are now cleaning agents that have a combination of ingredients.

The client must be taught how to clean their pet's ears properly. If the dog resists cleaning due to pain, treat with anti-inflammatory medications for 3-5 days before starting routine cleaning therapy.

Topical therapy

Topical therapy alone is usually sufficient to treat otitis externa cases. If an ear canal is proliferative, then combine topical therapy with systemic glucocorticoids. There are variety of topical ear medications available. The main ingredients in these products are glucocorticoids, antibiotics, and antifungal/yeast ingredients.

Topical glucocorticoids are important for their antipruritic and anti-inflammatory effects in the ear. These can be used as single agents in some cases of atopic otitis. In cases of *Pseudomonas*, topical steroids are essential to reduce the damage caused by the bacterial endotoxins. Adrenal suppression has been documented following treatment with topical ear medications containing steroids. Therefore, be cautious with their long-term use. However, the benefit of decreasing inflammation in the ear must be weighed against potential side effects.

Topical antibiotic therapy must provide a strong likelihood of killing the organism with a low risk of developing resistance. The first choice topical ingredients for gram-positive bacteria are neomycin, gentamicin, and florfenicol. For gram-negative organisms, the recommendation is polymyxin-combination topicals and fluoroquinolones at high concentrations. A common reason the infection persists is lack of owner compliance and improper medication application. The ideal concentration for an effective kill occurs with any product if enough volume of the medication is applied to the ear and remains in contact with the infected area.

Antifungal agents are used to treat *Malassezia* otitis cases. Most yeast infections occur concurrently with bacteria, and therefore, the combination otic preparations work well for both organisms. Common antifungal ingredients include miconazole, clotrimazole, ketoconazole, terbinafine, and posaconazole. Nystatin and thiabendazole are too weak for *Malassezia* infections, especially for moderate to severe cases.

<u>Side effects of topical therapy</u>

Ototoxicity

Ototoxicity occurs when a topical or systemic medication enters the inner ear and damages the cochlear or vestibular functions. Clinical signs of ototoxicity are hearing loss and vestibular signs. This may occur with otitis media as the inner ear membrane may be more porous due to inflammation of the area. Chlorhexidene, gentamicin, tobramycin, amikacin, propylene glycol, alcohols, and polymyxin are known

to be ototoxic. The risks of ototoxicity due to one of these ingredients is low and should be weighed against the benefits of using these medications for proper treatment of infections.

Contact reactions

Contact reactions are an uncommon side effect of topical ear medications. Neomycin is a common culprit, however, the vehicle in topical medications could also be a causative agent. Typical clinical signs include erythema and papules of the concave surface of the pinna, inflammation/hyperplasia of the otic canal, and there may be a purulent exudate with no organisms seen on cytology. Treatment involves discontinuing the topical medication and flushing the ear with saline. Corticosteroids tapered over a week can reduce inflammation.

Conductive hearing loss

This is a phenomenon recently seen with long-acting topical ear medications and ear packs. The occlusive nature of these products leads to a conductive hearing loss. The majority of these cases resolve after flushing the ear with saline or an ear cleaner over several days to remove the topical ear product.

Systemic therapy

Indications for systemic therapy for otitis externa include presence of otitis media, proliferative changes obscuring the ear canal lumen, inability of owner to administer topical therapy, and ineffectiveness of topical therapy.

Systemic glucocorticoids are an important in the treatment of otitis externa. They should be used when there is significant ear canal inflammation and when there is marked proliferative ear canals with lumen obstruction. If the otic canal is not open, topical medications cannot reach the entire ear canal to kill the infection.

Systemic antibiotics are rarely used for otitis externa. Oral antibiotics are recommended when there is evidence of otitis media and should be based on culture. The antibiotic should be dosed at the high end of the recommended range. For *Pseudomonas* infections, fluoroquinolones are ideal, and the top choice is marbofloxacin at 5 mg/kg daily. Otitis media should be treated systemically with antibiotics for 2 months or longer, especially if there is evidence of osteomyelitis in the tympanic bulla.

Oral antifungals can be used for *Malassezia* otitis media cases. Systemic ketoconazole, itraconazole, and fluconazole are effective.

Follow up on otitis externa

A follow up exam, approximately 14 days from the start of therapy, should be performed to assess treatment progress. A full otoscopic exam of the ear and cytological examination is necessary at that time. The importance of diagnosing the primary cause for the otitis should be addressed with the client to prevent a recurrent infection. For the atopic otitis patient, long-term management of the atopy is key. Medications like Apoquel, Cytopoint, and Atopica are revolutionary drugs to control atopic dermatitis signs but are limited in controlling recurrent atopic otitis. Immunotherapy, routine ear cleaning, and topical corticosteroids to control inflammation of the otic canals are effective at managing atopic otic cases.

In conclusion, some otitis cases are challenging despite proper exam, diagnostics, treatment, and control of the primary cause. To prevent further damage to the ear and possible end stage ear, referral to a dermatologist may be necessary if the otitis does not resolve.

Resources:

Mueller and Kirk's Small Animal Dermatology. Miller, W.H., Griffin, C.E., Campbell, K.L. 2013. 741-767.

Paws and Claws: Common Conditions that Affect the Feet of Dogs

Millie Rosales, DVM, DACVD
Email: miamivetderm@gmail.com

Introduction

Pododermatitis is a descriptive term to mean inflammation or infection of the skin of the paw; it is not a definitive diagnosis. Affected areas on the paw may include the interdigital spaces, foot pads, nailbeds (paronychia), and nails. Foot problems are common in dogs since their feet are exposed to a wide array of surfaces. Trauma from rough ground surfaces, contact reactions from irritant chemicals like weed killers or fertilizers, or clipper irritation from a grooming episode are common and easy to identify. There are, however, other conditions that can affect the paws. Clues to differentiate between the different disease processes are discovered from the history and physical exam findings.

Claw disorders as the only manifestation of the disease process on a paw are rare. When there is a claw abnormality, there are typically other lesions present on the paw and digit(s). It is helpful to understand the different terminologies used for claw abnormalities: onychodystrophy (abnormal claw formation), onychogryphosis (hypertrophy and abnormal curvature of claw), onychomadesis (sloughing of claws), onychomalacia (softening of claw), onychomycosis (fungal infection), and onychoschizia (splitting of claw).

Foot anatomy

A dog's paw includes the claws, digital pads, dewclaw, metacarpal or metatarsal pad, and in the forelimbs the digital pad. Depending on the breed, the palmar and plantar interdigital skin has deep folds and webbing that can create a frustrating fold pyoderma, especially in dogs with allergies. Studies show that digital pads 3 and 4 are the main weight bearing pads of the paw, and that the front paws bear more weight than the rear paws. Digital pad 5 and the metacarpal and metatarsal pad also carry a substantial amount of the load. Paw conformation varies between breeds. Greyhounds, for example, have narrow, long paws with minimal distance between the digital pads. Labrador retrievers, however, have wide-based paws with greater distance between the pads.

Paw disorders

A systematic approach is required to work up paw disorders in a dog. This includes a detailed history, including age of onset, seasonality, any signs of systemic involvement, and if other areas of the body are affected. Physical examination of the patient must include the entire body, not just the paw(s).

The most common causes that affect just one paw are trauma, foreign body, or neoplasia. Pododermatitis of one paw due to allergies to food or atopy is uncommon, but when it occurs, other skin lesions and pruritus are usually present in other areas of the body.

Conditions in which multiple paws are typically affected are discussed below.

Infectious causes

Bacterial and Yeast

Bacterial, typically due to *Staphylococcus*, and *Malassezia* infections of the paw are the most common secondary cause of pododermatitis in a dog. Primary causes can be complicated by these infections. Clinically, these paws have a variety of presentations depending on the primary condition and severity. Mild or acute cases may just have erythema, especially of the interdigital areas. In more chronic cases, there may be alopecia, lichenification, hyperpigmentation, crusts, and seborrhea. In some dogs, a brown discoloration of the paws indicates the presence of *Malassezia*. *Malassezia* paronychia and/or claw infections cause a red-brown discoloration of the nailbed and claws. *Malassezia* also causes a rancid odor. These infections are easily detected with skin cytology. If deep pyoderma is present, then tissue cultures are needed. These two infections contribute to pruritus and must be treated with the appropriate antimicrobial therapy. Typically, this requires both topical and systemic treatment; however, depending on the extent of the infection, some mild cases may be treated just topically. If the bacterial infection on the paw is deep, systemic antibiotics must be administered for a minimum of 6-8 weeks plus one month past clinical cure.

Dermatophytosis

The three most common dermatophytes that cause clinical disease in dogs are *Microsporum canis*, *Microsporum gypseum*, and *Trichophyton mentagrophytes*. Dermatophyte infection is often localized to the paw, face, pinnae, and tail. Clinical signs vary depending on the species of dermatophyte and host-fungus interaction. Clinical findings of dermatophytosis of the paw range from superficial lesions, like alopecia, crusts, scales, and seborrhea-like eruptions, to furunculosis with draining tracts, which typically occurs with *Trichophyton*. Onychomycosis (one digit or multiple digits on one paw) may or may not present concurrently with the pododermatitis. Minimal to no pruritus is typically present. However, *Trichophyton* can be significantly inflammatory and can cause pruritus. Secondary bacterial infections, like *Staphylococcus*, can complicate dermatophyte infections and contribute to pruritus. Definitive diagnosis of dermatophytosis is through fungal cultures of hairs and scales, which also identifies the fungal species. Histopathology is not as sensitive as a culture. However, since *Trichophyton* infection can mimic autoimmune skin diseases, a biopsy of the skin should be collected along with cultures for clinically suspicious cases.

Clinical management of all dermatophyte cases must include topical antifungal therapy. For pododermatitis cases, rinses or dips are indicated, allowing the antifungal agent to dry on the skin and exert its effects. Lime sulfur 2% is the most common dip. Systemic therapy is recommended for dermatophyte cases that have lesions elsewhere on the body, more than one paw affected, furunculosis lesions, or where topical therapy alone is ineffective. Ketoconazole, itraconazole, and terbinafine are effective systemic treatments. Treatment should continue until two to three negative fungal cultures are obtained. Environmental control through premise disinfection must be part of the treatment, especially since this is a zoonotic disease.

Other fungal infections that are uncommon and specific to geographic regions are: phaeohyphomycosis, blastomycosis, coccidiomycosis, phythiosis, and lagenidiosis. Please refer to dermatology or microbiology veterinary literature for further information on these infections.

Leishmania

Leishmaniasis is a caused by a protozoan organism of the *Leishmania* spp. Over 30 *Leishmania* species are identified and most are zoonotic. Most cases occur in the Mediterranean and in Portugal. The disease is endemic in South and Central America. In the United States, endemic foci are reported in Texas, Oklahoma, Ohio, Michigan, and Alabama. Dogs coming from endemic areas could show signs of the disease months to years later. The disease is transmitted by blood sucking sandflies. The most common clinical lesion is an exfoliative dermatitis with silvery white scales. Nasodigital hyperkeratosis may also be present. Onychogryphosis and paronychia can be seen on the claw and digit. Systemic illness is varied. Diagnosis is based on finding the organism on cytology from a skin lesion, histopathology, PCR assays of tissue or blood, or serologic tests for anti-*Leishmania* antibodies. In the majority of cases this infection cannot be cured, treatment can bring clinical remission but relapses occur. Commonly used drugs are meglumine antimoniate, allopurinol, and aminosidine.

Distemper

Paramyxovirus causes canine distemper. Besides respiratory, gastrointestinal, and neurological signs, the virus may also produce skin lesions. The classic lesions are hyperkeratosis of the nose and foot pads. The patient is systemically ill, and there is poor vaccination history.

Parasitic: Hookworm infection

Hookworm, or *Ancylostoma* or *Uncinaria*, dermatitis is now a rare disease of the paw, due to the routine use of monthly heartworm preventatives that control intestinal parasites. This condition is basically one of kenneled dogs on grass or runs that have poor sanitation. Unsanitary dog parks can also be a source of infection. Third stage larvae enter the dog's skin on areas of the body that contact the ground. The paws are erythematous and can be swollen and painful. Chronic cases have the typical hyperkeratotic foot pads. Pruritus can be present and varies in intensity. Diagnosis is made by clinical signs, a positive fecal exam for hookworms, and a history of poor sanitation of the dog's environment. Treatment involves an appropriate anthelminthic and cleaning the patient's environment. Monthly prophylaxis with heartworm preventatives is recommended.

Allergies

Food allergy and atopy are the most common primary causes of canine pododermatitis. The paws may be the only allergy presentation, with no other lesions elsewhere on the body. In most of these allergy patients, the paws are initially erythematous. Subsequently, the pet chews and licks, which creates secondary trauma (i.e., alopecia, swelling, excoriation) and secondary infections (i.e., bacterial and *Malassezia*) develop. Secondary infections must be addressed properly as they exacerbate the clinical symptoms. Some dogs present with interdigital cysts or nodules. Diagnosis of these allergies is made through the combination of history and clinical signs. In a patient with nonseasonal clinical signs, food allergy must be ruled out before diagnosing the pet as atopic. Atopy is a diagnosis of exclusion. A proper diet trial should eliminate all clinical signs in a food-allergic patient. If the patient is atopic, then allergy medications to stop the pruritus should be considered along with intradermal/serologic allergy testing for allergen specific immunotherapy. The allergy medication that is best for the atopic pet depends on the individual patient. Apoquel and Cytopoint work well to diminish pruritus. For those atopic cases that present with interdigital cysts, then cyclosporine may be a better option. Since atopic patients are prone

to secondary infections, frequent topical antimicrobial therapy is beneficial, especially in those dogs with significantly webbed feet.

Demodex

It is important to rule out Demodex in all cases of pododermatitis in dogs. Demodex of the paws may be juvenile or adult onset. Demodex can affect just the paws but usually affects other areas of the body as well. In some patients, demodex infection of the paws can be chronic and resistant to therapy. The lesions on the paws can be as simple as alopecia and erythema. For more chronic cases there may be severe edema, lichenification, hyperpigmentation, and furunculosis. When draining tracts are present, it can be painful and pruritic. Careful exam of the paw and interdigital spaces may show plugged follicles or comedones. If the skin of the paw is not severely inflamed, then a superficial skin scrape should suffice for diagnosis. However, if there is significant inflammation and swelling, then a biopsy is best. Most cases of pododemodicosis have secondary bacterial infection. This infection must be addressed along with treatment for the mites. Demodex treatment is easy now with the use of isoxazolines. Treatment should be monitored monthly with skin scrapes. If treatment is working, the skin scrapes should show less mites, less younger life stages, and dead mites over the next weeks to months. Treatment can be discontinued after 2-3 negative (no mites) skin scrapes. Since most dogs often remain on an isoxazoline for flea/tick control, treatment can continue.

Immune mediated conditions

Pemphigus foliaceus (PF) and vasculitis are common autoimmune conditions that affects the paws. These conditions also affect other areas of the body but can be exclusive to the paws. The primary lesion in PF is the papule that rapidly progresses to a pustule and then to erosions and crusts. Predisposed areas of the body in PF are the head, face, and ears. The foot pads are also commonly affected. Clinically, the pads present with fissures and crusts that are often painful and can make a dog lame. Diagnosis is based on histopathology, with the classic finding of subcorneal pustules with acantholytic cells admixed with neutrophils and eosinophils. Treatment involves the use of immunosuppressive drugs, with glucocorticoids being the cornerstone of treatment. Glucocorticoids, like prednisone or prednisolone, are typical starting agents, along with other steroid sparing drugs like azathioprine. Other medications, that can be used along with corticosteroids, are cyclosporine, mycophenolate, and chlorambucil. There are recent anecdotal reports of the use of Apoquel for PF. Doses of immunosuppressive medications are tapered slowly over weeks to months until the lowest dose and frequency is reached that keeps the patient in remission. PF must be clinically and histopathologically differentiated from other conditions like dermatophytosis due to *Trichophyton* and demodicosis.

The use of the term vasculitis is not a definitive diagnosis per se, as it can be associated with multiple causes. It can be the result of a drug reaction, vaccine, or infection. Vaccine reaction appears to be more common in smaller breeds, like Poodles, Yorkshire Terriers, and Maltese. In some patients the cause is never determined and the disease is considered idiopathic. The pathomechanism for vasculitis is assumed to involve a type III hypersensitivity reaction. Typically, the skin is the only organ system involved, but other organs, like the kidneys, may be affected. Skin lesions occur commonly in the extremities and areas of pressure, like the pinnae, tips of tail, foot pads, claws, and elbows. When the paw is affected, the most common presentation is central ulcers or central hypopigmentation on the foot pads. The paw pads may become smooth and thin. Histopathology is recommended for diagnosis. Treatment depends on severity and extent of lesions. The spectrum of medications used vary from

immunomodulatory to immunosuppressive. In mild cases, doxycycline and/or pentoxifylline is recommended. In more severe cases, glucocorticoids along with azathioprine or cyclosporine can be used.

Metabolic conditions

Hepatocutaneous syndrome, also called superficial necrolytic dermatitis or metabolic epidermal necrosis, is a skin condition caused by metabolic abnormalities due to liver dysfunction or rarely a glucagon secreting pancreatic tumor. The cause for the hepatopathy is often unknown, but there are reports of association with mycotoxin ingestion and anticonvulsant medications. The pathogenesis of this condition involves degeneration of keratinocytes likely associated to cellular starvation or some nutritional imbalance. It is proposed that low amino acids in the skin and deficiency of fatty acids, biotin, and zinc play a role in this condition. This syndrome is seen in older dogs. Skin lesions are often found in pressure point areas (i.e., elbows and hocks), distal limbs, foot pads, genitalia, the ventrum, and mucocutaneous junction (i.e., perioral, periocular). Clinically, the affected areas, and especially the foot pads, have a crusting dermatitis with ulcers and fissures. These patients can be systemically ill with signs of inappetence, lethargy, and weight loss. Laboratory tests show elevated liver enzymes, especially ALP and AST, low albumin, +/- hyperglycemia and anemia. Postprandial bile acid levels are abnormal. On ultrasound, there is a classic “honey comb” or “swiss cheese” pattern to the liver. Diagnosis is confirmed with histopathology. Hepatocutaneous syndrome has a poor prognosis, with most patients dying or euthanized within 5 months of the development of lesions. Treatment involves supporting the skin nutritionally with high quality protein (i.e., egg yolk), zinc (zinc methionine 2 mg/kg/day), and fatty acid supplementation. Intravenous amino acid supplementation is available, but it is very expensive and may not be of great benefit. Secondary bacterial and *Malassezia* infections are common and should be addressed concurrently.

Nutritional disorders: Zinc deficiency

Zinc is an important component for healthy skin. Zinc is an essential factor for rapidly dividing cells, like in the epidermis, for biosynthesis of fatty acids, metabolism of vitamin A, and normal immune function. Lesions on the skin due to zinc deficiency include erythema, alopecia, crusts, and scales that typically affect areas of friction like the foot pads, distal extremities, and mucocutaneous junctions. The coat becomes dull. Secondary infections with bacteria and/or *Malassezia* are common.

Decreased zinc absorption due to excessive amounts of calcium, iron, copper, or phytates in the diet can lead to zinc deficiency. However, it can also be a congenital cause, as in decreased zinc absorption and utilization due to a genetic defect in Bull Terriers with lethal acrodermatitis. Two types of zinc-responsive skin syndromes have been recognized.

Syndrome I primarily occurs in Alaskan Malamutes and Siberian Huskies and is associated with defective intestinal absorption of zinc despite these dogs being fed a complete, balanced diet. Bull Terriers and other breeds can also be affected. Initial lesions are erythema followed by alopecia, crusting, and scaling of the muzzle, chin, periocular areas, and pinnas. Crusts appear on pressure points, like the elbows, and on the foot pads. Onychomalacia may be present on the claws. Many of these patients are pruritic. Secondary bacterial and *Malassezia* infection is common. Most cases resolve with zinc supplementation. Where appropriate, the diet should be corrected as well. The recommendation is 2-3 mg/kg daily of elemental zinc. Zinc methionine and zinc gluconate have a higher bioavailability of zinc and are less likely

to cause gastric irritation than zinc sulfate. Zinc may work synergistically with omega-6/omega-3 fatty acids, as some dogs respond better when both are combined.

Syndrome II zinc-responsive dermatosis is caused by zinc deficient diets or diets high in phytates or minerals, like calcium, that interfere with zinc absorption. Hyperkeratosis forms over areas of repeated trauma, like pressure points and foot pads. Secondary infections are common. For many of these cases, correction of the inadequate diet is enough to resolve skin lesions in 2-6 weeks. Zinc supplementation can speed up the response.

For both syndromes, a thorough history, especially on the patient's diet, exam findings, and skin biopsies are key to diagnosis.

Neoplasia

Cutaneous lymphoma is an uncommon neoplasia of the skin of dogs and cats. Histologically, cutaneous lymphoma can be divided into nonepitheliotropic lymphoma and epitheliotropic lymphoma. The latter is more common in dogs. When this neoplasm affects the paw, it causes the foot pads to become hyperkeratotic, ulcerated, and depigmented. Most patients have lesions, including erythema, scales, plaques, and nodules, elsewhere on the body. Mucocutaneous areas can also be affected. Most affected dogs are older in age, with an average age of 9-11 years. Diagnosis is based on biopsy. Treatment is usually palliative. Topicals, like corticosteroids and retinoids, and systemic drugs like chemotherapeutic agents, corticosteroids, retinoids, and interferon may provide symptomatic relief. This condition has a poor prognosis. Most dogs are euthanized within 6 months to a year of diagnosis.

Interdigital cysts

Interdigital cysts are one of the most frustrating conditions to manage in dermatology. Clinically, these lesions appear as fluctuant cysts or hemorrhagic bullae to firm nodules of varying sizes on the dorsal interdigital aspects of the paw. It can affect one or several interdigital spaces of one paw or several paws. If the cysts rupture it drains a serosanguineous or purulent exudate and leaves an erosive or ulcerated lesion. These lesions can be painful and pruritic and can cause lameness. Lesions can resolve spontaneously or wax and wane. These are more often seen in short-coated breeds or dogs weighing more than 30 kg.

One theory proposes these cysts are formed as a result of abnormal friction to the ventral interdigital webbing due to congenital or acquired foot deformities. The abnormal friction causes thickened callused interdigital skin which leads to plugged hair follicles and formation of comedones. The plugged follicles continue to produce keratin which leads to dilatation of the follicle and eventual cyst formation and rupture. Free keratin into the dermis incites a foreign body reaction, which results in furunculosis complicated by secondary deep bacterial infection. Fistulous tracts from the ruptured cyst drain into the dorsal interdigital space, creating the interdigital cysts. Clinically, the ventral interdigital aspect of the paw that coincides with the dorsal cyst has swelling and comedones.

In these interdigital nodular cases, demodex must be ruled out with biopsies. Bacterial infections are common and since the infections are deep, tissue biopsy for bacterial culture should also be performed. The infections should be treated appropriately based on culture.

Besides pedal conformation being a factor, many of these cases have an underlying allergy to food or environmental allergens. Addressing the allergy and secondary infections may be enough to resolve the interdigital problem for some patients. Many, especially those with atopy, need life-long treatment with antimicrobial topicals to control secondary infections as well as allergy medications to control pruritus and inflammation. Foot care is also an important consideration. Keep the foot clean. Minimize foot trauma by keeping the dog away from rough surfaces. Foot trauma can also be caused by constant licking and chewing on the paw from allergies, therefore, addressing the allergy helps. Weight reduction or correcting orthopedic issues may help those patients where too much pressure is being placed on the paw(s). Chronic cases benefit from long term use of glucocorticoids or cyclosporine. Dr. David Duchols describes a laser surgery technique for these cases.

Claw disorders

Conditions that only affect the claws are rare. Typically, when the claws are affected other areas of the paw and body also have lesions, therefore, the rest of the body must be examined thoroughly. One key question in history taking of the patient with claw disease is regarding the vaccination history, especially in the suspected immune mediated case. Claw disease can develop 2 weeks to several months after vaccine administration.

Below are conditions that typically present with primarily claw only abnormalities.

Trauma

Trauma is the most common cause of claw disease, and it is easy to diagnose from exam findings and the history. Trauma often occurs from running on hard or rough surfaces or gravel which leads to an injured nail. Secondary infections can occur from debris embedded into the nail.

Infections

Dermatophytosis, particularly *Trichophyton*, and *Malassezia* are common infections that affect the claw. Dermatophytosis often causes abnormal appearance and softening of the claw, whereas *Malassezia* causes the claw to become a brown-red color. With *Malassezia*, there may also be greasy exudate on the claw fold and pruritus. *Malassezia* is typically secondary to allergies, like food or atopy. Dermatophytosis of the claw requires long term antifungal treatment (i.e., itraconazole) as therapy must continue until the abnormal claw has grown out. *Malassezia* treatment can take a few weeks. Hypertrophy and abnormal curvature of the nail are seen with *Leishmaniasis*.

Neoplasia

Neoplasia usually affects one digit and derives from the nailbed to affect the claw and distal digit. In dogs, squamous cell carcinoma, melanoma, soft tissue sarcoma, and mast cell tumors are most common.

Immune mediated

The most common immune condition to just affect the claw is symmetric lupoid onychodystrophy (SLO). German Shepherd and Rottweilers appear predisposed, but it can occur in any breed. Gordon Setters possibly have a genetic predisposition. The key finding is claw loss or softening of the claw. It is reported that clients initially note claw pain displayed by licking of the paws and lameness, followed by claw

splitting or claw softening. It affects one to several claws on more than one paw. Paronychia and subungual hemorrhage can be seen. Affected feet are often painful. Once the claw sloughs, regrowth of the nail is short, misshapen, brittle, and dry. Except for the claws, the dogs are healthy otherwise. History and clinical signs are very characteristic, and it is suggested this is enough for diagnosis. Biopsies of the nailbed and histopathologic evaluation of the sloughed claw is unrewarding. Definitive diagnosis involves a biopsy of the digit, including the claw bed. The best way to do this is to remove the third phalanx with the intact claw. When affected, the dewclaw is the best claw to remove for histopathology.

Initial treatment involves high dose fatty acids, vitamin E, doxycycline and niacinamide, and pentoxifylline. If treatment is working, new claw growth should be seen within 2 months, although some abnormal claw may still be present. Once the condition has been in remission for 2-3 months, drug doses can be tapered to once per day. If these treatments are ineffective, then systemic glucocorticoids, cyclosporine, or azathioprine can be tried next. Prognosis is good, except for some nails may still appear abnormal. Some form of treatment is needed for life.

In summary, although there are a variety of conditions that affect the paws in dogs, careful examination of all areas of the paws and nails, as well as the rest of the body, plus a good history narrows the list of differentials to help reach a diagnosis.

Resources:

Duclos, D., Hargis, A.M., Hanely, P.W. Pathogenesis of canine interdigital palmar and plantar comedones and follicular cysts, and their response to laser surgery. Veterinary Dermatology. 2008, 19: 134-141.

Mueller and Kirk's Small Animal Dermatology. Miller, W.H., Griffin, C.E., Campbell, K.L. 2013. 231-248, 304-313, 288-289, 346, 354-357, 440-444, 479-488, 540-542, 689-691, 732-739.

Dermatology Pearls

Millie Rosales, DVM, DACVD
Email: miamivetderm@gmail.com

After fifteen years in referral private practice, I would like to share with you some pearls of wisdom to help you with your dermatology patients.

A bump, a rash, what do you call that lesion?

It is helpful to understand the morphologic characteristics of skin lesions and to recognize the difference between primary and secondary lesions. Primary lesions are the initial lesions seen due to the underlying disease and develop spontaneously, e.g., macule, patch, papule, plaque, pustule, vesicle, wheal, nodule, and cyst. Primary lesions can be transient, leaving behind secondary lesions, which are the epidermal collarette, scar, excoriations, erosions, ulcers, lichenification, and callus. Some lesions may be primary or secondary depending on the underlying condition, e.g., alopecia, scale, crust, follicular casts, comedone, and pigmentary abnormalities. Describing skin lesions properly is helpful for other colleagues or referral practices that read the medical records to interpret the progression of the skin disease and response to therapy.

Understanding bacterial skin infections

Since bacterial skin infections are one of the most common conditions seen in general practice, it is important to treat them appropriately. Bacterial skin infections are either superficial or deep. Clinically, superficial lesions consist of papules, pustules, crusts, and alopecia. Deep infections typically have draining tracts, swelling, and can be nodular. Treatment of a superficial infection requires 3-4 weeks of antibiotics. Deep infections require minimum 6-8 weeks of antibiotic treatment, and then one month past clinical cure. With that said, there is some evolution towards minimizing systemic antibiotic use due to the overwhelming number of cases with methicillin resistant *Staphylococcus pseudintermedius* (MRSP) infections of the skin. MRSP should always be in the differential list for a skin infection that is not resolving with proper antibiotic treatment. If a patient has been continuously exposed to antibiotics with no improvement of its skin infection, then a culture should be performed. The recommendations now, if at all possible, is to limit systemic antibiotic use and reserve it for severe superficial skin infections or deep pyodermas. Mild cases should be treated topically with an antibacterial shampoo. If a patient repeatedly develops skin infections, consider the following: make sure the infection has been treated properly (i.e., assess for error in dose/dosing of antibiotic), rule out a resistant bacteria on the skin, and ensure the underlying cause has been addressed.

Don't forget to look for yeast infections on the skin

The most underdiagnosed fungal infection is *Malassezia*. *Malassezia* can cause a significant amount of pruritus in a patient. *Malassezia* also tends to cause a rancid odor and greasy texture to the skin. *Malassezia* goes hand in hand with bacteria like *Staphylococcus*, therefore, both are typically treated at the same time. Mild cases can be treated topically with an antifungal shampoo that contains miconazole, ketoconazole, or climbazole. Moderate to severe cases of *Malassezia* typically require oral antifungal treatment with either itraconazole, fluconazole, ketoconazole, or terbinafine.

If it looks like ringworm, it's probably Staph

Epidermal collarettes due to *Staphylococcus* are often confused for ringworm. Therefore, the most over-diagnosed fungal infection is dermatophyte. When in doubt, confirm the diagnosis of dermatophyte by sampling hairs for fungal culturesand send it out to a laboratory. Also perform skin cytology of the lesions. If cocci are seen on cytology, then begin treatment for pyoderma while awaiting fungal cultures.

Do not let the sun set on a dermatology case without a good skin scrape

Do not forget to scrape all skin cases. Demodex mites should be ruled out in all patients that present with skin problems. When performing the skin scrape properly, the skin should bleed. Examine the slide under 10x microscopy with the condenser down to create more contrast for better visualization of the mites.

Food allergy diet trials are worth it

Food allergy and atopy can be indistinguishable if there is no seasonality. Atopy is a diagnosis of exclusion. It is important to rule out food allergy first. Serology tests for food allergy are unreliable. The gold standard diagnostic for food allergy is a diet trial. This involves switching the pet to either a novel protein or hydrolyzed prescription diet or novel home cooked diet for at least 3 months. Do not use pet store diets to test for food allergy. During the diet trial the patient must not eat any other foods, home food, treats, or flavored medications. If the pet continues to exhibit skin and ear problems while on the diet trial, then food allergy is ruled out. After food allergy is eliminated, atopy becomes the default diagnosis. Once a patient is diagnosed as atopic, then serology or intradermal allergy testing is performed for the purpose of immunotherapy. Do not use allergy testing to diagnose a pet as atopic.

You don't have to see a flea on a flea allergic pet

The classic signs of flea allergy on a dog are rear-end pruritus and skin lesions. If these classic signs are present, you do not have to see a flea for the pet to be flea allergic. An indoor only pet can still get fleas. The flea may come in with the pet owner or another pet. It is important to know the flea life cycle to understand why a few fleas are enough to cause a problem. Environmental flea control is important in the treatment of a flea allergic patient. In dogs, oral flea adulticide, especially the isoxazolines, are preferable over topical flea products that can easily wash off with bathing and swimming. The flea still has to bite and take a bloodmeal with the isoxazoline, but the speed of kill is now so quick with these products that the flea will quickly die before it reproduces.

The good and the bad of steroids

Steroids work great to relieve pruritus and inflammation on the skin. Use of a short-acting steroid tapered over a few days to weeks typically does no harm. Steroids must be used judiciously for long-term control of allergies or other chronic skin conditions. Calcinosis cutis is a rare side effect that results from extensive steroid use. Lesions mimic a pyoderma to the untrained eye often resulting in continued use of steroids and further exacerbating the condition. Long term use of steroids can also cause thinning and flakiness to the skin and telangectasia. Biochemistry laboratory to evaluate liver enzymes and glucose should be considered in dogs with long term steroid use. In addition, exercise caution when using steroids concurrently with antibiotics for a skin infection. Steroids work great to reduce pruritus,

but their anti-inflammatory effects can make a skin infection appear to have resolved, causing the client to stop medications prematurely. Antibiotics stopped before their full course have the potential to result in resistant bacterial infections.

Apoquel and Cytopoint work to reduce pruritus

Apoquel and Cytopoint reduce pruritus. If there is no pruritus, then these medications will not resolve the skin problem. Apoquel and Cytopoint are rarely effective to control recurrent ear infections.

Biopsy for histopathology is an under used diagnostic tool

When all else fails and the skin problem is not resolving, biopsy the skin and send it out for pathology. Epitheliotropic lymphoma, calcinosis cutis, and autoimmune conditions are easily confused for skin infections and allergies.

USING TECHNOLOGY TO DELIVER A BETTER EXPERIENCE FOR YOUR CLIENTS AND YOUR TEAM

Stacee Santi, DVM
Vet2Pet
Durango, Colorado, USA

As a veterinarian, you want to be a trusted resource for your clients and patients, but the world has changed drastically in the past 10 years. You need to rethink how you approach veterinary practice in order to work more efficiently, take care of your clients, patients, and yourself—and make it home for dinner.

These ideas to help you streamline your workflow, expand your client engagement strategy, and enhance your practice's profitability.

TWO-WAY CHAT

Many client communication platforms have a two-way chat function that allows clients to contact you with questions or concerns. On the Vet2Pet app, a client simply selects 'Start a new chat' and designates the message's urgency. Although the owner may misjudge a situation's urgency, this component communicates the owner's perception of urgency, so you know how quickly to respond.
An exchange may look something like this:
Pet owner: "I think my dog ate a sock."
Practice: "Oh dear, when do you think she ate it? What are her symptoms?"
Pet owner: "I'm not sure. I can't find my sock and she is vomiting."
Practice: "We'd better see her. This can be serious. I have an opening at 2 p.m. Can you drop her off?"
Pet owner: "Yes, that works."

Benefits of two-way chat

There are many benefits to two-way chat:

- Each exchange saves at least one phone call
- Incoming chats can be handled by a remote team member
- You can handle multiple clients simultaneously
- Chats can be answered at your convenience, whenever you have a minute (e.g., during a meeting, at your kid's soccer game, etc.)
- It provides a better, more rapid service
- It prevents your clients from turning to Google to solve their pet's problem
- It establishes you as their pet's trusted provider

How to charge for two-way chat

There are many ways you can charge your clients for this service, such as:

- Provide two-way chat as a free service to all clients for a short period of time — This is a great way to get comfortable with two-way chat, and it provides insight into the types of chats you will receive. After a month or two, you can decide when and how you want to charge for virtual services.
- Offer a "Peace of Mind" package — Allow clients to pay a monthly or yearly fee for full access to two-way chat. This can be a great way to generate passive recurring revenue, and once you have enough subscribers, you will be able to pay a team member to man the dashboard, which will take your client care to the next level.
- Teletriage with an in-person appointment option only — Identify a team member to receive incoming chats, identify those that need an exam, and convert chats to appointments.
- Teletriage that advances to paid telemedicine consults — For chats that require an exam, let the client know that a veterinarian is needed to continue. Offer the option to come in, or advance to a telemedicine chat with the veterinarian, and inform them of the fees associated with each option.

SURPRISE AND DELIGHT YOUR CLIENTS

Clients love being surprised with an unexpected treat from you. Brainstorm ways to creatively surprise them, such as:

- Sending a picture of their pet — Send the pet owner a picture of their hospitalized pet with you or your team members. Photos that include situations such as X-ray positioning or extra attention show the owner the value of the services you are providing.
- Mailing a "Get Well Soon" box — Set an alert in your patient management system for any illness invoice that totals more than $500. Then send these patients a get well present, such as a box of white-chocolate dipped dog biscuits with a personalized card.

MINIMIZE CLIENT CONFUSION

Many of the services you provide can't be seen, and your clients may barely understand them. Normal lab results are the perfect example. You know they are important, so help your clients understand their value by changing the way you deliver results. I use Loom (loom.com) to record a video of myself explaining a patient's lab results. Loom is easy to use, has a Chrome extension, and offers a free basic package. After installing the widget onto your browser, simply click it to share your screen, yourself, or both, and make a recording. The saved video can be sent with a cloud-based link, which means there is not upload or download time.

INSTITUTE CONTACTLESS PAYMENT

The check-out experience is one of the most tedious parts of any service visit. After dining at a restaurant, waiting for the waitress to take your credit card is annoying, and your clients will likewise become frustrated with a clunky, inefficient check-out experience. Contactless payment is a critical component to becoming a more efficient practice.

CHARGE FOR VIRTUAL TIME

Knowing how to charge for your virtual time is challenging, since you have been providing this service, in the form of telephone call-backs, for free. Although you know your time is important, how do you make this transition?

If a client insists on speaking with a veterinarian, your staff can let them know that you have a few phone consultation slots available for the same day. For a fee (e.g., $25), you will call them back today, or they can be placed in your phone queue, which is approximately one week out. This option allows your clients to essentially "jump the line" for a small fee, or they can wait for you to call them when you get time. Schedule your consultation slots for 12:00, 12:15, 12:30 and 12:45 p.m. This trick will get you home on time for dinner, since you won't have to stick around to return client phone calls at the end of a long day.

DESIGNATE A VIRTUAL CLIENT CARE COORDINATOR

As you incorporate more of these digital engagement strategies into your practice, you may want to consider appointing a team member as your digital client care coordinator. This person can handle all of your virtual tasks, and more, including:

- Posting to social media
- Engaging with clients on social media
- Sending and replying to emails
- Entering lab results
- Managing post-visit surveys
- Triaging patients via two-way chat
- Submitting pharmacy requests from email, phone messages, and your app
- Answering the phone
- Making reminder calls
- Managing team communications and inspiration
- Reaching out to lapsed clients
- Fielding Facebook Messenger messages

This can easily be a remote position, unlimited by geography. Even if you hire a part time team member, having a virtual client care coordinator will provide an immense opportunity to further engage with your clients, which will translate to increased profitability.

TECHNOLOGY TOOLS TO COMMUNICATE WITH YOUR VETERINARY TEAM

Stacee Santi
Vet2Pet
Durango, Colorado, USA

Have you ever been in a team meeting that sucked? Or worse yet, hosted a team meeting that lacked engagement, motivation, and positive energy? With these tips, boring meetings will be a problem of the past. I'll share the key factors for having a successful, engaging, positive team meeting, and address how to use technology to knock out the boring need-to-know stuff.

PREPARATION DETERMINES OUTCOMES

As a rule of thumb, a successful meeting requires a planning and preparation period that is at least twice as long as the planned meeting time. If you are planning an hour-long meeting, you can expect to put in at least two hours of preparation. In reality, most team meetings are thrown together at the last minute with one or two people in charge of a narrowly focused agenda. This tends to result in rabbit holes, complaining sessions, and limited take-aways.

Also, the typical veterinary practice, like most businesses, has some rather boring, routine operational items that require team awareness and attention. Historically, these are turned into discussion and announcement items on the agenda, but there are technology tools to help make these communications more exciting, so your team will walk away with the information they need.

IN-PERSON MEETINGS

Make no mistake about it, team meetings—real, in-person, live meetings—are one of the single most valuable communication tools a practice can utilize. Or, at least they should be. They are mission control, the hub of the business lifeline. This is where important decisions are made to guide the business and impact patient care. It is where team motivation is re-ignited and everyone rides off into battle. Oh wait—I'm thinking of *The Patriot* starring Mel Gibson. But, isn't this the real purpose of team meetings? Engage, inspire, activate. Before we talk about how to use technology to communicate outside of team meetings, let's talk about how we can leverage the team meeting to its ultimate potential.

The cost of a team meeting can be astronomical; therefore, it is vitally important that you devote adequate planning and preparation time to each meeting. To calculate the cost of your team meeting, take your team's average hourly rate multiplied by the number of team members who will be in attendance. Now, double that number to account for payroll tax, health insurance, and paying unscheduled team members to attend. Next, calculate missed revenue from the appointments you will miss while the business is closed, and add this figure in. Divide this total amount by the total number of meeting minutes to get a ballpark figure of the cost per min to hold this little shindig. Shocked? Now, let's talk about how to get the most out of your meeting.

Pre-game plan

During your planning and preparation, there are several key items you should consider:

- Objectives — Every meeting must have a stated purpose and desired outcome. All too commonly, meetings lack a clear objective. Unfortunately, this leaves attendees wondering if the meeting pertains to them, and to what degree they should participate. And, if you fail to set ground rules, you can bet that your more vocal employees—who sometimes share the minority opinion—will dominate the meeting and drown out your shy team members.
  Bottom line: If you don't have clear meeting objectives and desired outcomes, your meeting will likely be an expensive waste of time for everyone.
- Ground rules — It is important to establish the rules of engagement for your meeting:
  Rule #1: Everyone must be on time—including, and most importantly, leadership. Remember the cost per minute you calculated? Starting your meeting 5 to 10 minutes late can cost you hundreds of dollars.
  Rule# 2: You must have a clear "no cell phone policy" for your meetings. Allowing your team to set their phones to "silent" may not be sufficient for many of us who are addicted to our devices. If there are team members who have kids, or specific situations that require them to be on emergency call, have someone on the leadership team monitor their device for them, or inform their family member to call the boss's phone if there is an emergency during the meeting.
- Agenda — A key ingredient to the pre-game plan is the agenda. Ideally the agenda should be published prior to the meeting so your guests know what to expect. This also allows guests who are a

bit more contemplative time to collect their thoughts prior the meeting, which will lead to a more robust conversation.

- Meeting length — Be sure to establish the meeting length ahead of time, and stick to it. This means you will need to allot a set amount of time to each agenda item to keep the meeting on track. If your team goes off on a 30-minute tangent related to the first agenda item, you'll run out of time before addressing your entire list. An ideal meeting is one hour or less in length. The longer a meeting drags on, the less productive it becomes, because people get bored and restless, and become exponentially disengaged. It is more productive to hold two one-hour meetings instead of one two-hour meeting.
- Guest list — Careful attention to the invite list will ensure that your meeting reaches maximum productivity. Does the entire agenda require all hands on deck to be present? If not, don't shy away from a split meeting, where some guests come later, or leave after certain topics are discussed. Having idle bystanders listen to a conversation that has nothing to do with them will be unproductive, and costly to boot.

Game on

Start your meeting by sharing the purpose and desired outcomes with the attendees. Review the agenda, if needed. In-person meetings with real, live participants are held to discuss, debate, and possibly disagree on a topic, and robust discussion should be encouraged by the leader. However, it is important to establish rules of respectful discussion. Don't allow one or two vocal team members to take over, while quieter employees hesitate to share their thoughts. Keep your meeting on time by holding to hard stops. Setting a timer to ensure conversation stops after the allotted amount of time is a great way to handle this.

Post-game wrap-up

Leave time at the end of your meeting to assign specific action items, ensuring you identify the responsible party, deliverable items, and due dates. To ensure your meetings stay up to snuff, have your employees rate each one on a scale of 1 to 10. Ask questions such as, "How efficient and productive was this meeting?" and "What would have made it a 10?" Have each person write down their rating on a piece of paper, or complete a short survey card, and drop it in a basket on their way out the door (while they collect their cell phones).

TEAM COLLABORATION TOOLS

Now that you have a clear understanding of how to host powerful team meetings that engage your team, let's address the list of mundane operational tasks that you still need to get across to your team. Email is the absolute worst, old-school way to share this information. Why? There are many reasons email will fail:

- Not everyone has an email account
- People often fail to check their email on a regular basis
- Emails can end up in the recipient's spam box, never to be opened
- Unreceived emails leave the door open on accountability

Fortunately, recent technological advances provide affordable, accessible, and simple solutions.

Chat boards

Slack is one example of a fantastic tool that makes team communication easy. Slack can be accessed from a desktop, laptop, smartphone, or tablet, and messages can be one-on-one or parsed to a group. Recurrent group chats can be set up as "channels" for segments of your company like "Reception desk" or "Patient updates." In addition to day-to-day communication, Slack can help a manager get the word out quickly about an operational update, like a backorder situation or the latest COVID protocol.

Planning

Trello is hands down my favorite technology tool when it comes to project management and mapping out your business goals. Practice tasks like organizing team meetings, inventory management, and employee performance evaluations can be set up with timers and checklists to help maximize output and minimize effort.

Google Drive

Looking for a place to store that new microscope protocol or video on how to clean the endoscope? Google Drive can save the day by providing access to your most up-to-date documents. Simply create a Drive and invite all of your team members, and they can access anything you store there. Google docs, sheets, and slides easily replace Microsoft Word, Excel, and PowerPoint. This is a great place to keep important resources, such as your employee handbook, so there's no confusion about where to find them.

Zoom
Saving businesses all over the world during COVID, Zoom makes it possible for teams to come together when an in-person meeting is difficult. It's a great way to hold doctor meetings or leadership meetings, and doesn't require staff to leave their home on their day off. The free version caps meetings at 40 minutes, which is perfect to ensure meetings start and stop promptly.

Team meetings and employee communication are the most important activities that will make or break your business. Using the right tools for the job will ensure success for you and your team.

HOW TO BUILD A LOYALTY PROGRAM THAT BOOSTS REVENUE AND RETENTION

Stacee Santi, DVM
Vet2Pet
Durango, Colorado, USA

Veterinary loyalty programs offer an amazing way to celebrate your clients all year long, but especially at the holidays. Here are 7 tips to building a loyalty program that will set your veterinary practice up for maximum success:

#1: Remember your “why”

If you don’t know why you are doing something, you are destined to fail. Make no mistake, the purpose of a loyalty program is simple: appreciation of the clients you have, influencing your clients to choose your practice for all their pet’s needs, and adding a little spice to persuade clients to accept the doctor’s recommendations for their pet. It’s a trifecta win:

1. The client wins by unlocking rewards that help them save money.
2. The practice wins by driving visits and purchases.
3. Most importantly, the patient wins when their owner says yes to the veterinarian’s recommendations.

We all know that if pet owners would only do everything we recommend, their pet would live their best and longest life. It’s our calling as veterinary professionals. The loyalty program helps get you a little bit closer to that end goal.

#2: Be strategic

It’s imperative that you give strategic thought to setting up your loyalty program, otherwise you will just be creating a fancy discount program. We all know the margin in veterinary medicine can’t support big discounts, especially if you are running under 10% EBIDTA. The key factor here is that all rewards drive more visits, not product purchases. When we drive more visits, we have the potential for higher-level service recommendations, like dental cleanings, lab work, and other important wellness treatments.

One of the biggest mistakes I’ve seen over the years is using your loyalty program to drive more product purchases.

Instead of “Buy 9 bags of food and get the 10th free,” try “Buy 9 bags of food and get a complimentary wellness exam” to drive a larger invoice.

#3: Keep it simple

People get scared of new or “difficult” things, and can easily succumb to analysis paralysis and fail to engage. Keep your loyalty program simple to understand and attractive to participate in. In fact, my rule of thumb is that if you can’t explain your loyalty program in less than 30 seconds, it’s probably too complicated, and you should simplify.

#4: Be enthusiastic

No matter what, make the most of your loyalty program by bringing enthusiasm and excitement. This means when a client spends a lot of money at the practice and earns a large number of paws at once, make a big deal about it! Congratulate them and give a high-five right there when they see the paws light up on their app's loyalty card. And be sure to give a big callout every time a reward is redeemed. I might even go so far as to suggest having a bell you ring at the front desk to celebrate. It is your opportunity to say, "Wow! We are so excited for you. This couldn't happen to a nicer person or cuter pet. It's one way for us to say we appreciate you."

#5: Change it up

The same old loyalty program can get boring after a while, so spice things up by sending out notifications with new ways to earn a paw every month, or every other month. Your clients will be more invested in reaping rewards if there are novel methods to snatch up those precious paws. Pro tip: Add a bonus loyalty item focused on a wellness initiative, like microchips or the annual heartworm test.

#6: Make sure the client has downloaded the app before checkout

The magic will happen at checkout, so ensure your client knows that at the beginning of the visit. Not only will they have an opportunity to browse in the app and learn about other ways to rack up loyalty paws, like refer a friend or giving a review, they will be primed when the time comes to hand over the credit card.

#7: Understand the neuroscience

As veterinary professionals, we love the science, so let's have a look at what exactly is happening and why this will work. Certain things trigger the reward center of the brain to release dopamine and oxytocin. These hormones are responsible for happiness, euphoria, and trust. There are a variety of triggers, such as eating, gambling, drugs, games, sex, and winning a prize. With a loyalty program, we are strategically pairing the activity of paying the invoice with the dopamine boost of having loyalty paws appear in your app, or, if you're so lucky, getting to claim a reward previously earned during the checkout process.

This is one of the most important points of a loyalty program: the timing.
The paws or rewards need to be delivered within seconds of paying the invoice, otherwise the behavior won't trigger the endorphin release, and your program will merely be a fancy way of discounting. I strongly advise against automating this because two things will happen:

1. Purchases won't be celebrated at the front desk, let alone discussed, so the client will not likely be influenced to make additional purchases at the time of sale.
2. With the current technology integration with apps and practice management software, there is a significant delay in applying the loyalty paws in the app (usually 24 hours), which means there is minimal dopamine surge at the critical time of paying the invoice. In addition, many automated systems can't detect if the invoice was paid or not, so, if anything, the client is getting rewarded for racking up the invoice instead of paying the invoice.

Top reward paws

Although you can fully customize your loyalty program, here are a few tried-and-true reward paws that work well for most practices:

- 1 paw for every $100 spent per invoice — This reward helps increase ATC (average client transaction) by taking that $495 invoice to over $500 for an extra paw. Plus, it improves patient care by encouraging the purchasing of recommended dental care products, preventives, or supplements.
- 5 paws for referring a friend (to referrer and referee) —Referrals are the best. By encouraging your clients to share the love, you will continue to build a strong client base.
- 3 paws for reviewing the practice — Clients are rewarded for providing feedback on your practice. If their feedback is positive, they will automatically be encouraged to post online.
- 3 paws for purchasing 12 months of prevention — This reward helps battle Chewy and other online vendors, and works well for cats and dogs.
- 1 paw for placing your Rx refill order through the app — Overwhelmed with phone calls during COVID? Reduce phone calls with this reward.
- 1 paw for completing a patient history form prior to arrival — Streamline your operations by offering this reward through automated appointment confirmation messaging with your online check-in forms.

Top rewards

When choosing rewards, opt for those that drive further service and revenue, except for the ever-popular free nail trim. Here are some winners that practices have great success with:

- $100 credit toward a future purchase (Cost = 20 paws)
- $50 toward a dental cleaning (Cost = 10 paws)
- Complimentary nail trim (Cost = 5 paws)

Avoid loyalty program pitfalls

There are a few pitfalls you need to watch for when building a successful loyalty program, including:

- Reward ≠ free wellness exam — Clients don't understand the monetary value of a wellness exam. Reward your clients with something that speaks to them: money.
- Don't make your clients beg — Your clients shouldn't have to ask for their reward paws. They won't feel like they're winning, and will fail to experience that dopamine boost if they have to beg for paws.
- Exclude no one — Don't forget to reward all your clients, even those without your app.

Don't reward on the invoice amount — Instead, reward on the amount paid at time of checkout. No pay, no play. It is important to require full payment of the invoice at the time of checkout to participate in the loyalty program. Otherwise, get ready to open a can of worms from partial payment to the dreaded "I get paid on Friday" promise.

Giving back to your community

Since loyalty programs are all about giving and rewarding your clients for being awesome pet owners, get into the holiday spirit with your rewards. Here are a few of my favorites you can add to your reward list to perk up the giving mood all year long:

- Allow clients to round up to your favorite charity to get to the next $100 mark — When your client is faced with that $495 invoice, but can round up to their next reward paw by donating $5 to your local animal shelter, they can't say no. You can also start your own practice-run charity through the American Veterinary Medical Foundation (AVMF).
- Give paws for client donations — Everyone gets a paw for contributing to Toys for Tots or Coats for Kids, or for donating non-perishable items to the shelter in town.
- Give paws for showing your library card — This is a great way to encourage kids to read and use their local library.
- Give paws for A's — Support your community's education by handing out a paw for every "A" on a report card.
- Allow clients to donate their reward to a less fortunate client — You'd be surprised how much use this reward gets, especially around the holiday season when people are in a more giving mood.

A solid loyalty program helps you take care of your existing clients, plus it helps you provide the best in patient care by teaming up with the pet owner and rewarding them for doing well. You can't go wrong with your loyalty program, unless you make it all about anal glands.

PET OWNERS HAVE SPOKEN: YOU'RE DOING IT WRONG! CLIENT COMMUNICATION PREFERENCES FOR MEDICAL UPDATES

PROVIDE MORE FREQUENT PATIENT UPDATES

My 17-year old cat, Polly, recently needed I-131 treatment for hyperthyroidism, as she did not respond to methimazole therapy. I made the appointment at a referral hospital six hours away, and dropped her off for a six-day stay. During the drop-off, they let me know they would provide phone updates once a day for the first three days, and after that by request only. Did I want additional updates? Of course I did!

When the first call came, I didn't recognize the number and the call went to voice mail. The second day, I was in a meeting and missed the call. On the third day, I was ready, and finally got to speak with the person calling, who informed me Polly was doing well.

As a pet owner, I wanted more, and I know I am not unique in that regard. I wanted to know if Polly was eating, and if she liked her neighbor. A photo of Polly resting comfortably would have been greatly appreciated. So, I teamed up with two colleagues to survey more than 1,000 pet owners about veterinary patient updates. The study[1] included four key questions.

Question #1: How important are updates to you for your hospitalized pet?

Question #2: What is your preferred method to receive updates about your pet?

Key point: Ninety percent of patient updates are provided by phone, when this is actually the least efficient way to update clients about their pets. Only 51% of clients actually prefer to receive a phone call update, which means there is a great area of missed opportunity here.

Question #3: How frequently would you want to be updated if your pet was hospitalized overnight?

Frequency at which veterinarians actually update owners:
Hourly: <1%
Every 2 to 3 hours: <1%
Every 4 to 6 hours: <1%
Every 8 hours: 8%
Every 12 hours: 48%
Every 24 hours: 42%

Key point: Pet owners want to be updated about their pets more frequently than they are, and with today's technology, they know it's possible.

Question #4: How much would you be willing to pay for more frequent updates?

Key point: More than half of pet owners are willing to pay a premium for more frequent updates. This is an opportunity to make clients happier, show them value, and strengthen your bond, while also increasing revenue.

How will you provide more frequent patient updates?

Obviously, more frequent updates provide a better experience for your clients, but how will you incorporate this into your practice? Here is my suggestion:

- Make a sign for your front desk that says, "Would you like more updates during your pet's stay?" and include instructions to download your hospital app.
- Identify pets who are to receive more frequent updates. I use luggage tags (4imprint.com) that can be attached to the pet's collar, kennel door, or treatment sheet that say "Somebody loves me. Like, a lot. Please send them updates about me in the app."
- Create a box for updates on the patient's treatment sheet.
- Send a personalized update with a few details (e.g., Bailey just ate her breakfast, I just changed Yogi's bandage) every 4 to 6 hours.

By keeping your clients in the loop, they are able to see the value in what you are doing for their pet during their hospitalization.

[1.] Kogan, L, Shoenfeld, R, and Santi, S, Medical Updates and Appointment Confirmations: Pet Owners Perception of Current Practices and Preferences, Front Vet Sci 20 March 2019.

THE 5 STEP PHARMACY PROGRAM: SUCCESS TIPS TO MAXIMIZING YOUR PHARMACY REVENUE IN A CHEWY WORLD

Stacee Santi, DVM
Vet2Pet
Durango, Colorado, USA

Refills, it's a problem. Why is it a problem? Let's think about the client experience for refills currently. What happens when a client wants to get a refill of medication or food from you?

Clients have two choices. Most times they can either call the practice and get put on hold for a long time or they can walk into the clinic, which is super inconvenient to the staff. Now, if I'm a client and I happen to be at my lunch break and I walk in because I want to pick up a refill of my pet's medication it is most likely that the receptionist will say something like this to me, “Did you call ahead and make your request?”. This shaming behavior is something we do at the practice to scold our clients. Once the client gets past that and has placed their order, they have to wait for it to get approved. Then the client has to wait for it to get filled. And then pay.

Clients are looking for a quick experience and currently they have to wait a lot when they choose their veterinarian to be the provider of their pet’s medication. Clients are used to Amazon experiences where they want to have a fast, efficient service. Everything at the clinic is a really slow and tedious process. And it's no surprise that online providers like Pet Med Express and Chewy have been able to slide in and improve on this experience.

Step 1: Make ordering easy

Most clients do not want to use their phone to make a phone call. They do not want to be limited to doing business with you only by calling or walking in. It is important to open up other avenues for clients to order medication. One of the most efficient ways to order product or food is from a practice’s mobile app. Clients can quickly place their refill request through the app. And when the incoming order is received, it can be processed. A push notification can be sent back to the client to let them know the order is ready for pickup.

Step 2: Make pickup easy

I have a lot of friends that have kids and babies and they look like a lot of work, especially when you're in the car with them. If this was me, there is no way I would want to come into the clinic to pick up the medication after picking the kids up from school. It would be way easier for me to say no to that scene and go online. For this reason, be sure to have a pickup curb sign and allocate one of the spots in front of your clinic for mobile pickup.

Step 3: Promote your own online store

Many veterinarians have their own online store. That’s fantastic. I can’t think of a single reason not to have your own store. However, many veterinarians refrain from promoting their own online store because of the lower margin than purchasing on-site in the practice. The problem with this strategy is that clients will wind up on Pet Med Express or Chewy and instead of having a lower margin on a chronic medication, the practice will get nothing. It is important to promote your own online store before clients have a chance to find one on their own.

Step 4: Forward refills

The same approach the industry took to forward appointment booking in the early 2000's can be applied in a similar fashion to medication refills. When prescribing a long-term product, before the client leaves, discuss the refill then (before day 59 of the 60-day prescription). Find out how your client would like to have their medication refilled, pick up at practice or shipped directly to home. The client will love this concierge approach and again, no reason for the client to start googling providers since it is already taken care of.

Step 5: Automated refills

Another perk of having your own branded online pharmacy is being able to set clients up for auto-ship for chronic medications. This is such a helpful thing to set up for clients. It's really a "set it and forget it" situation. And, because people take the path of least resistance, having this set up for them will give them one less reason to seek alternative solutions.

In the end, it will be impossible to beat Chewy and Pet Med Express on price but don't forget the power of convenient and personalized service.

The ABCs of ECGs part I

Cardiac Electrophysiology review and case review

Meg M. Sleeper VMD, DACVIM (Cardiology)
Professor of Cardiology; University of Florida School of Veterinary Medicine

Electrical properties of the heart

Keeping the basic electrical properties of the heart in mind while reviewing electrocardiograms greatly simplifies the interpretation process. Knowing the principles of cardiac cell behavior makes interpreting a complex ECG a process like solving a puzzle. pThese principles are:

A. **Automaticity**-only pacemaker cells are capable of beating spontaneously
B. **Excitability**- all resting myocytes maintain a transmembrane electrical potential and are capable of responding to an effective stimulus by generating an all or nothing action potential.
C. **Refractoriness**- all cardiac myocytes undergo a period of recovery following excitation when cells are incapable of responding to stimuli of any magnitude. Excitability is gradually restored. This period makes it impossible to tetanize cardiac muscle.
D. **Conductivity**- low impedance junctions (intercalated discs) located in the ends of muscle fibers at the intercellular junctions give the atria and ventricles the property of a functional syncytium. Therefore, if propagation cannot be expedited along the preferential conducting pathway, depolarization can be spread directly (although more slowly) from one cell to the next. Thus, one of the primary causes of widened QRS complexes is ventricular ectopy.
    1. Rate of conduction is dependent on cell size
    2. Conduction is slower at the AV node, which is normally the only electrical continuity between the atria and the ventricles. This slowing allows mechanical atrial systole to catch up with the electrical discharge (and the ventricles to fill before ventricular systole occurs).
E. **Contractility**- Peak tension that can be developed from a specific resting fiber length. The ECG gives no information about contractile function of the heart.

Pacemaker cells are situated along the conduction pathway and are governed by the hierarchy of dominance. This hierarchy is: Sinus node>>internodal atrial tracks>>>AV node>>Bundle of

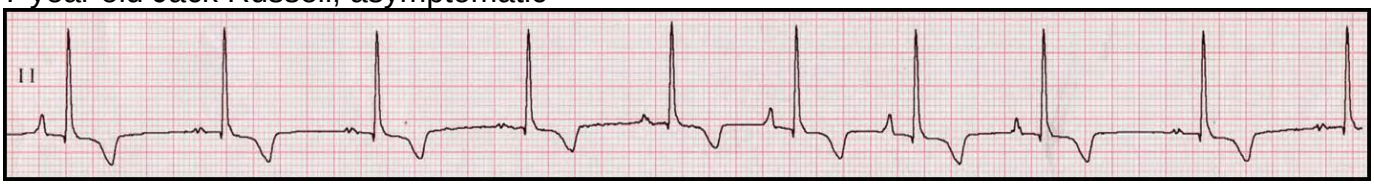
His>>Bundle branches>>Purkinje fibers; all undergo gradual diastolic depolarization during phase 4 (automaticity). Non-pacemaker cells do not normally exhibit automaticity.

Mechanisms of cardiac arrhythmias

Arrhythmias result from abnormalities of impulse generation that alter rate, regularity or origin of excitation and/or change the sequence of atrial and ventricular depolarization due to interference with conduction of the impulse. Below are some example ECGs which will be discussed during this session.

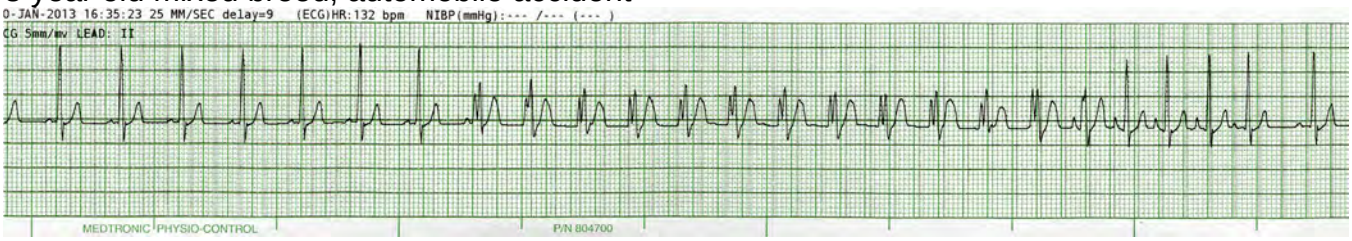
ECG example #1

7 year old Jack Russell; asymptomatic



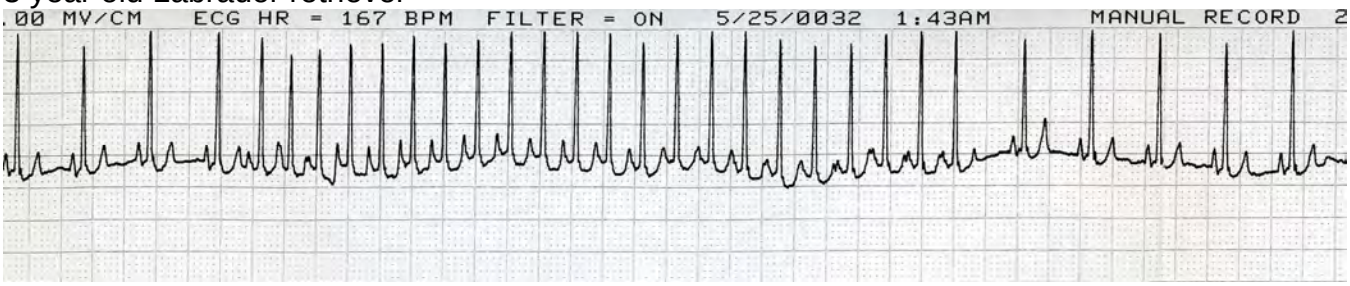
ECG example #2

3 year old mixed breed; automobile accident



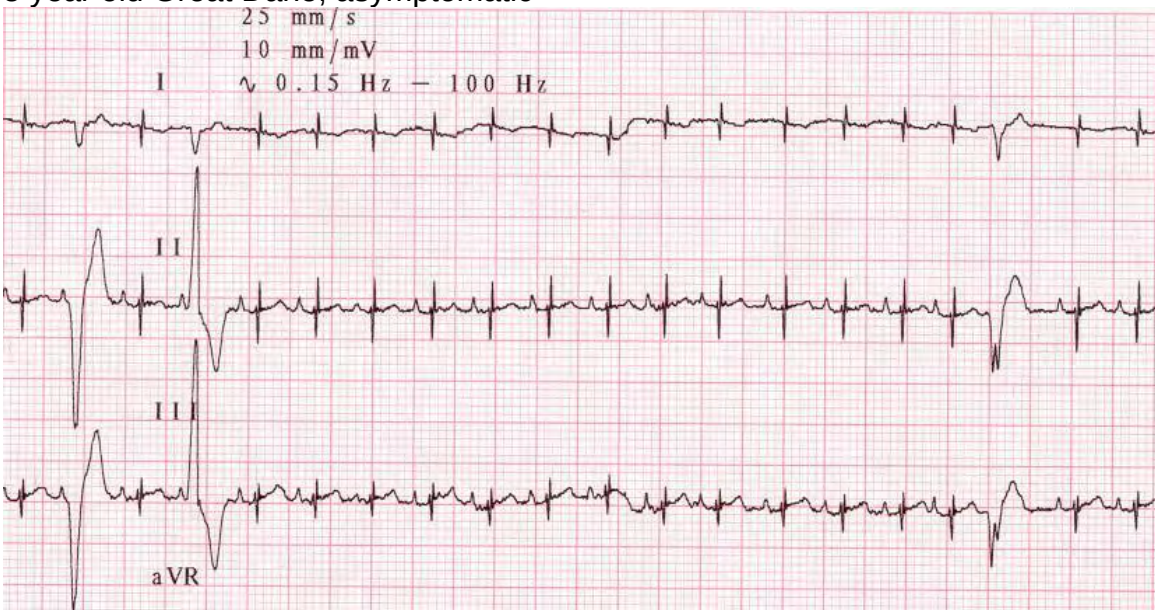
ECG example #3

3 year old Labrador retriever



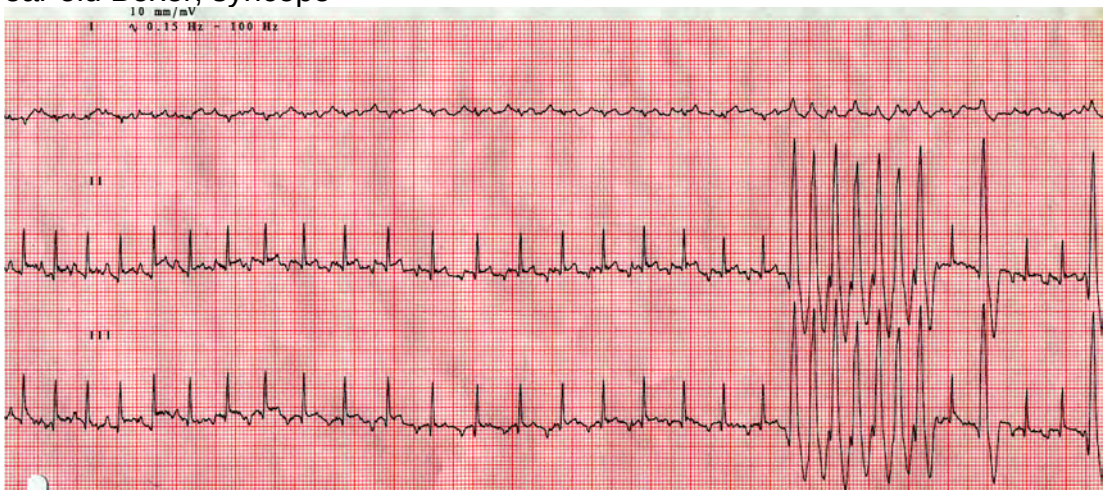
ECG example #4

5 year old Great Dane; asymptomatic



ECG example #5

5 year old Boxer; syncope

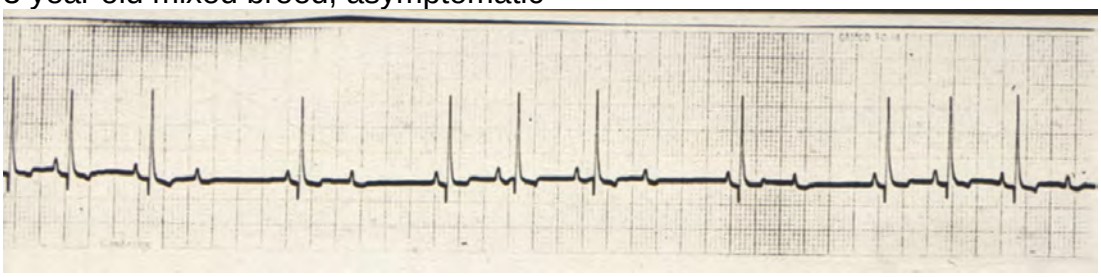


The ABCs of ECGS part II
Case review

Meg Sleeper VMD, DACVIM
Professor of Cardiology, University of Florida School of Veterinary Medicine

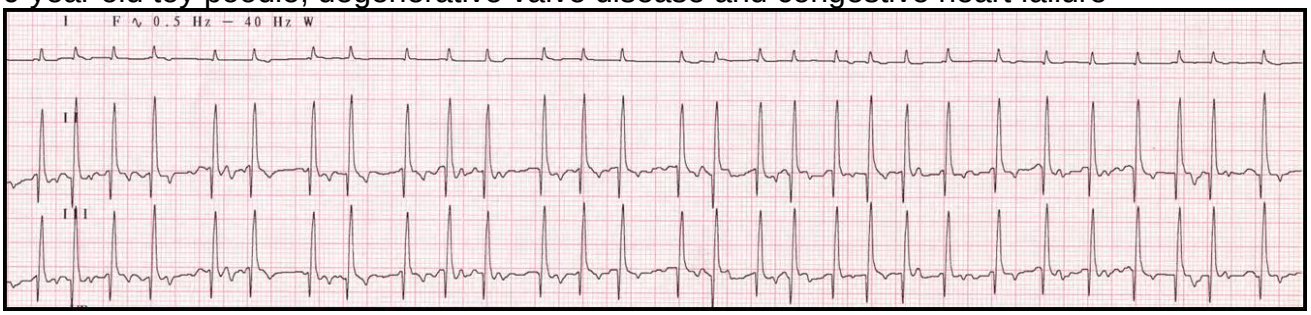
ECG example #6

3 year old mixed breed; asymptomatic



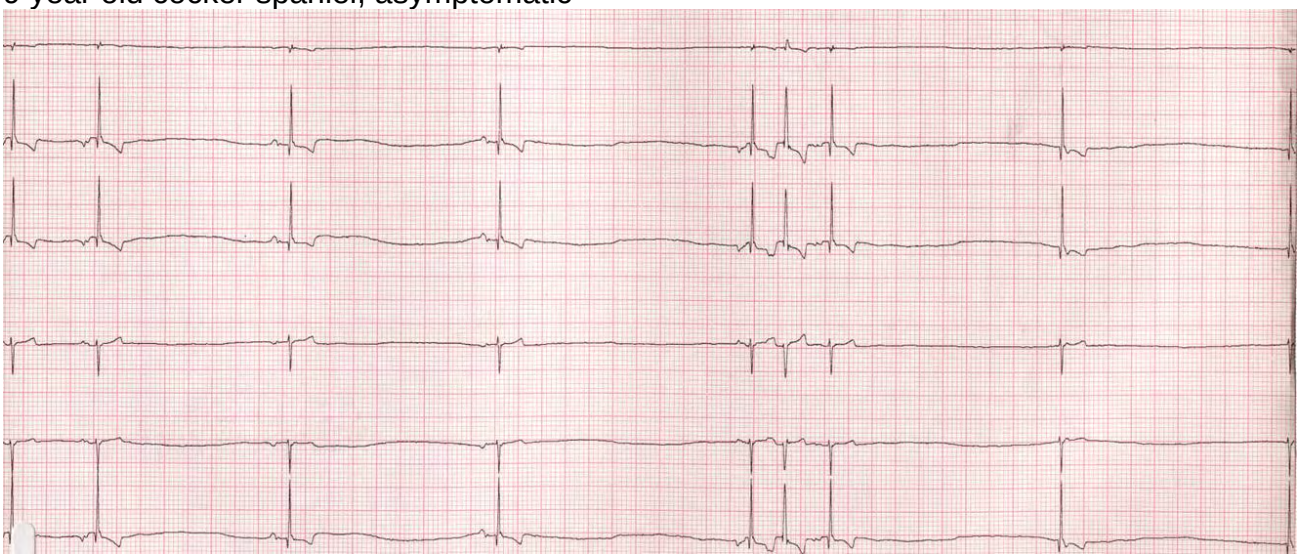
ECG example #7

9 year old toy poodle; degenerative valve disease and congestive heart failure



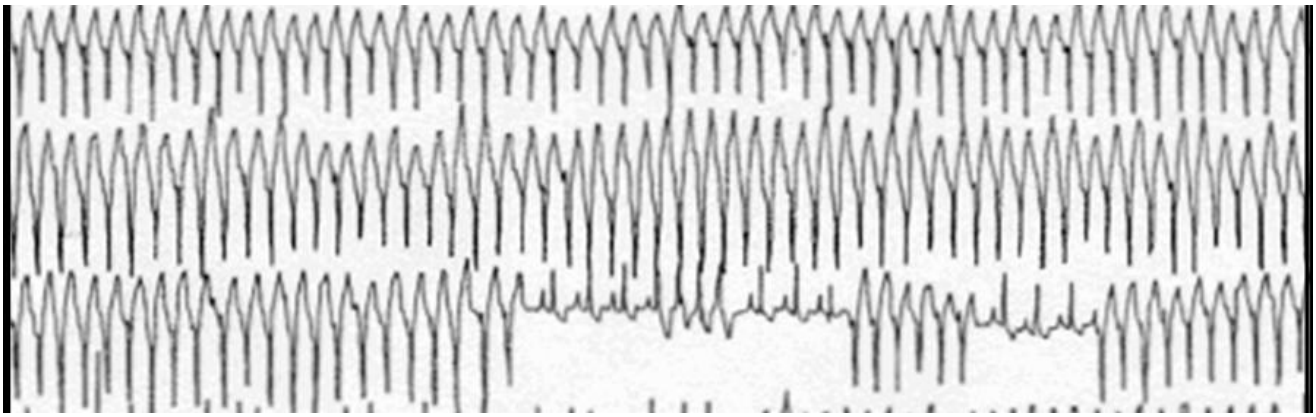
ECG example #8

9 year old cocker spaniel; asymptomatic



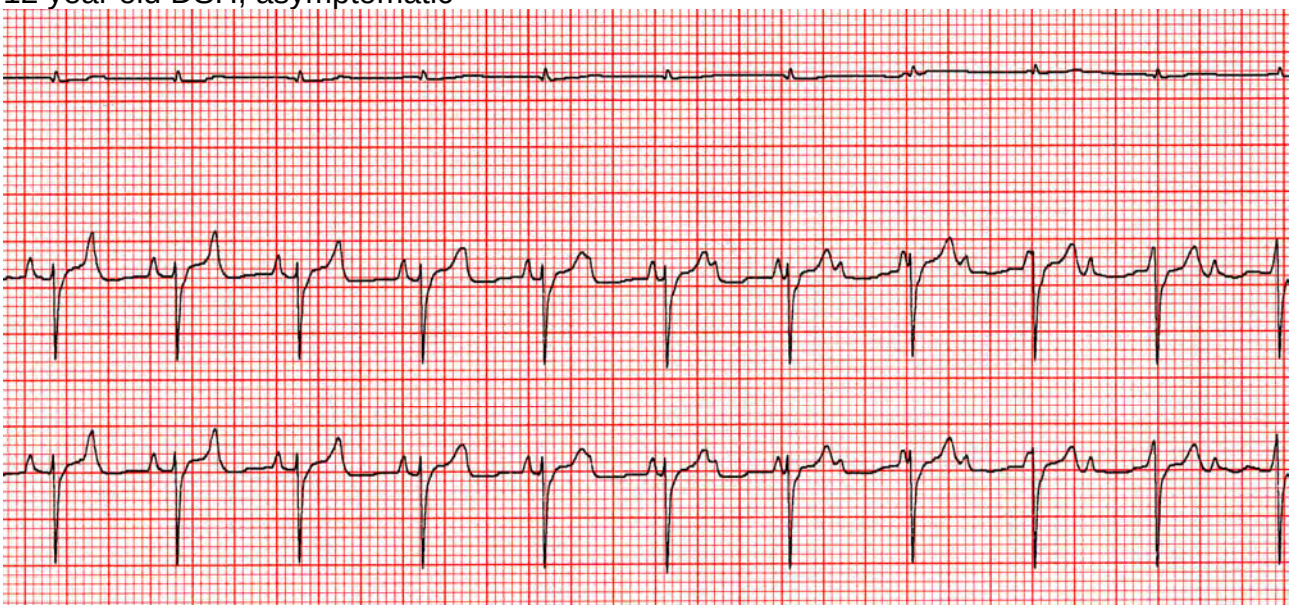
ECG Example 9

8 year old Gordon Setter; collapsed



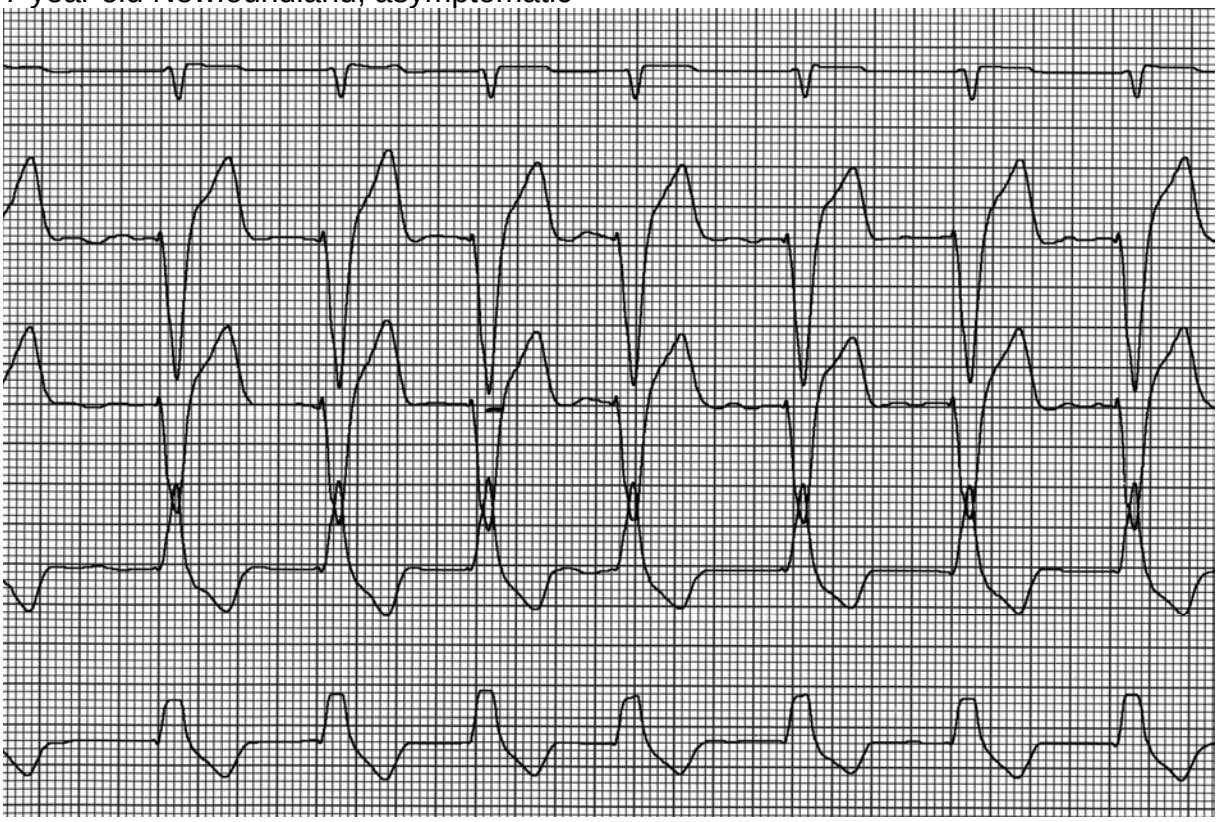
ECG Example 10

12 year old DSH; asymptomatic



Example 11

7 year old Newfoundland; asymptomatic



Drugs for treating supraventricular tachycardia (canine doses):

- Diltiazem
  - 0.5-5mg/kg q 8 hours (oral)
  - 0.1-0.2 mg/kg IV bolus, then 2-6 mcg/kg/min CRI
- Digoxin
  - 0.003-0.005 mg/kg q 12 hours (oral)
- Beta blocker
  - Atenolol: 0.25-2.0 mg/kg q 12-24 hours (oral)
  - Esmolol: 50-100 mcg/kg IV bolus every 5 min up to 500 mcg/kg maximum; 25-200 mcg/kg/min CRI

Drugs for treating bradycardia (canine doses):

- Atropine: 1-4 mg/kg IV, IM
- Propantheline bromide: 0.25-5 mg/kg q 8-12 hours oral
- Terbutaline: 1.25-5 mg/dog oral q 8-12 hours;
- Theophylline: 10 mg/kg q 12 hours oral (extendeded release)

Drugs for treating ventricular tachycardia:

- Canine-
  - lidocaine: 2 mg/kg IV slowly in boluses followed by CRI at 30-80 ug/kg/min CRI
  - procaineamide: 10-30 mg/kg IM PO q 6 hours; 2 mg/kg IV over 3-5 min up to a total dose of 15 mg/kg; 25-50 ug/kg/min CRI
  - mexilitine: 5-8 mg/kg q 8 hours oral
  - sotalol: 1-2 mg/kg q 12 hours oral
  - amiodarone: loading dose of 10 mg/kg PO q 12 hours for 1 week, then 5 mg/kg PO q 12-24 hours
- Feline-
  - lidocaine: 0.25-0.75 mg/kg IV over 5 minutes
  - procaineamide: 2-5 mg/kg PO q 6-8 hours
  - atenolol: 1-2 mg/kg q 12-24 hours oral

Current Recommendations for Treating Dogs with Degenerative Valve Disease

Meg M. Sleeper VMD, DACVIM (Cardiology)
Clinical Professor of Cardiology; University of Florida School of Veterinary Medicine

Degenerative valve disease (DVD) is the most common form of heart disease in the dog. It's common presence in small breed dogs, particularly the cavalier King Charles spaniel, toy and miniature poodle, Bichon frise, etc. suggests a genetic predisposition in some breeds, however a specific genetic mutation has not yet been identified. The ACVIM consensus statement offers a simple classification system for dogs with acquired valve disease that is helpful for prognostic and therapeutic planning.

Stage A- dog is not clinically affected with valve disease, but is of a breed which is at risk for later valve disease development (i.e cavalier King Charles spaniel)
Stage B- valve disease is present, but no evidence of congestive heart failure
Stage B1- no cardiac remodeling (the heart size is normal)
Stage B2- cardiac remodeling (heart size is increased)
Stage C- current or historic congestive heart failure
Stage D- refractory heart failure

Stage A dogs are clinically normal, but because of breed predisposition, may develop heart disease in the future. For owners of these dogs it is useful to discuss signs associated with heart disease and depending on their plans, discussion regarding breeding decisions may be warranted.

Stage B dogs by definition have a cardiac murmur. For staging purposes, thoracic radiographs, systemic blood pressure and a minimum database (creat, urine SG, PCV/TS) is recommended. If the heart size is normal on thoracic radiographs, the dog is considered to be in stage B1 (no cardiac remodeling). No cardiac medications have been shown to alter the progression of disease at this stage of disease. However, regular monitoring for cardiac enlargement with thoracic radiographs is recommended every 12 months. If heart enlargement is detected, the dog is considered to be in stage B2. The EPIC trial results demonstrated that pimobendan therapy resulted in delayed development of congestive heart failure when B2 dogs were started on pimobendan. Based on that trial, B2 criteria included an LA/AO of >1.6 and an LVIDD normalized to body weight > 1.7. Additional ancillary recommendations for B2 dogs include: supplementation with an N-3 fatty acid supplement and counseling the owner to keep a log of the dog's resting or sleeping respiratory rate.

Note that an echocardiogram is not necessarily needed in a dog with a signalment and findings highly likely to be degenerative valve disease (for example a small breed dog with a left apical systolic murmur). However, an echocardiogram is recommended in cases that "do not follow the book". For example, medium size dogs, which may have degenerative valve disease (DVD) or dilated cardiomyopathy (DCM); dogs in which the murmur is loudest on the right; dogs with an unusual pattern of cardiac enlargement.

Stage C dogs are dogs that have or have had congestive heart failure due to degenerative valve disease. In the acute stage, treatment of symptomatic congestive heart failure is similar whether the underlying cause is DVD or DCM. Life-threatening pulmonary edema is most effectively treated with intravenous furosemide. Depending on severity of clinical signs, 1-2 mg/kg is administered every 1-2 hours until there is a 25% reduction in respiratory rate, at which time frequency of dosing can be decreased. A constant rate infusion of furosemide (0.1-1.0 mg/kg/hr IV) appears to be superior to bolusing furosemide in severely affected patients. Dogs should be placed in an oxygen rich environment (oxygen cage). As soon as the dog can safely be given oral medications, pimobendan should be administered (unless the medications have already been administered). Pimobendan is orally available within 1 hour of administration. Depending on the response to oxygen therapy, furosemide and pimobendan, additional therapy may be beneficial. Nitroprusside is an afterload reducer, which can be titrated to effect. Hydralazine is another afterload reducer, however it can result is reflex tachycardias. The addition of a positive inotropic agent such as dobutamine can also be helpful, particularly in dogs with profound myocardial failure. In some dogs, mild sedation with butorphanol is helpful.

Chronic maintenance therapy for dogs with congestive heart failure consists of sufficient furosemide to control congestion, an angiotensin converting enzyme inhibitor (ACEi) to reduce afterload and renin angiotensin aldosterone activity, and pimobendan. It is important to use the lowest dose of furosemide that controls congestion; in most dogs 1-2 mg/kg 2-3 times a day is adequate. If a higher dose is necessary, additional or alternative diuretics should be considered ("triple diuretic therapy" with the addition of hydrochlorothiazide and spironolactone or torsemide).

The most commonly used ACEI in veterinary medicine in the United States is enalapril (0.5 mg/kg twice daily), however benazepril also appears to be a good option in the dog. The benefit of ACEI therapy is conveyed over time and therefore the drug is not necessarily initiated during the initial CHF event, but once the dog is clinically stabilized. ACEI can impact the glomerular filtration rate because of dilatory effects on the efferent renal artery. Therefore it is important to evaluate the animal for azotemia within 1-2 weeks of starting this class of medications.

Pimobendan is a benzimidazole-pyridazinone inodilator (drug causing increased contractility and vasodilation). Venodilation and arteriodilation are via inhibition of PDEIII. Increased contractility is secondary to PDEIII inhibition effects and from a calcium sensitizing effect. Calcium sensitizers affect the interaction of calcium and the troponin C complex and therefore increase the extent of contraction for a given amount of intracellular calcium. This mechanism appears to have advantages because myocardial energy expenditure is lower than with positive inotropic agents operating via the cAMP pathway. In human studies, pimobendan may actually reduce myocardial oxygen consumption. Pimobendan also has favorable effects on left ventricular relaxation and end-diastolic pressure-volume relations. In small animal models, it has been shown to inhibit pro-inflammatory cytokines. At this time, the drug is approved only for use in dogs that have already developed congestive heart failure although one study showed early use in Dobermans with occult DCM delayed onset of heart failure. A study is currently underway to evaluate if pimobendan is beneficial in dogs with DVD prior to congestive heart failure (prior to stage C of disease).

For those patients with refractory heart failure (**Stage D**), additional medications may be beneficial in certain cases. For example, further reduction of afterload with amlodipine or heart rate control with digoxin and/or diltiazem may be warranted. Generally "triple diuretic therapy" (the combination of furosemide, hydrochlorothiazide and spironolactone) and/or torsemide is reserved for this stage of heart failure.

The drugs available to improve cardiac output work on the following formula:

$$\text{Cardiac output} = \frac{\text{Preload X Contractility}}{\text{Afterload}} \text{ X Heart rate}$$

Although reducing preload results in a reduction in cardiac output, because the Starling curve is flattened in patients with heart failure, a reduction in preload will optimally decrease filling pressure to alleviate pulmonary edema with minimal impact on cardiac output. The diuretics are the primary preload reducers with furosemide being the one most commonly used. Nitrates, such as nitroglycerine, are also preload reducers.

Drugs that decrease afterload, arterial dilators, result in improved cardiac output. Pimobendan is a vasodilator and one of its beneficial effects is a reduction in afterload. The ACEi agents are also vasodilators. Additional arterial dilators include amlodipine and hydralazine.

The only effective drug for chronic use that improves contractility is pimobendan. Although digoxin was historically considered a positive inotropic agent, the effect is trivial and it should not be chosen over pimobendan for inotropic support. In the acute heart failure setting, sympathomimetic agents, most commonly dobutamine, are often used for inotropic support. This synthetic catecholamine is rapidly metabolized and must be administered as a constant rate infusion.

When the heart rate is either very elevated or very slow, it will negatively impact cardiac output. In dogs with symptomatic valve disease, atrial fibrillation is the most common tachycardia that requires medical management. There are three main classes of drugs that can be considered for heart rate control in dogs with supraventricular tachycardias, such as atrial fibrillation: Beta adrenergic blockers, Calcium channel blockers and Digoxin. Beta blockers should be administered cautiously in dogs with poor myocardial (or uncertain) myocardial function as they reduce contractility. For this reason, beta adrenergic blockers should never be administered to patient in overt heart failure. Digoxin is a very effective negative chronotrope for chronic use, but the risk of toxicity with intravenous administration is so high that this administration route is not recommended. In the emergency setting, intravenous diltiazem can be used to control rapid supraventricular tachycardias.

References available upon request.

Dilated cardiomyopathy and current recommendations for therapy

Meg M. Sleeper VMD, DACVIM (Cardiology)
Professor of Cardiology; University of Florida School of Veterinary Medicine

Idiopathic dilated cardiomyopathy (IDCM) is characterized by chamber enlargement and markedly decreased contractile function. It is the second most common form of acquired heart disease in the dog. Diagnosis is typically made by thoracic radiography, electrocardiography, and echocardiography. In various retrospective studies of canine DCM and congestive heart failure, age of onset is usually middle to old age and large breed dogs, in particular the Doberman pinscher, great Dane, Newfoundland and Irish wolfhound are overrepresented. Mutations have been identified in the Doberman pinscher and Boxer breeds, however dogs without the mutations can also be affected with DCM. An inherited cause of DCM has also been confirmed in the Portuguese water dog breed. In this breed, DCM is early onset (juvenile) and inherited as an autosomal recessive trait.

Typical clinical signs exhibited in dogs presenting with DCM are similar in all breeds, however the frequency of these signs is strikingly different in many of the commonly affected breeds. Doberman pinschers and Boxer dogs usually present for signs associated with left sided congestive heart failure or for signs associated with ventricular arrhythmias. However, sudden death is the first sign of heart disease in a subset of affected dogs. In other breeds, such as the Newfoundland or great Dane, arrhythmias and/or sudden death are much less likely to occur and clinical signs in general are less severe. Atrial fibrillation occurs most frequently in the giant breed dogs.

In two situations, the treating clinician can realistically hope to improve systolic dysfunction:

1. persistent tachycardia (tachycardia induced cardiomyopathy)
2. nutritional deficiency (nutritional cardiomyopathy)

In both of these situations, the clinician must treat heart disease with traditional medical therapy while also addressing the underlying problem. Nutritional cardiomyopathy will be further discussed later.

Life-threatening pulmonary edema is most effectively treated with intravenous furosemide +/- nitroprusside or hydralazine. A constant rate infusion of furosemide (0.1-1.0 mg/kg/hr IV) appears to be superior to bolusing the drug in severely affected patients. Nitroprusside is an afterload reducer which can be titrated to effect. Hydralazine is another afterload reducer, however it can result is reflex tachycardias. The addition of a positive inotropic agent such as pimobendan and/or dobutamine can also be helpful, particularly in dogs with profound myocardial failure.

Chronic maintenance therapy for dogs with congestive heart failure due to DCM consists of sufficient furosemide to control congestion, an ACE inhibitor to reduce afterload and renin angiotensin aldosterone activity, and pimobendan (a positive inotrope and vasodilator). Digoxin is an inexpensive, positive inotrope which has the added benefit of heart rate control. However, it is a very weak positive inotrope and its primary use in small animal cardiology is for supraventricular tachycardias such as atrial fibrillation.

Pimobendan is a benzimidazole-pyridazinone inodilator (drug causing increased contractility and vasodilation). Venodilation and arteriodilation are via inhibition of PDEIII. Increased contractility is secondary to PDEIII inhibition effects and from a calcium sensitizing effect. Calcium sensitizers affect the interaction of calcium and the troponin C complex and therefore increase the extent of contraction for a given amount of intracellular calcium. This mechanism appears to have advantages because myocardial energy expenditure is lower than with positive inotropic agents operating via the cAMP pathway. In human studies, pimobendan may actually reduce myocardial oxygen consumption. Pimobendan also has favorable effects on left ventricular relaxation and end-diastolic pressure-volume relations. In small animal models, it has been shown to inhibit pro-inflammatory cytokines.

Several studies have demonstrated improvement in congestive heart failure class when pimobendan therapy is used in dogs with DCM. In addition to being beneficial in dogs with secondary congestive heart failure (CHF), the Protect trial demonstrated there is benefit to starting dogs with dilated cardiomyopathy on pimobendan prior to the development of overt clinical signs.

Dietary-associated DCM first was recognized in cats in the late 1980s and then in dogs in the mid-1990s. Recently, a dietary associated DCM has been linked to some grain free diets and the disease was found to improve with nutritional management, including diet change. This association was not recognized with all grain free foods, and therefore there are likely factors other than simply the omission of grain that are important. The predominant legumes in these grain free diets are peas or lentils. In at least one study, none of the affected dogs had reduced taurine blood levels and therefore the role of taurine remains uncertain, and the cause of this presumed diet-related DCM is not known. Therefore, there are currently 2 groups of dogs with diet associated DCM: those related to taurine deficiency and those dogs with DCM secondary to separate, but yet unknown dietary factors.

REFERENCES

Available upon request

Update on Managing Feline Heart Disease

Meg Sleeper VMD, DACVIM (cardiology)
Professor of Cardiology; University of Florida Veterinary School

Hypertrophic cardiomyopathy (HCM) is the most common heart disease in cats. Fifteen to thirty-four percent of healthy cats have echocardiographic evidence of left ventricular concentric hypertrophy (increased ventricular wall thickness) attributed to HCM. HCM is caused by a primary defect within the heart muscle cells, which causes the ventricle to become thick (i.e., concentric hypertrophy) and develop excess scar tissue (i.e., fibrosis), which makes the ventricle stiff and unable to relax normally. HCM is inherited in Maine coon cats and Ragdoll cats as an autosomal dominant trait with incomplete penetrance. This means that HCM affects all individuals that have the mutation, but to varying degrees. HCM ranges in severity from mild to severe disease. Pathophysiologic sequelae to severe HCM may be development of diastolic congestive heart failure (i.e., pulmonary edema and/or pleural effusion), arterial thromboembolism, or sudden death.

The cardinal pathophysiologic characteristic of hypertrophic cardiomyopathy is impaired diastolic filling of the left ventricle, due to abnormal relaxation of the heart muscle and increased ventricular muscle stiffness. Diastole is comprised of active isovolumic relaxation, rapid passive filling, diastasis, and atrial systolic filling. Cats with HCM have abnormal relaxation and increased stiffness that impairs passive ventricular filling. Impaired relaxation is caused by abnormal calcium handling, increased myofilament sensitivity to calcium, intracytosolic calcium overload, altered left ventricular loading conditions, and myocardial ischemia from small coronary artery disease. Increased ventricular stiffness is caused by concentric left ventricular hypertrophy, myofiber disarray, and myocardial fibrosis. Delayed relaxation and increased ventricular stiffness increase diastolic filling pressure, which may lead to development of left heart failure. Pulmonary edema and/or pleural effusion develop as the main manifestations of left-sided congestive heart failure in cats with HCM. Systolic anterior motion (SAM) of the mitral valve develops secondary to anterior-ventrally displaced, hypertrophied papillary muscles that pull the mitral valve into the left ventricular outflow tract during systole.

Other factors that may exacerbate or worsen SAM of the mitral valve include severe basilar septal concentric hypertrophy, increased contractility, and tachycardia.

Moderate or severe SAM of the mitral valve greatly increases left ventricular systolic pressure, which increases severity of concentric LV hypertrophy and potentiating the vicious cycle of hypertrophy and the potential for worsened diastolic function. Arterial thromboembolism may occur in cats with left atrial enlargement. Factors involved in development of a left atrial thrombus include blood stasis, possible endothelial disruption, and a possible procoagulable state with increased platelet aggregation and coagulopathic markers.

Restrictive cardiomyopathy (RCM) is less common that HCM, but also primarily causes diastolic dysfunction. Dilated cardiomyopathy, now a rare cause of heart disease in the cat, is the only cardiomyopathy causes systolic dysfunction (although often there is also diastolic dysfunction). Differentiation of the form of cardiomyopathy requires an echocardiogram, which demonstrates increased left ventricular or interventricular septal end-diastolic wall thickness (with HCM), normal wall thickness with a restrictive filling pattern (with RCM) or thinner than normal wall thickness and reduced contractility (with DCM). With any of the cardiomyopathies left atrial dilation may be present. Congestive heart failure is diagnosed by clinical signs and thoracic radiographic evidence of cardiomegaly, pulmonary edema and/or pleural effusion, and often pulmonary venous distension may also be present with any of them.

Electrocardiography is an insensitive test to screen for HCM, but it is important in cats with an arrhythmia or history of episodic weakness or collapse. Hyperthyroidism, systemic hypertension, subaortic stenosis, and acromegaly are differential diagnoses for increased left ventricular wall thickness seen with HCM. Basic comprehensive blood work including complete blood count, serum chemistry, total thyroxine level, and urinalysis is recommended in cats diagnosed with HCM, especially in middle-aged to older cats, or in cats with heart failure. Baseline renal function is important to assess prior to medical treatment of heart failure. Significant anemia should be identified, as this may significantly worsen ventricular volume overload (i.e., preload) in cats with HCM. Total thyroxine concentration is an essential diagnostic test in middle-aged to older cats with echocardiographic evidence of concentric hypertrophy, because hyperthyroidism is a secondary cause of concentric hypertrophy. Serum growth hormone levels may be mildly increased in cats with HCM, but they are much higher in cats with acromegaly. Insulin-like growth factor-1 $\pm$ serum growth hormone should be measured in cats with concentric hypertrophy of the left ventricle and clinical abnormalities suggestive of acromegaly.

Treatment of cats with HCM is dependent on many variables, which may include presence of left atrial dilation, severity of systolic anterior motion of the mitral valve, tachycardia, severity of left ventricular hypertrophy, client and patient motivation and ability to chronically administer medications. Treatment options for asymptomatic cats include atenolol, or less preferably diltiazem. If elected, atenolol is the treatment of choice for moderate or severe systolic anterior motion of the mitral valve (left ventricular to aortic pressure gradient measured on echocardiography of **≥**50 mm Hg), in the appropriate clinical context.

There is significant controversy regarding when to initiate treatment, and what is the most appropriate medical therapy in asymptomatic cats with HCM and no heart failure. Beta blockers (i.e., atenolol) or calcium channel blockers (i.e., diltiazem) are the most commonly used drugs in asymptomatic cats with HCM, and they may reduce severity of hypertrophy. Beta blockers are more effective than calcium channel blockers to reduce severity of systolic anterior motion of the mitral valve and control heart rate to prevent tachycardia. Clinical evidence from placebo controlled, blinded clinical studies indicate that early use of angiotensin converting enzyme inhibitors or aldosterone antagonists in cats with asymptomatic HCM and no heart failure is not warranted. Prophylactic anticoagulant therapy is indicated if there is echocardiographic evidence of spontaneous contrast (i.e., red blood cell aggregation) or an intracardiac thrombus. It is controversial whether cats with significant left atrial dilation should be placed on prophylactic anticoagulation. Prophylactic anticoagulant therapy is not necessary in cats with mild HCM and normal left atrial size, except in the rare incidence where such a cat has a history of confirmed ATE.

Congestive heart failure is treated with furosemide and an ACE inhibitor. Anticoagulant therapy with clopidogrel, aspirin, or low molecular weight heparin is necessary in cats currently or previously suffering from arterial thromboembolism, cats with echocardiographic evidence of spontaneous contrast or thrombus, and may be considered in cats with moderate to severe left atrial dilation (left atrial to aortic ratio of **>**1.9 and or evidence of atrial blood stasis). Prognosis of mild and/or asymptomatic HCM is good, and cats may live for many years without problems. The prognosis worsens once cats develop congestive heart failure, with average survival times ranging from 92–654 days. Cats with HCM and ATE have the poorest prognosis, with average survival times ranging from 61–184 days.

Furosemide is the most effective and life-saving treatment of cats in congestive

heart failure and can be given at a dose range of 1–2 mg/kg PO q 24 hr to TID for outpatient therapy depending on the severity of heart failure. The minimal effective dose should be used. An initial moderate to high dose may be started and then rapidly tapered based on respiratory rate, effort, and evaluation of severity of heart failure by thoracic radiographs. Treatment of acute heart failure includes parenteral furosemide (1–2 mg/kg q 1–8 hr). The dose and frequency should be rapidly tapered once the respiratory rate decreases to 50 bpm or less and the effort is decreased. Oxygen therapy with 60–70% FiO2 can be done initially and then decreased to 50% or less within 12 hours to avoid further barotrauma secondary to high inspiratory oxygen concentration. Transdermal nitroglycerin causes venodilation in people, but it has not been evaluated in cats. Its use is debatable in cats and can be given short-term to hospitalized cats for no longer than 2 days; the main drawback appears to be a lack of efficacy rather than adverse effects. Negative inotropic therapy including beta blockers and calcium channel blockers may be used in some cats with chronic heart failure but are not given in acute heart failure unless there is a hemodynamically significant tachyarrhythmia. ACE inhibitors such as enalapril or benazepril may help provide adjunctive treatment in cats with heart failure. Rather than starting as an emergency in-hospital treatment, the ACE inhibitor may be started at home once the cat is stabilized, eating and drinking, and hydrated. Prophylactic anticoagulation may be started in cats with high risk of arterial thromboembolism, including those with spontaneous echocardiographic contrast, left atrial thrombus, history of prior ATE, or severe left atrial dilation. Congestive heart failure, arterial thromboembolism, and sudden cardiac death are the most common and devastating clinical sequelae in cats with cardiomyopathy. Monitoring of asymptomatic cats with HCM includes repeat echocardiograms every 4–12 months depending on the severity and progression of the disease. To evaluate for early heart failure, radiographs should be obtained periodically (every 3–6 months) in cats with significant left atrial dilation. Monitoring of cats with HCM and heart failure includes repeat thoracic radiographs and renal panels to assess clinical response and guide medical therapy. Thoracic ultrasound can be done to evaluate severity of pleural effusion and guide location for thoracocentesis. Systolic blood pressure and thyroxine levels should be periodically evaluated in middle-aged to older cats that are at risk for developing systemic hypertension or hyperthyroidism.

Rabbit Diseases – Practical Diagnostic and Treatment Options

Cheryl B. Greenacre, DVM, DABVP-Avian, DABVP-Exotic Companion Mammal,
Professor and Section Head, Avian and Exotic Animal Medicine
Department of Small Animal Clinical Sciences, College of Veterinary Medicine,
University of Tennessee, 2407 River Drive, C-247, Knoxville, TN 37996

Introduction

Most of our general medicine principles for large and small animals apply to rabbits, with a few exceptions usually due to size, anatomy, and physiology. Medical information available on rabbits can be found in veterinary companion animal venues and also in the laboratory animal literature. Below is a compilation of pertinent information from the available literature for practical use in the companion rabbit.

Literature available

Books
-*Ferret, Rabbits and Rodents, Clinical Medicine and Surgery*, 4th edition, Elsevier, 2020, Quesenberry KE, Mans C, Orcutt C. (Affectionately known as the “pink” book)
-*Textbook of Rabbit Medicine*, 2nd edition, Butterworth-Heinemann, 2013, Varga M.
-*Exotic Pet Behavior, Birds, Reptiles, and Small Mammals*, Saunders, 2006, Bays TB, Lightfoot T, Mayer J.
-*Rabbit and Rodent Dentistry Handbook*, Zoological Education Network, 2005, Capello V, Gracis M, Lennox AM.
-*Radiology of Rodents, Rabbits, and Ferrets, An Atlas of Normal Anatomy and Positioning*, Saunders, 2005, Silverman S, Tell LA.
-*A Colour Atlas of Anatomy of Small Laboratory Animals, Volume One: Rabbit, Guinea Pig*, Saunders, 1990, Popesko P, Rajtova V, Horak J.
-*Exotic Animal Medicine for the Veterinary Technician*, 2nd ed., Iowa State Press, 2003, Ballard B, Cheek R.
-*Exotic Animal Formulary*, 5th edition, Elsevier, 2018, Carpenter JW.
-Journal of Exotic Pet Medicine
-Conference Proceedings (VMX, AEMV, ExoticsCon, etc.)
-House Rabbit Society, www.rabbit.org
-American Rabbit Breeders Association, arba.net
-Veterinary Information Network, www.arba.net
-Association of Exotic Animal Veterinarians (AEMV)

INFECTIOUS DISEASES

Parasitic

Ear Mites
Rabbit ear mites are due to *Psorptes cuniculi*. Clinical signs include a dry, brownish crust in the ears, usually bilaterally, and scratching and shaking of the ears. Diagnosis is based on visualizing the mite under the microscope from an ear crust sample. Treatment is topical (in the ears usually) ivermectin at 200 micrograms/kg, and repeat in 10-14 days, or one dose of selamectin. There is no reason to clean the crusts out of the ears as tempting as it may be. The crusts will fall out in about 5 days and with a lot less trauma than if you try to clean them manually.

Fur Mites
Rabbit fur mites are due to *Cheyletiella parsitovorax* also known as the “walking dandruff mite” in people. It is a somewhat benign zoonotic parasite. Clinical signs include alopecia and pruritis over the dorsum. Diagnosis is based on identifying the mite under the microscope from a tape prep of the affected area. Treatment consist sof topical, oral, or SC ivermectin at 200 micrograms/kg and repeat in 10-14 days, or one dose of selamectin.

Intestinal coccidiosis
Coccidiosis, caused by *Emeria* spp. (more than 12 sp. identified), is a common cause of diarrhea in young rabbits. Diagnosis is based on a fecal float. Treatment consists of sulfadimethoxine or sulfaquinoxaline. Sulfaquinoxaline has a 10 day meat withdrawal. Use of equine ponazuril (US) and toltrazuril (Europe) has been described. Intestinal coccidiosis should not be confused with the less common, but much more severe, hepatic coccidiosis caused by *Eimeria stiedae* that attacks the bile duct lining. Clinical signs include unthriftiness, diarrhea and abdominal distension. Response to treatment is less successful than treatment against intestinal coccidiosis.

Encephalitozoonosis
Encephalitozoonosis is caused by the coccidian parasite *Encephalitozooan cuniculi*. Rabbits are exposed via urine from the mother at one day of age (or in utero) and infection is very common (~80% of rabbits have been exposed). Clinical signs are not common, but when they occur can either manifest as an anterior uveitis (involving the lens) or as a neurological disease including paresis, paralysis, incontinence, ataxia, or torticollis. Diagnosis is difficult because the antibody titer is almost always positive and a rising titer may not necessarily suggest clinical disease. A highl C-reactive protein level is suggestive of active disease. Vestibular pasteurellosis is a primary differential of torticollis. Fenbendazole or albendazole has been used to treat rabbits with *E. cuniculi*, but may cause a pancytopenia/anemia therefore weekly CBC’s are imperative to stop the treatment before life threatening anemia occurs. Treatment is long term, lasting a least 2-4 weeks past normalization of clinical signs. Associated opaque lesions in the anterior chamber of rabbits (phacoclastic uveitis) can be removed via phacoemulsification.

***Hymenolepis nana* (Dwarf tapeworm) in rodents**

Clinical signs of dwarf tapeworm include no signs or varying degrees of diarrhea and dehydration in rabbits, rodents, and other animals. Diagnosis is via a fecal float and *H. nana* may be difficult to distinguish from other tapeworm eggs such as *H. diminuta*. Treatment includes fluids and praziquantel. *H. nana*, also known as the dwarf tapeworm, is zoonotic and therefore the public health department should be notified. The grain beetle is the most common intermediate host of *H. nana*. Appropriate treatment can clear the infection in animals treated promptly, but death can occur in some animals if not treated.
The highly contagious dwarf tapeworm (<2 in.) has a worldwide distribution and is common in children, institutional settings, and areas of poor sanitation. Adult humans commonly have no clinical signs, but those with a heavy infection or young children exhibit diarrhea, nausea, weakness, loss of appetite, and abdominal pain. For more information visit the following CDC website: https://www.cdc.gov/dpdx/hymenolepiasis/index.html

Bacterial

Rabbit syphilis

Rabbit syphilis is caused by *Treponema* sp.. Clinical signs include erosions and scabs of the skin around the perineal area, nose, mouth, and sometimes even the ears. Diagnosis is based on identification of the organism on a direct saline smear seem under the microscope from an affected area, but are difficult to find. Sometimes response to treatment can be used as a diagnostic tool. Treatment consists of penicillin procaine G with benzathine SC every 7 days for 2-3 treatments. Do not give penicillin orally or a fatal diarrhea may occur. This is a sexually transmitted disease so treat all rabbits that have direct contact.

Pasteurellosis

Pasteurellosis is a very common disease of rabbits and manifests in many different tissues. The causative organism is *Pasteurella multocida*. Clinical signs include upper or lower respiratory signs, neurological signs such as vestibular disease, or abscesses in skin or soft tissue. Although almost any bacterial infection in rabbits is blamed on *Pasteurella* sp., cultures and PCR tests have shown that only about 40% of abscesses are due to *Pasteurella* sp. Diagnosis is based on culture/sensitivity of abscess lining and/or PCR of a nasal swab. A CBC may show a leukocytosis or heterophilia, but surprisingly, abscesses in rabbits do not necessarily raise the WBC. Treatment is based on culture and sensitivity, but until the results return, enrofloxacin is recommended if pasteurellosis is suspected. Remember that pasteurellosis can only be treated, not cured. It may recur during times of stress. A CT scan is best for detecting abscesses in the tympanic bulla and if present may be treated with a lateral bulla osteotomy.

Abscess control

Abscesses must be removed in total or marsupialized as rabbit pus is thick and will not drain due to heterophils lacking the enzyme myeloperoxidase. Antibiotic impregnated polymethylmethacrylate beads can be placed in areas of infection, such as an abscess, after thorough cleaning and debriding, The placement of beads can be delayed until the tissue has undergone some second intention healing before using first intention healing methods when closing beads into the tissue. Antibiotic elutes from the beads into the nearby tissue and is effective for 2 to 3 mm around each bead.[3,17,18] Approximately 5% of the antibiotic present is released into nearby tissue in the first 24 hours, but systemic levels are low. The exact point at which no further elution occurs is not known, but beads impregnated with gentamycin had completed eluted by 37 days after placement into tissue. Many factors affect the rate of elution including bacterial or debris contamination at the site, blood supply to nearby tissue, the number of beads placed, the amount of antibiotic in the bead, the bead's proximity to the affected tissue, and the appropriateness of the antibiotic chosen for the pathogen present. The beads can be removed later or left in place. Antibiotics that work well in beads are those that are heat stabile to withstand steam sterilization and those that come in a powdered form, such as ceftiofur, cephazolin, and gentamycin.

Viral

Rabbit Hemorrhagic Disease

Quick Summary

Rabbit Hemorrhagic Disease (RHD), also known as RHDV-2, is a highly contagious foreign animal disease caused by a calicvirus that is associated with near 100% mortality in both wild (*Sylvilagus* spp.) and domestic (*Oryctolagus cuniculus*) rabbits. Epistaxis and death are the hallmarks of RHD. The disease is now spreading in the US in both the wild and domestic rabbit

populations. A safe and efficacious vaccine to protect rabbits in Europe has existed for years, but is only available in the US on a state-by-state basis. If you suspect RHD immediately contact your state veterinarian (TN State Veterinarian is Dr. Samantha Beatty, (615)837-5120.

History of Rabbit Hemorrhagic Disease

RHD-1 first emerged in China in 1984. The virus then tracked westward from China to eastern and western Europe. It was also identified in Mexico in 1988. In 1989 in Italy, 64 million farmed rabbits had died. RHD-1 was purposefully released in Australia in 1995 to control rabbit populations and within 18 months had reduced the wild rabbit population by 90% in Southern Australia. RHD-1 was recorded in the US 7 times between 2000 and 2010, but no spread ever occurred. Fortunately, a vaccine was developed by 1995 to protect farmed, research, and pet rabbits in Europe.

A new variant of the RHD virus, designated RHDV-2 was identified in France in 2011, the UK in 2015 and Australia in 2017. RHDV-2 has replaced endemic strains of the original RHD virus in Europe and Australia and is now the major field strain. A vaccine was developed against RHDV-2 by 2016 and there are now 2 safe and efficacious vaccines against RHDV-2 used in Europe. As RHD is designated a Foreign Animal Disease in the US, vaccines against RHDV-1 and RHDV-2 are not approved for use in the US.

Current Outbreak of RHDV-2 in the US

The first case of RHDV-2 in the US was identified on a farm in Ohio in 2018 in domestic rabbits. Since 2018, RHDV-2 has been detected in domestic and feral domestic rabbits in British Columbia (Canada), Ohio, Washington, and New York. As of April 13, 2020, RHDV-2 was identified in wild rabbits (*Sylvilagus audubonii* and *Lepus californicus*) in New Mexico and within weeks RHDV2 spread to wild and domestic rabbits in Arizona, California, Colorado, Nevada, Texas, and Utah. It may be spreading along interstate highways since the virus is highly stable and can remain actively infectious on surfaces for >200 days. The loss of wild prey species in these states will have significant ecological impacts on other species that feed on rabbits and also on co-prey species that will see in increase in predation. The native endangered pika will also be greatly affected. (See USDA Factsheet on Rabbit Hemorrhagic Disease at http://www.fs-rhdv2.pdf, or on avma.org or arba.org))

Ecological and Economic Impact

This disease can have a drastic ecological and economic impact on the US. The ecological impact will affect wild rabbits and decrease food resources for the predators who eat them as well as increase the predation of co-prey animals. The economic impact of RHDV-2 will affect all domestic rabbits in their various uses: pets, exhibition, farmed for meat, angora fur, pelts, or breeding, and research. The number of rabbits farmed (approximately 1 million), those bred for use in scientific research (2018, USDA = 14,962), and rabbits kept as pets (about 6 million), that could become infected in turn affects the pet industry, the pet food industry, advances in research, and veterinarians. The economic impact of such s loss in the US is difficult to quantify, but losses in Europe after the arrival of this disease have been devastating. Multiple groups are already addressing concerns of RHDV-2 spread in the US and are involved in the health and welfare of wild and domestic rabbits including the US Fish and Wildlife Service, the American Rabbit Breeder's Association with over 30,000 members, veterinary groups such as the Association of Exotic Mammal Veterinarians (AEMV) and the various laboratory animal groups including the American Society of Laboratory Animal Practitioners (ASLAP), and community and shelter groups such as the House Rabbit Society. Without being allowed to use a vaccine on domestic rabbits, either licensed or unlicensed, millions of domestic rabbits in the US could be affected by RHDV-2 including those that are

Vaccine Use
There are two licensed killed vaccines in Europe that can only be used in the US with special permission from the state veterinarian in the state where given. Most state veterinarians will only allow the importation of the European vaccine to their state in the US if the disease is already in their state. A veterinarian practicing in Washington state (and recently California state) was able, with help from their state and federal veterinarians, to import vaccines from Europe to vaccinate pet rabbits.

Education of Public:
o RHDV-2 is NOT a public health concern
o House rabbits indoors if possible.
o Do not allow pet, feral, or wild rabbits to come in contact with your rabbits or gain entry to the facility or home.
o Always wash your hands with warm soapy water between pens and before and after entering your rabbit area.
o Keep a closed rabbitry. Do not introduce new rabbits from unknown or untrusted sources.
o If you bring new rabbits into your facility or home, keep them separated from your existing rabbits. Use separate equipment for newly acquired or sick rabbits to avoid spreading disease.
o Control flies, rats, cats, dogs, birds, etc. that can physically move the virus around on their feet or body.
o Do not collect outdoor forage and browse to feed rabbits since it may be contaminated.
o Remove brush, grass, weeds, trash, and debris from the rabbitry to reduce rodents.
o Protect feed from contamination by flies, birds, rodents, etc.
o Remove and properly dispose (i.e. bury or incinerate) of dead rabbits promptly.
o When moving rabbits or restocking pens disinfect all equipment and cages with 10% bleach mixed with water or other approved products. Properly dispose of bedding. Items made of wood are difficult to disinfect and best discarded.
o Breeders should review their biosecurity plans for gaps and all rabbit owners should establish a working relationship with a veterinarian to review biosecurity practices for identification and closure of possible gaps.

NON-INFECTIOUS DISEASES

Gastric stasis secondary to some other disease

Gastric stasis can be secondary to pain, liver torsion, dehydration or trichobezoar, also known as “wool block” in rabbits, and is common especially during times of shedding. Other predisposing factors include obesity, low fiber diet (low in hay or greens). It is thought that some hair is normal within the stomach and that clinical signs of a “hairball” occur when decreased cecal/GI motility and decreased GI fluid is present. Clinical signs include anorexia, depression, dehydration, decreased or no fecal production or diarrhea, and in severe cases a toxic gumline as in a horse with severe colic. Elevated liver enzymes/alkaline phosphatase should prompt an immediate ultrasound to check for liver torsion. Treatment consists of rehydrating by both the oral and IV routes, offering fresh greens, commercially available critical care herbivore formula (there is even a formula made now that fits through a nasogastric tube), buprenorphine for pain, a gastric protectant or ranitidine, +/- a feline hairball laxative, +/- metaclopramide/cisapride, and +/- pineapple juice. The papain enzyme in pineapple juice may or may not degrade hair, but it should do no harm, although some say the sugar may upset cecal flora. Oral and parenteral fluids are the most important part of the above treatment. If there is an intestinal blockage with a

small ball of hair or foreign bodydf, then a gastrotomy can be performed secondary to milking the mass to the stomach.

Uterine/ovarian Adenocarcinoma
Intact female rabbits over the age of 5 years have a very high incidence of uterine adenocarcinoma. The Silver Marten breed has an 80% incidence. If clinical signs are present, they include anorexia, depression, hematuria, dysuria or milk production. Diagnosis is based on palpation, radiographs, or ultrasound that suggests an enlarged, possibly fluid filled uterus, sometimes with masses or cystic endometrial hyperplasia. Treatment consists of ovariohysterectomy, but a radiograph should be performed prior to surgery to rule-out metastasis to lung or bone.

Urolithiasis/Urine sludging
Urinolithisis, including renal, ureteral, cystic or urethral calculi can occur in rabbits and guinea pigs. Urine sludging or calcium carbonate sand can occur as well. Urolithiasis is usually associated with a bacterial cystitis. If a diet high in calcium is offered, such as alfalfa pellets and hay, then this may predispose to urolithiasis by accumulation of calcium in the urine, but some individuals can get urolithiasis even on a diet ofTimothy hay based pellets and timothy hay. Diagnosis and treatment is similar as in a dog or cat, including radiographs, urinalysis, urine culture, stone analysis, stone culture, CBC, and chemistry profile. Remember to always include aerobic as well as anaerobic culture of the urine. Treatment is based on culture, but enrofloxacin, trimethoprim-sulfa, metronidazole, and chloramphenicol are examples of antibiotics used in rabbits. Treatment should continue for at least 2 weeks past a normal urinalysis and urine culture. Surgery may be needed to remove a urolith and flushing of the bladder may be necessary to remove sludge. Fluid therapy is imperative initially. Dietary modifications include decreasing the excess calcium in the diet by changing to Timothy hay based pellets and Timothy hay.

Ulcerative pododermatitis
Sore hock, or ulcerative pododermatitis, has many predisposing factors including obesity, wire flooring, or genetically scant amount of hair on the plantar surface (Rex breed). A complete physical examination in a rabbit should always include turning them gently on their backs and evaluating the plantar surface of the heel under the fur. Diagnosis is based on PE, but if the skin is not just pink and flattened, but is also eroded, then a radiograph is needed to determine if the bone just under the eroded skin is infected as well. Aerobic and anaerobic culture is needed before starting the prolonged treatment needed for this disease. Enrofloxacin is a good choice for osteomyelitis due to its good bone penetration until the culture proves the bacteria sensitive. Often months of antibiotic therapy are necessary. Sometimes an ascending synovitis occurs which requires aggressive, immediate therapy. Bandaging the feet, bunny booties of some sort, providing a soft, flat surface to rest are all helpful. In extreme cases, rather than euthanatize a rabbit, a leg amputation can be performed without much morbidity.

Dental Disease
Both incisors and premolars/molars (cheek teeth) grow continuously in rabbits. Some breeds of rabbits may be predisposed to developing dental malocclusion. Similar to brachycephalic syndrome in dogs, short nosed rabbits (dwarf varieties) may be genetically predisposed to dental issues. Malocclusion in rabbits is primarily due to genetics, but diet, trauma and infection can also play a role. A “normal” rabbit should never need its teeth trimmed. Rabbits have 6 incisors, two pair of maxillary incisors (one set are peg teeth) and one set of mandibular incisors. No physical examination is complete without examining the cheek teeth. An ear or nasal speculum can be used to examine the teeth, but often anesthesia is required for a

thorough visual exam. A CT or radiographs can also help diagnose dental disease and any associated infections. Anesthesia is necessary to trim teeth with a Dremel or dental drill and only a nose cone is needed since rabbits are obligate nasal breathers. Rabbits can also be intubated with a 2.0-2.5 mm ET tube. I prefer the blind method of intubation with the neck fully extended. Dental instruments needed include mini floats, speculums, ronguers, tongue/cheek guard, tongue protectors, and head light. The normal occlusal angle is 30 degrees in rabbits. Periodic (q1-3 months) trimming will be needed for life.

Rabbit and Guinea Pig Surgery

Cheryl B. Greenacre, DVM, DABVP-Avian, DABVP-Exotic Companion Mammal
Professor, College of Veterinary Medicine, Department of Small Animal Clinical Sciences, 2407 River Drive, C-247, University of Tennessee, Knoxville, TN, USA

Introduction

The term “neuter” as a verb refers to removing the testicles in a male, but the term is also used to refer to removing the uterus and/or ovaries in a female, and as an adjective refers to one that has had the sexual organs removed. Synonyms for neuter include to sterilize, castrate, spay, geld, fix, desex, alter, doctor, or emasculate. The term “castrate” can be a verb (to remove the testicles) or a noun (referring to one whose testicles have been removed). Vasectomy refers to the surgical cutting and sealing of part of each vas deferens, typically as a means of sterilization.

Surgical principles used in exotic companion mammals are similar to those used in dog and cat medicine, with a few exceptions relating usually to the comparatively different physiology and anatomy of these animals. Rabbits and rodents have heterophils instead of neutrophils. Heterophils lack the enzyme, myeloperoxidase, needed to liquefy pus. Therefore if an infection and abscess is present, then expect the pus to be firm, fibrous, tenacious, and with a thick lining. Also, do not necessarily expect a leukocytosis with abscess formation in the rabbit. Opioids used to control severe pain can slow GI motility, therefore listening for borborygmi should be part of a daily examination in the hospitalized rabbit. Pain can slow GI motility as well, therefore fine tuning of the amount and duration of opioids given versus pain is paramount. Rabbits, guinea pigs, and chinchillas being hind gut fermenters, have a large, thin-walled cecum that can be easily traumatized during abdominal surgery, so care must be taken to handle tissues delicately during surgery. Lacerations to the GI can be attempted to be repaired with 8-0 to 10-0 suture, and copious flushing of the abdominal cavity and antibiotic therapy. All aspects of care lend to a good surgical outcome including diet, if food is withheld at all, maintaining normal body temperature during the procedure, providing adequate pain relief, good surgical technique, proper instruments, sterility, a non-distracted surgeon, and no concurrent disease.

CASTRATION

Age at castration is usually before the animal becomes reproductively active, but there are some varying ideas on this theme. Rats castrated by 4-5 months of age had a lower incidence of mammary fibroadenoma than rats that were not castrated. The surgeon may want to wait until the animal is old enough to have some fat deposits to keep it warmer while under anesthesia.

Pre-surgical considerations

Small mammals are prey animals, so they do not want to be conspicuous if they are in pain or injured. So, try to observe your patient before they are aware you are observing them. Pain evaluation in rabbits and guinea pigs is very difficult and at best we use a newly described facial grimace score. Posters are available free on-line. We also use anthropomorphism (if I had a fractured leg I would want an opiate).

Butorphanol and buprenorphine are commonly used in rabbits. A new study has shown that tramadol, even at very high doses of 11 mg/kg, orally did not result in lasting therapeutic levels (lasted only 15 minutes). Rabbits metabolize tramadol into more than five different metabolites.

In our practice, after premediating with buprenorphine, isoflurane or sevoflurane is generally the anesthetic of choice. Mask induction about 20 minutes after premedication with buprenorphine and midazolam is common, as is post-surgical administration of bupreorphine. I prefer the calm and quick recovery with this method. Others use all injectable anesthesia. Sometimes we reverse the midazolam after surgery with flumazenil. Of note: genetically about half of rabbits have atropinase, rendering atropine useless, but glycopyrrolate can be used if needed. Some use intratesticular lidocaine and/or bupivacaine for analgesia as well (do not go over 2 mg/kg total body dose!).

Food withdrawal in rabbits is not necessary since rabbits do not vomit. Even though guinea pigs technically cannot vomit either, they will have less residual food in the mouth if food is withheld for about 2 hours prior to surgery and be careful to keep the head above the stomach and not to put undue pressure on the abdomen/stomach causing fluid to leak out of the cardiac sphincter. Other, smaller rodents, should only be removed off food for short periods of time (<2 hours) or not at all to prevent hypoglycemia during surgery. Heated air blankets work great at maintaining body temperature of small patients, and are recommended, but take care to not cause hyperthermia. Take temperatures during surgery and watch for tachypnea under anesthesia.

Anatomy and Reproductive Surgery by Species

Rabbits

Rabbits have external testicles and each lies with a separate hemiscrotal sac that is cranial to the penis, which is not typical for placental mammals. Rabbits have an open inguinal ring, therefore the testicles can move between the scrotum and the abdomen freely. The testicle is elongated in shape with peritesticular fat and a prominent epididymis. The secondary sex glands consist of the seminal vesicles, bulbourethral glands, and a prostate. Rabbits also have inguinal (perineal) glands.

Surgical approach to castration in the rabbit is usually a closed technique with an incision in the scrotum over each testicle, or if an open technique is performed then care

should be taken to close the inguinal ring to prevent herniation of abdominal contents into the hemiscrotum. A prescrotal incision can be performed since the penis is caudal to the scrotum, and is usually an open castration and closing the inguinal rings. Technically, an abdominal approach could be performed since there is the open inguinal ring allowing the testes to enter the abdomen. There are descriptions in the literature of an open technique without closing the inguinal ring, citing that the fat pad will prevent herniation of abdominal contents into the scrotum, but this author disagrees with taking that risk. The scrotal incisional tissue can simply be re-apposed (author preference), or tissue glue can be applied, or suture can be used (subcuticular, simple interrupted, or simple continuous). Suture may be associated with a suture reaction, or the animal may be irritated by it and chew the area. If a prescrotal incision is done, then suture is necessary.

If the rabbit has a history of respiratory disease, suggestive of pasteurellosis, then the author prefers to give enrofloxacin prophylactically for approximately 5 days post-operatively. Some sources say the male rabbit can still impregnate a female up to three months post castration due to residual sperm. Castration is usually performed to prevent breeding, but is also done to prevent spraying behavior, the small chance of testicular neoplasia, and to prevent possible chance of male on male aggression. Rabbit testicles descend at about 12 weeks of age.

Guinea pigs

Guinea pigs have external testicles that lie within the perianal sac very near to and cranial to the anus, but due to the open inguinal ring the testes can easily move into the abdomen. The penis is cranial to the testicles. Because of the open inguinal ring, a closed castration technique is recommended with an incision over each perianal sac, or if an open technique is performed, then care should be taken to close the inguinal ring. Alternatively, a prescrotal approach using two separate incisions, or an abdominal castration can be performed. Guinea pigs have a well-developed seminal vesicle that is about 10 cm long, and should not be confused for a uterus if performing an intraabdominal incision.

This author has first-hand experience of three separate guinea pig cases that had been castrated elsewhere and presented to our hospital on emergency with herniation of intestine into the scrotum, with subsequent peritonitis, sepsis, and death in all cases because an open castration technique had been performed without closure of the inguinal ring. The author prefers to cover the anus with a sterile adhesive drape to prevent inadvertent contamination of the sterile surgical field. The scrotal incisional tissue can simply be re-apposed and tissue glue applied, or suture can be used (subcuticular is preferred, but simple interrupted, or simple continuous can also be used), Some animals may experience a suture reaction, they may be irritated by the suture, or chew the area. The author prefers to close the guinea pig scrotal skin with a subcuticular (aka interdermal) continuous suture with 4-0 polydioxanone, the least reactive suture material. Due to the possibility of post-operative abscesses in guinea

pigs due to *Leptotrichia* sp. anaerobic bacteria, the author prefers to give chloramphenicol for approximately 5 days post-operatively.

OVARIOHYSTERECTOMY/OVARIECTOMY

Rabbits

Ovariohysterectomy of female rabbits is recommended before two years of age to prevent the incidence of uterine adenocarcinoma. One study recorded 60% of female rabbits over 4 years of age had uterine adenocarcinoma. The silver Marten breed of rabbit has up to an 80% chance of this tumor after five years of age. Besides preventing uterine neoplasia, reasons to perform an OHE include to prevent pyometra, cystic endometrial hyperplasia, pseudopregnancy, breeding, urine spraying, hormone related aggression especially with other female rabbits, and the rare post-parturient urinary bladder prolapse.

Rabbits are induced ovulators, like ferrets. Rabbits, rats, and mice have a duplex reproductive anatomy with a double cervix and a relatively long vagina. Rabbits have two cervices, therefore the proximal ligature can be at the level of the distal vagina, meaning just proximal to both cervices. Care should be taken not to traumatize the nearby thin walled cecum, and to also avoid the nearby ureters. There is also a thick (1 cm sometimes), fat filled broad ligament (mesometrium) making visualization of uterine vessels difficult, especially in overweight rabbits. Remember that rabbits are prone to adhesions, so flush any blood from the abdomen.

Guinea pigs

OHE of female guinea pigs is similar to rabbits and is used to prevent the high incidence of cystic ovary(ies) at 5 years of age. If a guinea pig is bred for the first time after 8 months of age there is a very high risk dystocia because of pelvic bone fusing. Currently, our practice prefers to perform ovariectomy (OVE) in guinea pigs since there is less trauma than a traditional spay and the most common problem in guinea pigs is cystic ovaries. Other rodent surgeries are similar to the guinea pig. OHE or OVE of pet female rodents is becoming more common, and is usually done to prevent reproduction in multiply housed animals, cystic ovaries, or to prevent neoplasia of reproductive or mammary tissue. The surgery is similar, albeit smaller, than any other OHE.

In our practice to perform an ovariohysterectomy, the guinea pig is preanesthtized with midazolam (about 0.3 – 0.5 mg/kg SQ) and buprenorphine (about 0.03 mg/kg IM - studies have shown that buprenorphine given IM absorbs better than when given SQ) about 20 minutes prior to induction of anesthesia with isoflurane. Three attempts are

made to intubate the guinea pig once it is anesthetized and then if not possible then we decide whether to go forward with the surgery or do it another day. It is best to do all other preparatory work needed (clipping, pre-scrub, +/- catheter, etc.) prior to intubation so that the guinea pig is deep when you attempt intubation. A common mistake is to try to intubate first thing before the guinea pig is fully under anesthesia. It also helps to place the isoflurane mask over just the nose while you are attempting intubation, so that they continue to receive isoflurane – this works because they are obligate nasal breathers. In dorsal recumbancy, an approximate 6 cm or smaller incision is made from about 1 cm cranial to the umbilicus caudally to expose the uterus. Some prefer to use intraincisional lidocaine and/or bupivacaine. The ovarian pedical is ligated as well as uterine vessels with either 4-0 PDS or medium metal clips. The uterus is ligated with 4-0 PDS. The linea is closed with 3-0 or 4-0 PDS in a simple continuous pattern, and the skin is closed with the same suture in a subcuticular pattern.

Gastrotomy

Most trichobezoars in rabbits can be handled medically as described in the medicine presentation, but rarely one will require surgery. Surgery is needed is there is a toxic gum line (such as a colic horse), a complete obstruction evidenced by severe gas distension, or rarely, a foreign body. Reinstating normal gut motility post GI surgery in the rabbit or guinea pig is difficult and requires aggressive therapy with fluids (both oral and parenteral), balancing pain medications with need, offering motility stimulators such as metaclopramide or cisapride. The surgery itself is similar to a dog or cat except that no blood or contamination can be left behind due to formation of adhesions and peritonitis. Therefore, perform copious lavage with warmed saline. Immedieatley during and after surgery we use buprenorphine, and then after 24 hours butorphanol. Meloxicam is given immediately after surgery. There is some concern of hypovolemia if meloxicam is given during anesthesia.

Abscess control (example otitis media, could also be dental related)

Diagnosis of otitis media can sometimes be difficult with the general lack of or varied clinical signs, the common inability to visualize the tympanic membrane, the common possibly normal presence of exudates within the ear canal.[9] When suspecting otitis media in a rabbit based on any of the aforementioned varied clinical signs, the best diagnostic tool is a radiograph or computed tomography (CT) of the skull to evaluate the wall thickness, content, shape and size of the tympanic bulla. The osseus portion of the tympanic bulla of rabbits is generally very thin, less than 1 mm in thickness. Any

increased thickness, irregular margin, or bony lysis of the tympanic bulla wall, or presence of material in the cavity of the tympanic bulla, is indicative of otitis media.

Aerobic and anaerobic culture of the tympanic wall lining is the best method to grow the primary organisms causing disease. Culturing bulla contents is less desirable. Culture of the exudates in the external ear canal will likely grow many contaminants. If the tympanic wall is intact, then only an aggressive approach, such as a tympanic bulla osteotomy will allow access to the contents or lining for culture. A tympanic bulla osteotomy also provides a port for drainage and flushing of pus, but this procedure is considered by many to be of little benefit and carry a poor success rate.[2,16] Long term, as in months long, parenteral antibiotic therapy is the recommended treatment for rabbits with otitis media. The current trend is to give enrofloxacin orally and give penicillin G with benzathine subcutaneously every 5 to 7 days.[17] The benzathine formulation of penicillin provides longer action. Antibiotic therapy based on culture is best. Flushing, cleaning and instilling antibiotics into the external ear canal does little to resolve the infection and rarely removes the all the exudates, but it may make the patient more comfortable and scratch at the ears less.

Antibiotic impregnated polymethylmethacrylate beads can be placed in areas of infection, such as an abscess, after thorough cleaning and debriding, The placement of beads can be delayed until the tissue has undergone some second intention healing before using first intention healing methods when closing beads into the tissue. Antibiotic elutes from the beads into the nearby tissue and is effective for 2 to 3 mm around each bead.[3,17,18] Approximately 5% of the antibiotic present is released into nearby tissue in the first 24 hours, but systemic levels are low. The exact point at which no further elution occurs is not known, but beads impregnated with gentamycin had completed eluted by 37 days after placement into tissue. Many factors affect the rate of elution including bacterial or debris contamination at the site, blood supply to nearby tissue, the number of beads placed, the amount of antibiotic in the bead, the bead’s proximity to the affected tissue, and the appropriateness of the antibiotic chosen for the pathogen present. The beads can be removed later or left in place. Antibiotics that work well in beads are those that are heat stabile to withstand steam sterilization and those that come in a powdered form, such as ceftiofur, cephazolin, and gentamycin. Penicillin, ampicillin, amoxicillin, and clindamycin have also been used.

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Medication Use in Backyard Poultry

Cheryl B. Greenacre, DVM, DABVP-Avian, DABVP-Exotic Companion Mammal Professor, College of Veterinary Medicine, Department of Small Animal Clinical Sciences, 2407 River Drive, C-247, University of Tennessee, Knoxville, TN, USA

Introduction

Backyard and companion poultry are now commonly being presented to veterinary practices for individualized and flock care. It is important to realize that even though someone may present you their dear pet chicken that they would never eat, you are responsible for knowing that it is still considered, and regulated, as a "food animal *species*" according to the United States Food and Drug Administration (FDA) and their regulations must be followed. Below are descriptions of the regulations and terminology encountered in regards to medication use in backyard poultry.

Backyard chickens are birds and all of our knowledge of avian medicine can be used in their care including general husbandry and care, handling, approach to medicine and surgery, and anatomy and physiology within the confines of federal regulations regarding medication use. Consulting an avian textbook, or exotic animal formulary for general information on birds is a good starting point.

Backyard poultry usually brings to mind chickens, although the term backyard poultry also includes turkeys, pheasants, ducks, geese, swans, quail, and other species. The diseases and care of backyard flocks is somewhat different than that of commercial broilers, breeders or layers and the following will pertain to backyard and companion poultry.

Prohibited Drugs

The FDA prohibits the use of these drugs with no allowable extra-label drug use in any food producing animal species such as chickens and turkeys: "chloramphenicol, clenbuterol, diethylstilbesterol (DES), fluoroquinolone class antibiotics, glycopeptides (all agents including vancomycin), medicated feeds, nitroimidazoles (all agents, including dimetridizole, ipronidazole, metronidazole, and others), nitrofurans (all agents including furazolidine, nitrofurazone, and others), adamantane and neuraminidase inhibitors (in all poultry including ducks); cephalosporin class of antibiotics except cephaparin (in all classes of chickens and turkeys); gentian violet (prohibited from use in food or feed of food producing animals), and indexed drugs (some exceptions for minor use species); extra-label drug use (ELDU) restrictions apply to all production classes of major food animal species (no ELDU for purpose of disease prevention, no ELDU that involves unapproved dose, treatment duration, frequency or administration route, and agent must be approved for that species and production class); ELDU restrictions DO NOT APPLY to minor-use food animal species."

All fluoroquinilones (like enrofloxacin) and cephalosporins are PROHIBITED drugs in poultry, which means you CANNOT give them to poultry. It does not matter the use of the poultry, so even pet poultry cannot be given prohibited drugs. You cannot get around this by having the owner sign that it is OK to give it – these are prohibited drugs and cannot be used. The reason? For the fluoroquinilones, one reason is that chickens amplify the creation of antibiotic resistant *Campylobacter* sp. which would create a situation in which humans could get a severe, even life threatening diarrhea from one of these organisms and physicians would have very limited to no treatment options.

Extra-Label Drug Use (ELDU)

There are other drugs that fall under the rules of Extra-label drug use (ELDU). ELDU is any of the following situations (with examples) where a drug is not given exactly as written on the label: use in another species (trimethoprim sulphamethoxizole directly orally to a duck when not labeled for use in ducks), use for a different indication (erythromycin administered as per label instructions but for pododermatitis rather than chronic respiratory disease, use at a different dose or frequency (administering spectinomycin for more than the first 3 days of life to a chicken), use via a different route of administration (erythromycin directly orally, not in food or drinking water).
Contact www.farad.org for specific information and instructions each time you use an ELDU in poultry since recommendations can change with updated data. FARAD (Food Animal Residue Avoidance Databank) provides an on-line service where you can submit a proposed drug dose, frequency, route, concentration, and duration, and within 48 hours they will provide you with a suggested withdrawal for that drug. Antibiotics that are commonly used in this manner include tylosin, the penicillins, sulfa drugs, tetracyclines, and macrolides such as clindamycin or erythromycin.

Labeled Drugs

Labeled drugs will have an A/NADA (Abbreviated/New Animal Drug Application) number and current information can be easily accessed by looking up a labeled drug by this number.
A NADA is used to seek approval of a new animal drug. An ANADA is used to seek approval of a generic new animal drug which is a copy of an approved new animal drug for which patents or other periods of exclusivity are near expiration. A labeled drug must be used exactly as is written on the label to be considered as labeled drug use. If the labeled drug is an antibiotic that is to be given in the food or water, then a Veterinary Feed Directive is needed. An example of a labeled drug is: Erythromycin (erythromycin thiocyanate, Gallimycin, Cross Vetpharm Group Ltd., NADA 010-092) - 185g/ton of feed, to aid in the prevention and reduction of lesions and in lowering severity of chronic respiratory disease; feed for 5 to 8 days; do not use in birds producing eggs for food purposes; withdraw 48 hours before slaughter.
Other sources of information regarding labeled drugs include:

1. US Food and Drug Administration (FDA) Center for Veterinary Medicine has a searchable database called “Animal Drugs @ FDA” where you can search for drugs by trade name or A/NADA number and can be found at https://animaldrugsatfda.fda.gov/adafda/views/#/search
2. The Minor Use Animal Drug Program has a database on their web site (http://www.nrsp7.org/mumsrx/Species) to search for approved drugs, either by active ingredient or trade name, and has an avian-specific section for minor species such as ducks, pheasants, partridges, and quail.
3. A list of veterinary drugs, label directions, and A/NADA numbers can also be found at https://www.drugs.com/vet

Veterinary Feed Directive (VFD)

A Veterinary Feed Directive (VFD) is needed for any feed or water additive given to a food animal. There must be a valid Veterinary Client Patient Relationship (VCPR). The VFD must include the following written directions (order): veterinarian’s information with signature, client information, where animals are located, and type and # of animals, date issued, expiration date,

indication for use, dose, withdrawal time, and specific verbiage [“Use of feed containing this VFD drug in a manner other than as directed on the labeling (extralabel use), is not permitted].

Eggs

There are NO dewormers available for use in a chicken that is laying eggs to be sold. Furthermore, there are very strict rules if you are selling eggs to the public, and the only thing that can be used for attempted deworming is oregano, kitchen/food grade diatomoaceous earth, or pumpkin.

Salmonellosis concerns

Educate owners regarding the risk of salmonellosis especially for children under 5 years, the elderly, or immunosuppressed persons since the risk in them is potentially life threatening. Chicks and ducklings can carry Salmonella in their droppings normally with no clinical signs of disease. Education materials are available from www.cdc.org. Our hospital asks people to sign that they have read an informational sheet on salmonellosis when they present with any poultry or reptile.

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CRUCIATE INJURY AND REPAIR IN THE DOG

Steven C Budsberg DVM, MS, DACVS
Professor of Surgery
University of Georgia
Email- Budsberg@uga.edu

Introduction

Disease of the cranial cruciate ligament (CCL) is the most common condition to affect the canine stifle joint. The postulated factors involved in the pathogenesis of CCL rupture are many and include: genetics, breed, age, gender, neutering, ischaemia, obesity, immune mechanisms, tibial plateau angle, intercondylar notch, and local biomechanics. CCL rupture occurs in all sizes of dogs but affects larger breed dogs more often than smaller dogs, and at a younger age. Epidemiological studies have indicated an increased prevalence of CCL disease in breeds such as the Newfoundland, Rottweiler and Labrador retriever, with infrequent occurrence in the Greyhound, Bassett Hound and Old English sheepdog. CCL rupture occurs more commonly in neutered animals, particularly females. It is unknown if this is secondary to abnormal weight gain, as it has been reported that 45.4% of spayed bitches are obese. It is now known that CCL disease has an inherited component in the Newfoundland and Boxer. The classic acute cranial cruciate ligament rupture occurs rarely in dogs. More commonly, slow degeneration of the ligament and osteoarthritis of the joint occurs, due to unknown etiology, before functional failure of the ligament occurs. A high percentage of dogs with unilateral cranial cruciate ruptures subsequently rupture the opposite cranial cruciate ligament. Therefore, there has been significant research into the potential causes of this common problem. Recent genetic evaluations have identified specific genes that may play a part in the development of CCL dysfunction.

Biology

With increased risk of CCL disease in certain breeds and neutered animals, one can ask many questions relating to the nature of the tissue in these animals. Is the CCL "normal" in these animals? For example, is the structure and turnover of the CCL normal? Is the biochemistry of the CCL normal? Earlier studies examined CCL biochemistry, ultrastructure and biomechanics of CCLs from two at-risk breeds (Labrador and Golden Retriever) and compared these to a low risk breed (Greyhound). Collagen fibril diameters were measured and it was found that the mean fibril diameter in the Labrador is significantly smaller than that of the Greyhound. Markers of collagen turnover in CCLs in these breeds of dog have also been assessed. These data suggest that collagen turnover in CCLs from at-risk breeds is increased. Recent work has focused on the cell morphology. Alterations in cell morphology may alter the ability of cells to produce healthy matrix and repair damage through disruption of collagen production. There are marked regional variations in the cell morphology of the canine CL complex. Additionally, chronic inflammation has been suspected in the pathophysiology of CLL dysfunction.

<u>Conformation biomechanical perspectives</u> – In recent years, the tibial plateau has been a subject of much debate. Does an excessive slope to the tibial plateau contribute to the incidence of CCL disease? One study suggests that dogs with CCL rupture do have an excessive slope to the tibial

plateau. However, other studies have failed to substantiate these data. Interestingly, one recent study has raised the possibility of tibial tuberosity conformation being a risk factor for CCL disease. Another study looked at a variety of conformational variables and suggested that cranial angulation of the proximal portion of the tibia, excessive steepness of the tibial plateau, and distal femoral torsion appeared more likely to be associated with CCL deficiency than femoral angulation, tibial torsion, intercondylar notch stenosis, and increased inclination of the patellar ligament. Currently there is no complete understanding of the reasons why CCL fail in dogs.

Management of CCL dysfunction

Conservative Management - Exercise restriction, weight loss, and physical therapy has been recommended for treatment of cranial cruciate rupture. It seems to be more often attempted in small dogs (<10 kg) and cats. In this author's experience, however, lameness does not often completely resolve in these animals and they commonly return for surgery.

Surgical Management - Currently recommended surgical techniques can be roughly divided into two groups; techniques that change the mechanics of the stifle to achieve stabilization, and techniques that act to restrict drawer motion with a physical device. The former group includes TPLO, TTA, and various modifications of the tibial wedge osteotomy. The latter includes long-described extracapsular suture techniques and intracapsular reconstructive techniques. Currently, some form of Tibial Plateau Leveling Osteotomy (TPLO), Tibial Tuberosity Advancement (TTA), and Extracapsular Suture, including the TightRope®, are the three most widely accepted treatment methods. No perfect treatment for cruciate rupture has been identified in the dog.

Extracapsular stabilization with sutures is still a useful technique with proven positive outcomes. The most common methods employ a synthetic suture passed around the lateral fabella and through the tibial crest or employ bone anchors in the distal femur and proximal tibia. In three recent publications, extracapsular stabilization was compared to TPLO and was found to provide significant improvements in function after surgery but was inferior to the TPLO. In a retrospective study, complications were recorded in 63 of 363 lateral fabellotibial suture surgical procedures (17.4%) and 7.2% required a second surgery to manage the complications. A second more recent study confirms an infection rate at 17 %. Factors significantly associated with a higher rate of complications were high body weight, propofol induction agent and young age of dog at the time of surgery. These findings are similar to other previous retrospective studies of extracapsular procedures including the TightRope® procedure.

Tibial Plateau Leveling Osteotomy (TPLO) has been applied to clinical cases for over two decades now. The TPLO mechanically reduces cranial tibial thrust in the weight-bearing phase by "leveling" the tibial plateau. It is important to note that there is no difference in tibial plateau angle between dogs that rupture or do not rupture their cruciate ligaments, but that correction in the angle biomechanically stabilizes the stifle. The overall complication rate after TPLO in 1000 cases was 14.8% (6.6% major), which included 2.8% meniscal injury and 6.6% infection. TPLO may be combined with tibial wedge osteotomy for dogs with complex tibial deformities or exaggerated tibial plateau angles. Earlier studies found a much higher complication rate, which is to be expected as the procedure was being refined. Recent studies have shown TPLO to be effective in small dogs and may be more efficacious than extracapsular repair in small dogs.

The Tibial Tuberosity Advancement (TTA) seeks to eliminate tibial thrust by positioning the patellar tendon perpendicular to the shear forces in the stifle, resulting in the same redirection of vector force as the TPLO. TTA theoretically relieves patellar ligament tension whereas TPLO may increase it. The overall complication rate after TTA has been reported to be between 25 and 31.5%. Three published studies reflect the early clinical experiences with the TTA technique. These three studies (249 cases) report a total overall complication rate of 20.0%-59% in cranial cruciate ligament deficient stifle joints repaired using the TTA. The major complications were between 12-38%, with a re-operation rate of 11.3-14.0%. Again, as with the TPLO, these studies were done early in the use of this procedure and the complication rate is probably much lower now. Recent publications suggests that a TPLO may be more effective at returning dogs to normal function compared to TTA, however more data is needed.

Meniscal Removal or Release

Medial Meniscal injuries are quite common following rupture of the cranial cruciate ligament. An incidence of 50-70% of meniscal injury, identified at the time of surgery for CCL injury, has been reported in dogs. The medial meniscus is most commonly damaged as it is more firmly attached to the joint capsule and medial collateral ligament than the lateral meniscus. However, lateral discoid tears and longitudinal tears of the lateral meniscus are reported. The lateral meniscus is attached to the femur by a ligamentous attachment and when cranial drawer occurs in a CCL deficient knee, the lateral meniscus remains with the femur and is loaded normally. However, the medial meniscus moves cranially resulting in the caudal horn being loaded abnormally. The management of these injuries usually involves removal of the damaged area. Meniscal resection induces osteoarthritis. Any surgical intervention on menisci should be carefully considered. Meniscal injuries are associated with pain necessitating surgical intervention. Resection of meniscal tears improves short-medium term outcome but carries a poorer long-term outcome. Damage can also occur following surgical treatment of a cruciate injury. Currently, a more perplexing issue is the practice of the meniscal release (either transection of the ligament of the caudal pole of the medial meniscus or the transection of the mid body of the medial meniscus) as part of cruciate ligament rupture management in conjunction with a TPLO or TTA. Furthermore, some surgeons have recommended meniscal release as the preferred method of primary treatment of any meniscal pathology.

Why Meniscal Release?

Late meniscal injury has been reported and is believed to be the result of continued cranial tibial thrust. Postliminary (“late”) meniscal injuries are reported in dogs. These injuries have been reported to occur from 3 weeks to 9 months post-operatively, with an average of 6 months after the first surgical procedure. Dogs with this injury will typically present as having had a normal recovery after the first surgery and then present with an acute lameness 6 weeks to 6 months in the previously operated limb. With rupture of the cranial cruciate ligament, there is loss of the passive restraint to the cranial tibial thrust allowing the femoral condyle to displace caudally over the tibial plateau. The medial meniscus is especially susceptible to injury due to the rigid attachment of the caudomedial meniscotibial ligament. This attachment essentially holds the meniscus in place while the femoral condyle crushes the caudal horn with excessive tibial thrust.

It has been shown that meniscal release results in increased contact stress between the femoral and tibial condyles, thus predisposing the cartilage surfaces to increased stress and likely subsequent degeneration and formation of osteoarthritis. Recently the TPLO and TTA have become popular surgical interventions for the cranial cruciate ligament deficient stifle aimed at neutralizing cranial tibial translation. Such procedures have two schools of thought behind them but neither has strong clinical data to support their claim. One group feels that there is a need to release the medial menisci as there is still movement between the femur and tibia and thus the meniscus is at risk, while the other group feels that those procedures are protective of the meniscus through elimination of the caudal pole impingement of the meniscus, thus obviating the necessity for the concurrent release.

Early anecdotal reports stated that without meniscal release, dogs undergoing TPLO procedures had a high rate of subsequent medial meniscal injury. These statements were never confirmed in any published peer review reports, yet the practice of meniscal release grew over the years. The actual incidence of meniscal injury following cruciate rupture is unknown. There is some data from studies following initial surgical visualization. Data collected from other methodologies of repair (extracapsular sutures and intraarticular graft replacements) suggested that about 12 percent of cases had subsequent meniscal injuries which required repair. However, dogs with clinical problems were the only ones who had second surgical explorations, thus the actual number may be higher. A recent relatively small study suggests that there is between 3% (joints explored with arthroscopy) and 10% (joints explored with an arthrotomy) of cases of subsequent medial mensical injury without meniscal release after TPLO. However, no large prospective, randomized, clinical trial has been completed evaluating the effects of meniscal release on the rate of secondary meniscal tears in surgically stabilized cranial cruciate deficient stifles. Recent retrospective studies indicate that the meniscal release procedure does not prevent secondary tears from occurring. These data counter earlier data suggesting the procedure was needed. Thus the conundrum facing us today do we release or leave the apparently normal meniscus alone? In vitro studies have produced data that do not answer all the questions and have some conflicting results. Not surprisingly, in vitro canine cadaveric data has shown that meniscal release has some significant effects on the joint. In one study, radial transection of the medial meniscus resulted in significant alterations in pressure magnitude and distribution through the axially loaded stifle joint. Other data found an increase in pressure on the cartilage of the medial tibial condyle with meniscal release and TPLO. Also, meniscal release was equivalent to caudal pole hemi-meniscectomy in regards to load-bearing, implicating the loss of hoop tension for this high and non-uniform pressure distribution. The effect of meniscal release on stifle joint stability was not different from caudal pole hemimeniscectomy, suggesting that the former had no advantage over the second in regards to contributing to stifle joint stability. Meniscal release also caused greater cranial tibial thrust in the CCL deficient stifle joint compared to the intact stifle joint in cadaveric limbs. The limited *in vivo* data strongly suggested that release of the medial meniscus does induce significant pathological changes in the stifle joint or in the function of the limb.

Conclusions

CCL dysfunction is very common in dogs. The disease process is complicated and rarely involves a supra-physiologic injury. Thus bilateral CCL rupture is not uncommon. Regardless of the technique used, extracapsular techniques or tibial osteotomies should result in improvement

in the surgical population following surgery. No current technique will halt the progression of osteoarthritis.

Obesity and Musculoskeletal Problems

Steven C Budsberg DVM, MS, DACVS
Professor of Surgery
College of Veterinary Medicine
University of Georgia
Email – Budsberg@uga.edu

Every day we are seeing more and more fat pets in our hospitals. Yet the realization and acceptance of the problem has been paradoxical. Our patients are heavier yet obesity is not often noted as a diagnosis, perhaps reflecting the perception of practitioners that obesity does not constitute a disease state.[1] Perhaps we should step back and remember the definition of a disease which is "a pathological condition of a part, organ, or system of an organism resulting from various causes, such as infection, genetic defect, or environmental stress, and characterized by an identifiable group of signs or symptoms." I think it easy, although perhaps debatable, to list obesity as a disease. Excess body weight has been associated with or may exacerbate a wide range of potentially serious conditions; not limited to locomotor and musculoskeletal problems, respiratory dysfunction, hypertension, cardiac disease, diabetes mellitus, and neoplasia.[2-16] Adipose tissue was once thought of a passive fuel depot, it is now recognized as an active endocrine organ that communicates with the brain and peripheral tissues by secreting a wide range of hormones and protein factors often collectively called adipokines. Examples include adipokines such as leptin, adiponectin, and several cytokines including TNF-alpha, and IL-6.[17]

The impact of obesity on osteoarthritis (OA) and musculoskeletal dysfunction in dogs and cats is well documented. In this lecture we will discuss the known associations between obesity and musculoskeletal problems. Then we will discuss the disease osteoarthritis, the most common long term medical consequence of obesity on the musculoskeletal system.

Numerous studies conclude that obesity negatively affects the canine musculoskeletal system. One of the most important studies documents the effects of excess weight in the development of osteoarthritis in Labrador Retrievers.[11-16] This seminal study has provided a wealth of data that is useful in treating our canine patients. While the results of this study are repetitively referenced, it is important that we spend some time reviewing the findings. Forty-eight puppies were paired by sex and body weight within their litter to participate in the study. Puppies came from 7 dams and 2 sires. At eight weeks they enrolled one dog from each pair in the control fed group and one dog entered the limit fed group. These dogs were then followed for life. Collected data included effects on the development of osteoarthritis (OA) in multiple joints and on the causes, time and predictors of death and ultimately life-span. Briefly, the prevalence and severity of OA in several joints was less in dogs in the limit fed group compared to the control fed group. Specifically in reference to the coxofemoral joint, limit (restricted) feeding delayed or prevented the development of radiographic signs of hip OA. The slowing of OA had a favorable affect on both the duration and the quality of life. The median life span of the control fed dogs was 11.2 years, while the limit fed dogs had a median life span of 13.0 years. Declining lean mass was predictive of death, most significantly at a year prior to death. Additional data has provided support to suggest nutritional over-supplementation (overfeeding) contributes to several developmental bone disorders including hip and elbow development and osteochondrosis.[10, 11] There is one report that found weight to be a predisposing factor in humeral condylar fractures,

cranial cruciate ligament ruptures and intervertebral disc disease in cocker spaniels.[18]

In cats there is a paucity of information linking orthopedic disorders and obesity. Limited data indicates that cats with increased body condition scores are nearly 5 times more likely to develop lameness requiring veterinary care.[19] Furthermore, there is an association between overweight cats and the development of Salter I fractures of the proximal femoral physis (slipped capital femoral epiphysis) without apparent trauma.[20,21] Finally heavier cats are potentially more susceptible to cranial cruciate injuries.[22]

Despite the different effects obesity has on the musculoskeletal system, the overriding long term consequence is OA. It is generally accepted that the most effective non-surgical approach to address OA pain in any diarthroidal joint is multifaceted including effective weight control, proper exercise, physical therapy, and analgesic medication, which have already been discussed today at length in other lectures.

Interestingly our calls for change are not new. Joshua[23] wrote in 1970 about obesity and said the role of the profession was to prevent rather than cure obesity. Dr Joshua went on to say that people recognized the dangers of obesity in their children and themselves and they must be made to face the problem in their pets. These statements echo the saying “The more things change, the more they stay the same”.

Additionally a couple of recent articles deserve mention and are free online. The first is a well written about obesity in our patients and the real physiologic it has on them..[24]The second one is an interesting article that provides an overall look at some of the recent discussions about canine obesity and osteoarthritis. [25]

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Antimicrobial Prophylaxis in Orthopedic Surgery

Steven C Budsberg DVM, MS, DACVS
Professor of Surgery
University of Georgia
Email- Budsberg@uga.edu

Definition and History: A working definition of antimicrobial prophylaxis in surgery is the administration of an antimicrobial drug to a patient, in the absence of infection, prior to surgery. The history of the use of these agents during surgery is interesting and reveals many of the problems which occur with their use. When antimicrobial agents became available to surgeons, they did not provide the panacea for prevention of all surgical infections. In fact, a twenty-year analysis indicated that no significant alteration of infection rates had occurred since the advent of prophylactic antimicrobial usage in human surgery. The study went on to identify the following misuses:

1. Excessive use in clean surgical procedures
2. Faulty timing of administration of the antimicrobial agent
3. Continued use beyond the time necessary for benefit.

Today, unfortunately, some of these misuses are still occurring in veterinary surgery.

The reason that misuses still occur in our profession is partly due to the limited amount of data based on clinical studies in veterinary medicine. Most of the studies available do not justify the use of prophylactic antibiotics in the study populations examined. Despite this fact, it is safe to say that a majority of surgeries done in veterinary practices are performed with antimicrobials given to the patient.

Wound Infections: In the evolution of wound infections, there are three main components. These are bacterial inoculum, bacterial nutrition, and impaired host resistance. The mere presence of bacteria is less important than the level of bacterial growth. Therefore, the goal of the surgeon is to maintain a favorable balance between patient and bacteria. It is important to remember that proper surgical technique and proper patient preparation, strict adherence to aseptic technique and application of atraumatic surgical technique are far more important in the prevention of infection than the use of antibiotics.

Patient Profile for Antimicrobial Prophylaxis: The next question to ask is "In which patients should I use antimicrobial prophylaxis?" There are no hard and fast rules to follow but the following examples can be used for some general guidelines. Many orthopedic procedures are defined as clean surgical wounds, and, in general, the use of prophylaxis is difficult to justify. Important factors to consider when giving antibiotics prior to surgery include: anticipated duration of the operation (degree of contamination), local wound factors favoring infection (e.g., extensive tissue trauma, placement of large implants) and systemic factors favoring infection (e.g., concurrent infections, diseases suppressing immunity).

Procedures in which it is difficult to justify giving antibiotics include:

1. Arthrotomies including removal of endochondral ossification defects or open joint reductions
2. Arthroscopy
3. **TPLOs ? or TTA's?**

Procedures which can be more easily justified for the use of prophylaxis are:

1. Total hip replacement
2. Complex multiple fractures
3. Open fractures
4. Systemically comprised patients

Timing of administration: Maximal therapeutic concentrations of the antibiotic must be present in the tissue at the time of contamination (i.e. beginning of surgery)!!! Experimental work has demonstrated a short, early period in which "decisive biochemical interactions" between the microorganisms and the host tissue occur. During this time the development of the primary bacterial lesion is susceptible to the action of parenterally administered antibiotics. The major effect is in the first minutes of the contamination and no effect is seen if antibiotics are given 3 hours after contamination has begun. Thus, if given intramuscularly, administer 30 minutes prior to your incision. If given intravenously, administer 15 minutes prior to the incision. Repetitive dosing during surgery should occur depending on the antimicrobial given. As an example with a first generation cephalosporin (cefazolin) every 2 to 2.5 hours is adequate according to published data. Serum half-life has been used as a guideline for this dosing, but it is not consistent with concentrations in the tissue (i.e., the drug is given at every half-life).

Choice of Antibiotics: No single antibiotic agent or combination can be relied on for effective prophylaxis in all the various settings found in surgery. Antibiotics used in surgery should be aimed toward the expected contaminating bacteria. The antimicrobial agent should also be bactericidal, have a low side-effect profile, be cost effective and be parenterally administered. In orthopedics, the expected contaminating organism is a staphylococcus from the skin of the patient, which usually produces beta-lactamases. Thus cephalosporins, semi-synthetic beta-lactamases, resistant penicillins, and clindamycin are acceptable choices.

Duration of Antimicrobial Prophylaxis: The use of antibiotics beyond the immediate postoperative period is unnecessary. * 6 – 12 HOURS POSTOPERATIVE * There is strong evidence that use of antibiotics for days after surgery is not only unnecessary but can actually be detrimental to the patient. Now I know there is some conflicting data with TPLO's that may disagree with that and we will look at the data.

Potential Advantages: The most obvious advantage is the prevention of infections. This, in turn, will decrease morbidity and mortality from surgery and decrease hospital stay and cost. Ultimately, this pulse form of usage can actually reduce total antibiotic use in surgical patients.

Potential Disadvantages: In veterinary medicine, we do not look critically at the potential disadvantages of antibiotic use. The most clinically important and seldom considered disadvantages are:

1) **The development of resistant organisms**.
2) Allergic reactions also are not considered, unless it entails a life threatening situation.
3) Non-allergic toxic reactions such as nephrotoxicity of aminoglycosides.
4) Costs, remember the cost of each dose given to a patient in which antibiotics are not necessary should not be overlooked.

Case Example

A simple example I always use that will hopefully help you make your decision on antibiotic use is this:

You have an infection rate of 4 % for a given procedure. You are attempting to decrease that rate to 2 %. To institute your plan, you perform 100 of the aforementioned surgical procedures. All animals receive a prophylactic antimicrobial drug. After evaluating all the patients, you discover that you have decreased your infection rate to 2%. Thus you have accomplished your goal. Now the question arises, was it worth it?
(i.e. Are you willing to give 96 dogs antimicrobial agents which are not benefitting them to prevent an infection in 2 animals?)

TREATMENT OF OSTEOARTHRITIS IN DOGS AND CATS

Steven C Budsberg DVM, MS, DACVS
Professor of Surgery
University of Georgia
Email- Budsberg@uga.edu

Introduction

While considered a very common problem in small animal medicine, osteoarthritis is very likely the most under diagnosed and misunderstood rheumatic disease in dogs and cats. Part of the problem veterinarians face with OA is that it is a slow, progressive, and often insidious problem. In the dog, primary OA is uncommon and OA development always occurs secondary to another joint pathology. The wide range of clinical signs makes OA a commonly misdiagnosed condition. The exact number of dogs affected with OA is well over 20% and in cats over the age of 10 it is estimated to be over 80%, probably closer to 95%.

Pathophysiology

Osteoarthritis is characterized by articular cartilage degeneration and changes in the periarticular soft tissues (synovium and joint capsule) and subchondral bone. Specifically, the pathologic changes of osteoarthritis encompass articular cartilage degeneration, which includes matrix fibrillation, fissure appearance, gross ulceration, and full-thickness loss of the cartilage matrix. This pathology is accompanied by hypertrophic bone changes with osteophyte formation and subchondral bone plate thickening. Failure to repair the damage affecting the surface cartilage is a distinctive condition of OA. Failure of chondrocytes in injured articular cartilage to restore a functional matrix in spite of high metabolic activity remains a complex and challenging problem. What this says to the clinician now is that there is no treatment regimen proven to arrest or reverse the cartilage degeneration.

Treatment Goal

Current therapy is primarily palliative, aiming to reduce pain and inflammation and maintain or improve joint function without altering the pathologic process in the tissue. Remember, most OA in the dog and cat is secondary to some other pathologic state, and thus the underlying cause must be identified in an attempt to minimize the long-term effects. Certainly, efforts are being made to provide treatments which may alter the course of the disease, but these therapies are still to a large part unproven.

Treatment Plan

Management of OA should be thought of as a multi-step approach with four to five important components. While some clinicians tend to reach for pharmacologic management alone, this is usually unsuccessful without concurrent management of exercise and weight reduction. Thus, starting to treat a patient with OA requires a lengthy discussion of all aspects of management with the client. Our discussion will follow the typical pattern we use in our practice. Remember, one must examine each case differently, assessing the age, normal activity levels, and, most importantly, the owner's expectant activity levels of the animal. Success largely depends on the accurate assessment of the client’s expectations for the pet.

Management Components

1. Weight Reduction

Weight control is necessary when dealing with OA. The vast majority of our patients seen with clinical manifestations of OA are obese. Owner education and proper dietary management must be considered in every case. In many cases, the implementation of weight reduction with rest and exercise modification diminishes or completely alleviates the clinical signs of OA.

2. Nutritional Support

The recent influx of diets on the market with a high N3:N6 fatty acid ratio is adding a whole new area of intervention. It is important to understand that there is an increase in N3 fatty acids in the diet and that specific N3 fatty acids are elevated (EPA and DHA).

3. Exercise modification/Physical Therapy

Protecting the osteoarthritic joint from excessive mechanical stress may limit clinical signs. Use of the joint in a manner that consistently results in discomfort is generally believed to lead to acceleration of cartilage destruction. Most patients with OA are comfortable with light to moderate exercise regimens that do not vary significantly. Enforced rest and exercise modification is different for each animal, but exercise extremes tend to exacerbate clinical signs. Swimming is a wonderful minimal load exercise, and in many parts of the country is available nearly year-round to our patients.

4. Pharmacologic Management

Analgesic and anti-inflammatory agents are the most common final component in the management of OA. However, there are some risks in using these agents, and one must consider all the possible ramifications prior to their usage. In principle, joint damage leads to an inflammation of the joint tissues, which may well result in mediator release and progressive joint destruction. In line with this reasoning, drugs which do interfere with inflammatory processes should reduce joint tissue damage, thus they may be regarded as being of prophylactic and therapeutic value. On the other hand, the main symptom of acute joint damage or acute clinical signs of OA is pain, which is a physiological signal to protect the joint from intensive and excessive use. The application of analgesic nonsteroidal anti-inflammatory drugs (NSAIDs) reduces this pain symptom and may, therefore, allow an overriding of this physiological warning signal. Under conditions in which NSAIDs are given and the patient then obviously overuses the limb, such as running a field trial, the use of NSAIDs is obviously destructive for the joint, although it enhances the physiological and psychological well-being. This is precisely why part of our whole treatment protocol specifically involves exercise modification.

Additionally, the concept of disease modification in OA is entering the picture of management. Compounds that are being developed to this end are known as disease-modifying osteoarthritis drugs (DMOAD) or structure modifying osteoarthritis drugs (STMOAD). Agents that have been previously called chondroprotective are now considered DMOADs or STMOADs. These drugs can have both effects on the inflammatory cascade and release of mediators and also direct effects on the target tissues (cartilage, bone, synovium).

Monoclonal Antibodies, specifically Anti-Nerve Growth Factor, are now making their way through clinical testing in both the dog and the cat. There is some exciting new clinical data supporting their use in our canine and feline OA patients.

Multimodal Therapy

There is a move towards greater use of a multimodal therapeutic approach to treat chronic pain in human medicine, and a multimodal approach has been suggested for the alleviation of chronic pain in veterinary species. The reason for suggesting a multimodal approach for the treatment of chronic pain results from what is now known about the changes induced in the central nervous system because of chronic pain—that is, the constant input of noxious signals from the periphery. Once generated, the noxious signal, in the form of an action potential, travels into the dorsal horn of the spinal cord. As in the periphery, the dorsal horn contains multiple transmitters and receptors, both those that have been identified, and putative ones, including peptides (substance P, calcitonin gene related peptide [CGRP], somatostatin, neuropeptide Y, galanin); excitatory amino acids (aspartate, glutamate); inhibitory amino acids (gamma-amino-butyric acid [GABA], glycine); nitric oxide; cholesystokinin; arachadonic acid metabolites; endogenous opioids; adenosine; and monoamines (serotonin, noradrenaline).

A huge breakthrough in the understanding of nociceptive processing came when it was found that the system was plastic - that inputs from the periphery could, via activation of a variety of receptors (principally the NMDA receptor), produce changes in the way nociceptive signals were processed in the spinal cord. The characteristics of this receptor are such that with repeated stimulation, it can produce a state of prolonged depolarization in the dorsal horn neuron. This cellular 'windup' is thought to produce the state of 'central sensitization' via the activation of a variety of second messenger systems, and the production of NO, eicosanoids and induction of immediate early genes. Central sensitization directly contributes to injury or disease induced pain. This is done by causing amplification of the signals and by altering processing of sensory information, such that previously non-noxious signals are now encoded as noxious. The NMDA receptor, however, appears to be central to the induction and maintenance of central sensitization. Also, the use of NMDA receptor antagonists would appear to offer benefit in the treatment of pain where central sensitization has become established (i.e. especially chronic pain). Opioid receptors are well known to be involved in pain states and the descending serotonergic system is known to be one of the body's endogenous 'analgesic' mechanisms.

Choosing the Right Combination of Therapies

How do we evaluate available information for its validity and applicability?
There are some basic questions that need to be answered for every type of study:
1. Are the results of the study valid?
2. What are the results?
3. Will the results help in caring for my patients?

It is important to understand the concept of a hierarchy of evidence. While every piece of evidence arising from clinical research is important, there are intrinsic quality differences that allow us to determine that some evidence is stronger and can help us determine the best care for our patients.

Weight Loss -What data is available to us?
There are several studies that provide data to support improved quality of life and lameness in the dog. The data for all is of moderate quality.

Nutritional Support (Functional Foods) - What data is available to us?
High N3 fatty acid ratio diets – Several clinical trials were identified using a diet high in N-3 omega (EPA and DHA) fatty acids. These studies identified assessing potential effects on clinical signs associated with OA in dogs. An overall rating of the strength of the evidence is moderate to high.

Exercise/Physical Therapy - What data is available to us?
There are limited studies that examine the effects of exercise on clinical dysfunction associated with OA in dogs. The studies suggest some improvements with different therapies. The data for all range from low to moderate quality and strength.

Pharmacologic Management – What data is available to us?
NSAIDs
Carprofen, Firocoxib, and Meloxicam – There are multiple studies to support the efficacy of carprofen, firocoxib, and meloxicam for the treatment of OA in dogs. There is a high level of confidence that the data presented regarding carprofen, firocoxib, and meloxicam is valid, and the conclusions of the studies are relevant to our patients. In a practical sense, we can have a high level of comfort that carprofen, firocoxib, and meloxicam are effective in treating the chronic pain and dysfunction associated with OA. In cats, one study was found, and it too demonstrated decreased pain and dysfunction with administration of meloxicam.

Others – There are several products that have one study (usually small numbers) that show some positive effects. These are difficult to evaluate and encompass into our daily practice, but they do warrant our attention and continued monitoring for additional data. Examples include intra-articular stem cell therapy, amantadine, elk antler velvet and the original study looking at glycosaminoglycan polysulphate (Adequan ®).

Amantadine, first recognized as an anti-viral agent, has gained popularity for the treatment of chronic pain disorders via inhibition of NMDA receptors. NMDA receptor activation, secondary to chronic stimulation of A delta and C fibers, is believed to be the primary component leading to “spinal windup”. One study compared the effects of adjunctive amantadine with meloxicam in a population of dogs with chronic OA refractory to NSAID therapy alone. Dogs treated with meloxicam in conjunction with amantadine had improved client-specific outcome measure scores and overall activity compared with the administration of meloxicam alone.

Tramadol is an opioid analgesic acting at the μ receptor while inhibiting serotonin uptake and norepinephrine reuptake. Tramadol also inhibits central pro-inflammatory cytokines and influences various neuronal cation channels while locally decreasing IL-6 and substance P. There is only one single study in dogs with OA evaluating the effects of oral tramadol in a blinded study using positive and negative controls. There was significant improvement noted in the positive control group (carprofen, 2.2 mg/kg twice a day) and tramadol (4 mg/kg 3 times a day) group compared with the placebo (administered 3 times a day) group using the canine brief pain inventory questionnaire. However, several other outcome measures in this study showed no improvement over placebo or baseline during the administration of tramadol. Thus, the limited data from this study is difficult to assess in terms of recommending tramadol use in dogs with

OA as a monotherapy.

Gabapentin, is structurally similar to the central inhibitory neurotransmitter GABA (gamma-aminobutyric acid). GABA is synthesized from glutamate, an excitatory neurotransmitter. During periods of chronic pain, there is up-regulation of glutamate and subsequent NMDA receptor activation with a relative decrease in GABA concentration. This results in loss of an endogenous feedback mechanism and an uninhibited nociceptive pathway. Though gabapentin's mechanism of action was initially assumed to be through GABAnergic transmission, the therapeutic effects are believed to be moderated through the alpha2 subunit of voltage-gated calcium channels resulting in central analgesia. To the author's knowledge, there are no available clinical or experimental studies evaluating the role of gabapentin in treatment of OA in dogs.

Biological Products

Anti-Nerve Growth Factor Antibody - As a member of the neurotrophin family, nerve growth factor (NGF) can bind the general neurotrophin receptor p75, as well as its high affinity cognate receptor, tropomyosin-related kinase (Trk)A. The NGF-TrkA pathway in particular appears to be critical in driving acute and chronic pain. Recently, canine and feline versions of anti-NGF antibodies have been developed. Two recent studies, where dogs with OA were treated with anti-NGF antibodies, yielded promising results. Additionally, there is one study in cats also showing promising results.

Current regenerative technologies for musculoskeletal injuries consist of three general categories. The first category is adult mesenchymal stromal cells, also known as mesenchymal stem cells (MSCs). MSCs are cells with high proliferative and self-renewal capabilities, are adhesive to plastic surfaces, show specific cell surface proteins, and have potential to differentiate in at least three lineages, including bone, cartilage, and adipose tissue. The second category is plasma-based products, such as Platelet-rich plasma (PRP). PRP consists of a pool of signaling proteins including growth factors, cytokines, and other adhesive proteins involved in healing mechanisms. The list is not exhaustive. Autologous and, more recently, allogenic stem cell therapies have shown some limited positive results in clinical trials when given to dogs with OA. Additionally, studies of limited size and scope have also shown initial positive results for autologous plasma/platelet treatments in dogs with OA. There is no clinical data available on conditioned culture media (CM).

Primary Chondroitin and Glucosamine products – There are conflicting results in limited clinical testing. It is difficult to say these products are effective or not.

Green-lipped Mussel Preparation – Three very small trials were identified using a compound with the main ingredient green-lipped mussel (Perna canaliculus) for the treatment of OA in dogs. While all studies subjectively showed a positive effect, the quality rating for some of the studies suggested some uncertainties exist relating to the scientific quality

Several other products or procedures show negative or no improvement in chronic pain. Again, these are difficult to evaluate but they may have additional studies that do show a positive

effect with larger numbers or different study designs. Thus, we might want to monitor the literature for additional data. Examples of this would include extracorporeal shock wave therapy and gold bead therapy.

Feline Osteoarthritis

Clinical Presentation

There are several reasons for under-appreciation of the clinical significance of DJD. First, the clinical manifestations of the problem in cats are more difficult to identify. Cats with DJD do not act like dogs affected by the same disease. Cats are not subject to the wide range of juvenile joint dysplasia conditions that result in a high occurrence of secondary osteoarthritis in young pure breed dogs, making this a very common finding in this species. Mobility disorders are much more readily identified in dogs where the owner is characteristically present while the animal is exercising and able to recognize lameness or changes in the activity pattern. Lastly, the radiographic appearance of an osteoarthritic joint in the cat is much subtler than the dog with less obvious proliferative osteophyte formation. This may result in the problem being overlooked or dismissed as clinically insignificant. Very little work on the assessment of DJD joint disease pain has been performed in cats. However, it appears from early work that an approach, similar to that in dogs, is likely to be most successful. That is, owners need to be centrally involved in the process. The difficult part of assessment of DJD pain in cats is that the activities that are altered by osteoarthritis are less fully understood than in dogs. A recent study of 28 cats with osteoarthritis showed that overt lameness was not the most common clinical feature. Instead, features like jumping up, jumping down, height of the jump, general movement, "grumpiness" on handling, and seeking seclusion are likely to be activities and behaviors that should be followed.

Diagnosis

Given the aforementioned discussion, how do we develop a methodology to diagnose DJD in cats with high sensitivity and specificity? First, the possibility of the diagnosis must be on the rule out list for any middle to older age cat presenting for changes or decreases in activity, or behavioral changes. Careful and complete history and physical examination is a must. These activities are time consuming and often overlooked in a busy day of seeing patients. If lameness or stiffness are noted, or it was reported that the cat has altered jumping activities (both height and frequency), then DJD must be very high up on the differential diagnosis list. On physical examination, pain or decreased range of motion in a joint are classic markers for DJD. If there is suspicion of DJD, radiographs are the next diagnostic test to be considered. If no specific joint can be detected in the forelimb, consider taking films of the elbow first, then shoulder and finally the carpus. If no one joint can be singled out in the hind limb, consider taking films of the hips, followed by the tarsus and stifle. Also consider radiographs of the thoracolumbar spine. While the significance of radiographic findings of DJD have been questioned in the past because of the lack of associated signs, it should be argued that the lack of correlation is due to the inability of the clinician and owner to appreciate the signs being shown. While three different studies found low correlation, the reasons for this are most likely that feline gait/lameness and mobility dysfunction is much more difficult to identify and that either signs were overlooked in the retrospective populations—or that overt lameness was not one of the main manifestations of the disease in cats. As owner assisted outcome measure tools become better defined and validated, it

is very likely that the correlation between radiographic changes and clinical changes will dramatically improve.

There is a wonderful article in
Feline Chronic Pain and Osteoarthritis
By Beatriz P.Monteiro
Veterinary Clinics of North America: Small Animal Practice
Volume 50, Issue 4, July 2020, Pages 769-788

I highly recommend you obtain this article if you are at all interested in treating cats with OA.

Treatment

Weight loss – No specific clinical trial data is available to evaluate but significant data on obesity strongly shows the benefits to weight loss.

Dietary Management - As with the dog, high N-3 fatty acid ratio diets are starting to become available, yet data is limited to strongly support their use. While these diets may be effective, none has been evaluated to any significant degree. Part of the reason for the lack of evidence-based information about treatment of feline DJD-associated pain is the lack of validated outcome measures, and partly because of a lack of understanding of how to diagnose the disease, as well as lack of understanding about its causes.

Physical Therapy – The use of "Environmental enrichment" to promote physical and mental stimulation in cats has started to become more commonplace. Traditional physical therapy and exercise modification is often difficult in cats given their personalities.

Pharmacologic Therapy –

NSAIDS - Currently, only NSAIDs have data showing a beneficial effect (pain alleviating and mobility enhancing) in painful feline DJD (Meloxicm and Robenocoxib). Meloxicam has a long-term label claim in Europe. Concern about use of NSAIDs in cats, especially on a chronic basis, is generally centered around the perception that NSAIDs are metabolized more slowly in cats than dogs. Most NSAIDs are cleared from the body through hepatic metabolism (often primarily glucuronidation) and then biliary and/or renal excretion of the resultant polar metabolites. Given the known propensity for reduced glucuronidation of drugs in cats compared with other species, differences in NSAID disposition between cats and other species might be expected. Aspirin, and carprofen have relatively prolonged elimination half-lives in cats compared with dogs. In contrast, similar or even reduced drug elimination half-lives are observed in cats, compared with dogs, for drugs cleared by oxidative enzymes, including piroxicam and meloxicam. Presence of alternate metabolic and non-metabolic pathways for drug elimination may compensate for slowed glucuronidation of NSAIDs in the cat. Chronic painful disease demands repeated administration of analgesic drugs, and there is little current information on the pharmacokinetic (PK) and toxic effects of repeated administration of NSAIDs in cats. A good, rational discussion of the use of NSAIDs in cats was published in the Journal of Feline Medicine and Surgery (2010) by Sparkes et al. It is available free online.

Tramadol - There is one study that has been shown to decrease central sensitization and to improve motor activity and global QoL in cats with OA (2–4 mg/kg every 12 h, for 5–19 days). While the data is limited, it does seem to show the treatment to be safe and provides some level of analgesia. However, oral tramadol is bitter, making it unsuitable for many cats. Remember, with all oral medications in cats, treatment is unacceptable if there is forced pilling chronically. These activities can impair owner-cat bond and all that is entailed with that daily potential physical and mental trauma.

Gabapentin – One study showed improved owner-identified impaired activities (CSOM) that in a small group of client-owned cats, treatment with gabapentin at 10 mg/kg, every 12 h for 2 weeks when compared with placebo. However, cats receiving gabapentin showed decreased motor activity, likely because of sedation. The data is limited in support of its use, but gabapentin has gained popularity in the management of feline chronic and neuropathic pain.

Biologics -

Feline-specific anti-NGF antibody is a promising therapy in management of chronic pain. Nerve growth factor contributes to peripheral and central sensitization, and its concentrations are increased in chronic painful conditions including OA. In one study in cats with OA, a single treatment with feline-specific anti-NGF antibody administered subcutaneously increased motor activity and improved CSOM scores up to 6 weeks. There are additional studies being conducted and stronger data may be soon available to veterinarians.

NSAIDs in the Management of Chronic Pain:

Steven C Budsberg DVM, MS, DACVS
Professor of Surgery
University of Georgia
Email- Budsberg@uga.edu

Introduction

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) are one of the most commonly used classes of drugs for managing chronic pain in small animals. There are several reasons for the dramatic increase in NSAID use in companion animals. There is now a better understanding of the need to manage acute and chronic pain in small animal medicine. Pain control is a very important mission for the practicing veterinarian. NSAIDs can be used to alleviate acute pain, either traumatic or surgically induced, and for chronic pain such as osteoarthritis and thud NSAIDs provide an effective means to accomplish this goal. FDA approved NSAIDs are very safe drugs; with only a small percentage of patients experiencing serious complications. However, these adverse events have achieved significant proportions based on the fact that so many dogs and cats are taking these agents each year—thus a small percentage becomes a very large number. Efficacy and toxicity are often individualistic and monitoring of each animal is mandatory in all cases. Choosing and monitoring NSAID usage is important; there are no definitive answers as to how this should be done. First it is wise to use products with a history of extensive clinical use. Use only one NSAID at a time and ensure correct dosing. Review the treatment plan frequently, and change to alternative NSAIDs if there is a poor response to therapy. Observe for potential toxicity as soon as administration is begun with increased vigilance and monitoring of high-risk patients. If indicated, establish renal and hepatic status of the patient prior to NSAID administration.

Mechanism of Action

Eicosanoids, which include the compounds known as prostaglandins, are derived from arachidonic acid (AA). It is the ability of NSAIDs to interfere with eicosanoid synthesis and the subsequent alteration of different physiologic systems that explains the numerous effects seen in the body with NSAID administration. Significant portions of the analgesic and anti-inflammatory clinical effects seen with NSAID administration are related to the inhibition of the COX enzyme isoforms. The COX enzymes initiate a complex cascade that results in the conversion of polyunsaturated acids to prostaglandins and thromboxane. With regard to pain, prostaglandins, primarily PGE2, contribute to the inflammatory response by causing vasodilation and enhancing the effects of other cytokines and inflammatory mediators. The production of PGE2 at various sites of inflammation appears to be mediated primarily by COX-2. Thus, when an inflammatory event occurs within the tissue, COX-2 enzyme production is induced, followed by an increase in prostaglandin concentrations. The selective inhibition of certain prostaglandins primarily produced by COX-2 should allow for the therapeutic analgesic and anti-inflammatory effects while greatly diminishing the unwanted side effects caused by COX-1 inhibition. However,

complete COX-2 inhibition is detrimental to many normal physiologic functions including the healing of gastric ulcers.

Site of Action

Data now support the concept that NSAIDs act on both the peripheral tissue injury site as well as at the level of the central nervous system (CNS). They inhibit the peripheral COX-2 enzyme to block the formation of prostaglandins such as PGE2 and PGI2, which function to dilate arterioles and sensitize peripheral nociceptors to the actions of mediators (e.g., histamine and bradykinin), which produce localized pain and hypersensitivity. PGE2 produced by COX-2 plays a pivotal role in sustaining acute pain sensation by increasing nociceptor cyclic AMP, which decreases the nociceptor threshold of activation. Centrally, COX-2-mediated prostaglandins such as PGE2 are involved in spinal nociception and central sensitization. COX-2 is expressed in the brain and spinal cord and is upregulated in response to traumatic injury and peripheral inflammation.

Recently there has been the introduction of an EP4 receptor antagonist to decrease the pain and inflammation in OA. Prostanoid EP4 receptors have been extensively implicated in mediating hyperalgesia and allodynia. All EP subtypes are expressed in sensory neurons, but EP4 may be regarded as the most important because it causes sensitization and is exclusively expressed in a subset of primary sensory dorsal root ganglia, which increases in subchronic inflammation. Data also suggests that PGE(2) inhibits proteoglycan synthesis and stimulates matrix degradation in OA chondrocytes via the EP4 receptor. Targeting EP4, rather than cyclooxygenase 2, could represent a future strategy for OA disease modification. Thus EP4 antagonists binds the EP4 receptor and blocks PGE_2 from exerting its biological effect. By blocking the binding of PGE_2 to its receptor, the signaling pathway for pain and inflammation is interrupted.

Clinical Applications

NSAIDs can be used to relieve pain in a variety of clinical settings. Efficacy and toxicity are often individualized, and individual monitoring is mandatory. There are numerous initiating pathways that produce pain that are not fully understood, and it would be naïve to think that all pathways will react in the same manner to different drugs. In addition, the heterogeneity of the patient response to a given NSAID in terms of efficacy and toxicity may be accounted for by slight variations in genetic expression or gene polymorphism of the COX enzymes known as the "COX continuum."

Choosing and Monitoring the Use of Nonsteroidal Anti-inflammatory Drugs

- Use products with history of clinical experience and good safety profiles.
- Use only one NSAID at a time, and ensure adequate dosage.

- Adapt therapy to suit patient requirements. Begin with the recommended dose for an extended period of time (at least 10 to 14 days) in animals with chronic pain.
- Avoid NSAIDs in patients with known contraindications to their use.
- Observe for potential toxicity. Increased vigilance and monitoring are required for at-risk patients. If indicated, establish renal and hepatic status of the patient before NSAID administration.

Contraindications

- Patients receiving any type of systemic corticosteroids.
- Patients receiving concurrent NSAIDs.
- Patients with documented renal or hepatic insufficiency or dysfunction.
- Patients with any clinical syndrome that creates a decrease in the circulating blood volume (e.g., shock, dehydration, hypotension, or ascites).
- Patients with active GI disease.
- Trauma patients with known or suspected significant active hemorrhage or blood loss.
- Pregnant patients or in females in which pregnancy is being attempted
- Patients with significant pulmonary disease. (This may be less important with COX-2–specific drugs).
- Patients with any type of confirmed or suspected coagulopathies. (This may be less important with COX-2–specific drugs).

Adverse Events

The most common problems associated with NSAID administration to dogs and cats involve the gastrointestinal (GI) tract. Signs may range from vomiting and diarrhea, including hematemesis and melena, to a silent ulcer which results in perforation. The true overall incidence of GI toxicity in dogs or cats treated with NSAIDS is unknown. Concurrent administration of other medications (especially other NSAIDs or corticosteroids), previous GI bleeding, or the presence of other systemic diseases may contribute to adverse reactions. Hepatotoxicosis caused by NSAIDs is generally considered to be idiosyncratic. Most dogs recover with cessation of treatment and supportive care. Renal dysfunction may occur with NSAID administration as a consequence of prostaglandin inhibition. Renal prostaglandin synthesis is very low under normovolemic conditions. When normovolemia is challenged, prostaglandin synthesis is increased and important to maintaining renal perfusion. NSAID use must be considered very

carefully in hypovolemic or hypotensive animals. This is especially important to remember with the increasing use of perioperative NSAIDs for pain management.

Other tissues that may be affected by NSAIDs are cartilage, bone and the cardiovascular system. Studies have demonstrated a variety of effects on proteoglycan synthesis when chondrocytes or cartilage explants are incubated with an NSAID in vitro. The most pronounced effects have been seen in chondrocytes from osteoarthritic joints, although a lesser effect has been demonstrated on normal cartilage. Aspirin is uniformly reported to cause inhibition of proteoglycan synthesis; conflicting data exist for other NSAIDs, such as etodolac, showing both potential negative and positive effects; and there is a final group, including meloxicam, piroxicam, tepoxalin, and carprofen, in which no effect or even some increased synthesis of proteoglycan has been noted. The significance of these in vitro findings remains unclear, and the clinical significance of these data in the clinical setting with naturally occurring disease is unknown. In regard to bone healing, it is interesting to note that prostaglandins also play an important role in bone repair and normal bone homeostasis. Experimental studies support the hypothesis that both nonspecific and specific COX inhibitors (COX-1 sparing) do impair bone healing. These statements are based on rabbit, rat, and mouse induced fracture models that show that COX-1–sparing agents do alter bone healing. However, the most recent data confirm that after cessation of NSAIDs, fracture healing returns to its normal rate, and therefore judicious use of postoperative NSAIDs can be recommended. Stated another way, any potential adverse effects must be weighed against potential benefits that include but are not limited to improved analgesia and earlier return to function (both mobilization of the limb and the patient and weight bearing), and data support use of NSAIDs in the immediate postoperative period as long as the administration is not continuous for several weeks. Finally, NSAIDs can occasionally have significant cardiovascular effects. Nonselective NSAIDs inhibit the platelet COX-1 enzyme and cause a significant decrease in the amount of thromboxane A2 (TXA2) produced by activated platelets. Thromboxane is an important promoter of platelet aggregation in most dogs and is released by activated platelets to recruit additional platelets to the site of vessel injury. Thromboxane is also a potent vasoconstrictor. A decrease in thromboxane release can result in prolongations of primary hemostasis. COX-1–sparing (COX-2–selective inhibitor) drugs do not have this effect on thromboxane production and likely do not clinically affect primary hemostasis, depending on the study methodology. The actions of thromboxane are balanced at the vessel level by the presence of prostacyclin (PGI2), which is produced by COX enzymes in the vascular endothelial cells. PGI2 is a strong inhibitor of platelet aggregation and also results in vasodilation. In the presence of endothelial inflammation (such as that caused by atherosclerotic plaques), the expression of COX-2 in the endothelial cells increases and may produce the majority of prostacyclin in that area. When COX nonselective NSAIDs are administered, the expression of both thromboxane from platelets and PGI2 from endothelial cells is decreased, preserving the balance. In certain circumstances of endothelial inflammation (e.g., with atherosclerosis), specific COX-2 inhibitors may decrease the endothelial production of PGI2 (mainly from COX-2) without a concomitant

decrease in platelet thromboxane (produced only by COX-1), and consequently may result in the development of a hypercoagulable state.

Recap of specific NSAIDs

The approved NSAIDs available to the clinician vary considerably around the world. It is very important for practitioners to remember that the clinical response to a particular drug is quite individualistic. Dogs may respond favorably to one product and not another, so if a NSAID is indicated in a case and the first product used does not achieve a positive clinical response, do not forsake NSAIDs but try a different product.

Carprofen

Carprofen is approved, both in oral and injectable formulations, to treat pain and inflammation associated with osteoarthritis (OA). Carprofen has improved limb function in clinical trials of dogs with naturally occurring osteoarthritis. Three long-term studies (84 days and 120 days) found that carprofen was well tolerated and subjectively dogs appeared to improve over the treatment period. In certain countries, a single injectable dose in cats is approved for pain. While there is ample data to support single dose use for perioperative pain, repetitive dosing in cats is not recommended until additional safety and efficacy data is produced with multiple dose protocols.

Deracoxib

Deracoxib is approved in an oral formulation in dogs for treatment of pain and inflammation associated with OA and postoperative pain associated with orthopedic surgery. It has been demonstrated to provide effective relief of pain in clinical osteoarthritis trials in dogs in a study which has never been published in a peer reviewed journal but has been presented in abstract form. The same situation is present with a study showing effectiveness in relieving pain related to orthopedic surgery.

Firocoxib

Firocoxib is approved, as an oral formulation, with an indication for the management of pain and inflammation associated with OA in dogs. Clinically it has been shown to improve limb function in dogs with osteoarthritis. Clinical trials suggest that firocoxib may have some superiority based on owner and veterinarian subjective evaluations with regard to lameness resolution when compared to carprofen and etodolac in dogs with OA. Long-term dosing of firocoxib showed continued improvements over the year of treatment.

Mavacoxib

Mavacoxib is approved by the European Union as an oral formulation in dogs for treatment of pain and inflammation associated with OA. Mavacoxib is a long acting agent with an approved

dosing regimen consists of a loading dose repeated at 14 days and thereafter at dosing intervals of 1 month.

Meloxicam

Meloxicam has been approved for use in dogs for the control of pain and inflammation associated with OA and is available in oral, transmucosal oral mist, and parenteral formulations. With the amount and quality of the published data available for use of meloxicam for management of acute postsurgical and well as chronic OA pain in dogs.

Meloxicam is approved for use in cats, but that approval is limited to a single dose to control pain and inflammation associated with orthopedic surgery, ovariohysterectomy and castration in the US. Long term use of meloxicam for the treatment of musculoskeletal pain in cats is approved in several countries. Use in cats from 5 days to indefinite dosing to provide analgesia for locomotor disorders including OA has been described at several different dosing levels (0.01 to 0.05 PO mg/kg) daily.While these data exist, clinical efficacy is supported by data generated from studies using the 0.05 mg/kg dose PO every 24 hours. At lower dosing regimens (0.01 to 0.03mk/kg PO q 24 hours), meloxicam has been shown to be well tolerated and safe in cats including cats with chronic renal dysfunction.

Robenacoxib

Robenocoxib is approved for only dogs or both dogs and cats depending on the country. Indications in the dog are for treatment of pain and inflammation associated with orthopedic or soft tissue surgery as well as the treatment of pain and inflammation associated with chronic osteoarthritis (depending on the country).

In the cat, approved indications (depending on the country) may include treatment of postoperative pain and inflammation associated with orthopedic and soft tissue surgeries as well as the acute pain and inflammation associated with musculoskeletal disorders. Length of approved treatment times for robenacoxib in the cat varies from 3 to 11 days.

Washout period between NSAIDs.

A question that is commonly asked by clinicians is whether or not a washout period is needed when switching NSAIDs. Several sources, including crowd sourcing websites, conference proceedings, pharmaceutical company promotional materials as well as journal articles, have advocated a washout period of varying lengths (1 to 7 days) when changing NSAIDs for presumed lack of efficacy. These recommendations are not based on clinical data but rather are derived from extrapolations of pharmacokinetic data and conservative speculation. There are several different situations that need to be addressed when discussing how to switch NSAIDs in our clinical patients. The first situation is a switch after a single dose of a perioperative parenteral NSAID (e.g. carprofen or meloxicam) followed by an oral NSAID the

following day. The only data to use in this situation is a study of normal healthy dogs that were given parenteral (sub-cutaneous) carprofen followed by deracoxib orally 24 hours later and repeat for four days. This was one arm of the study and when compared to continuous carprofen (sub-cutaneous and oral) or placebo there were no differences in clinical findings or gastric lesions. Thus from this limited data it appears that it may be safe to switch from a single injection of one drug to an oral formulation the next day if using another product. However, without testing injectable meloxicam and the other oral products with meloxicam or carprofen one cannot be definitive about these treatment recommendations.

The second situation is switching NSAIDs for perceived lack of a response and perceived efficacy. This is a difficult question that often faces a clinician and here is where there is a significant variation in recommendations. Many reports discuss waiting 5 half-lives of the first drug before initiating the second drug. The only clinical data which may shed light on this situation is a report of switching to firocoxib from another NSAID, which showed no increase in documented side effects whether firocoxib was started the next day up to day seven from stopping the original drug. These data would again suggest that a washout period is not necessary, but most clinicians follow the recommendation discontinuing a NSAID for 1 to 7 days before initiating another drug. The final situation is transitioning to or from aspirin. If aspirin is the initial drug it has been recommended that a minimum of a seven day washout period be followed before starting another NSAID, to provide time for platelet regeneration due to aspirin's irreversible effects on platelets. There is no clinical data to support this recommendation. The second situation could occur if for some reason a dog is on a product that is COX-1 sparing (a primary COX-2 inhibitor) and is then changed to aspirin, a seven day washout is recommended due to the gastric adaptation and production of aspirin-triggered lipoxins(ATLs). The concern here is that when a patient is on aspirin, ATLs are produced and have been shown to exert protective effects in the stomach by diminishing gastric injury most likely via release of nitric oxide from the vascular endothelium. However, concurrent administration of COX-1 sparing drugs with aspirin results in the complete inhibition of ATLs and can potentially cause significant exacerbation of gastric mucosal injuries. It is important to remember that the formation of ATLs has yet to be proven in the dog.

Saturday, July 10, 2021

Anaesthesia Crises

Dr. Erik Hofmeister

Outline

- Common problems
- Crisis philosophy
- Actual crises

Waking Up

- Anesthetic concentration too low
- Not connected to circuit
- Oxygen not on
- Vaporizer not on
- Vaporizer empty

Approach to Waking Up

- Immobilize patient, prevent from chewing tube
- Re-anesthetize patient
  - Injectable, inhalant
- Is patient connected? Gasses on? Vaporizer full?

Waking Up Management

- Increase gas concentration
  - Increase O_2 flow
  - Increase vaporizer setting
  - Increase ventilation
  - Avoid O_2 flush valve!
- Balanced anesthetic technique
  - Add opioid, sedative

Tachycardia

- Pain
- Getting light
- Hypercarbia
- Hypoxemia
- Drugs
- Hypovolemia

Anemia, cardiac disease, arrhythmias, hypoglycemia, hyperthermia, acidosis, endotoxemia

Approach to Tachycardia

- Is the patient comfortable? Is it asleep?
- Is the patient's volume OK?
- Is the patient on relevant drugs?
- Is the patient hypercarbic/hypoxemic?
- Is it too high? Symptomatic treatment needed?

Tachycardia Treatment

- Painful: hydromorphone 0.05mg/kg IV
- Light: increase gas, diazepam 0.2mg/kg IV
- Hypovolemic: Test bolus of 1 hour's fluids
- Drugs: stop CRIs and watch
- Hypercarbia: ventilate
- Hypoxemia: see hypoxemia
- Symptomatic: B-blockers
  - Esmolol, propranolol

Hypercarbia

- Ventilator error
- User error
- Endobronchial intubation
- Increase metabolism
- Machine fault

Approach to Hypercarbia

- Are the ventilator settings and inspiratory pressure appropriate?
- Is the patient's chest moving normally?
- Is the ventilator attached correctly? Are there any leaks?
- Is there good air flow on both sides of chest?
- Is patient hyperthermic, red, tachycardic, or had an abrupt increase in $ETCO_2$?

Hypocarbia

- Cardiac arrest
- Ventilator error
- User error
- Hypothermia
- Machine fault
- Nonrebreather

Approach to Hypocarbia

- BP/HR!!!!!
- Are ventilator settings appropriate? Check inspiratory pressure
- Check monitor/machine

Hypoxemia

- Decrease inspired O_2
- Hypoventilation
- Right to left anatomic shunt
- V/Q mismatch
- Diffusion impairment

Approach to Hypoxemia

- Obtain blood gas
- Check machine
- Is patient hypoventilating? If so, how severely?
- Did airway pressure or ventilator settings change recently- change in compliance?
- Surgeons doing anything funny?

Hypoxemia Treatment

- Make sure getting O_2
- Increase tidal volume (peak inspiratory pressure to 25-30 cm H_2O if needed)
- Change position (dorsal to lateral, tilt head up, lateral to sternal)
- Bronchodilators
  - Aminophylline 5mg/kg

Hypotension

- Bradycardia
- Poor venous return
- Poor contractility
- Vasodilation

Approach to Hypotension

- Is patient bradycardic?
- Is patient volume depleted?
- Does patient have drugs/disease which would impact contractility or vasodilation?
  - Inhalants, endotoxemia, acepromazine, thiopental, propofol, cardiac disease, lidocaine/bupivacaine OD, morphine IV, etc.

Hypotension Treatment

- Bradycardia – give atropine, glycopyrrolate
- Volume depleted – give fluid bolus of 1 hour's fluids
- Poor contractility – give inotrope
  - Dobutamine/dopamine 5µg/kg/min
  - Ephedrine 60 µg/kg

Vanished Doppler

- Doppler fault
- Technical fault – slipped probe
- User error
- Cardiac arrest
- Closed pop-off valve

Approach to Vanished Pulse

- Patient alive?
- Pop-off open?
- Doppler on/volume up?
- Probe correct position?

Bradycardia

- Drugs
  - NMBA reversal drugs, lidocaine, fentanyl, medetomidine, inhalant OD, etc.
- Disease
- Hypothermia

Approach to Bradycardia

- Is patient hypotensive?
- Is bradycardia rapidly evolving?
- Is HR really low?

- Treatment: atropine 0.02mg/kg IV or glycopyrrolate 0.005mg/kg IV

Hypertension

- Pain
- Getting light
- Hypercarbia
- Hypoxemia
- Drugs

Cardiac disease, arrhythmias, hypoglycemia, hyperthermia, acidosis, endotoxemia

Approach to Hypertension

- Ruleout per tachycardia
- Is it too high?
- Mechanical error?

Hypertension Treatment

- Treat underlying disease/problem
- Symptomatic treatment:
  - Increase inhalant anesthesia setting
  - Administer acepromazine 0.05mg/kg IV
  - Administer nitroglycerine or nitroprusside

Prolonged Recovery

- Drug OD
- Hemorrhage
- Cerebral ischemia
- Hypoglycemia
- Liver disease
- Renal disease
- CNS disease
- Young/old/thin/debilitated

Approach to Prolonged Recovery

- Was there profound hypotension during anesthesia?
  - Assess pupils, vision
  - Reverse drugs
  - Treat for cerebral edema- mannitol 0.5g/kg IV
- Check PCV/BG
- Reverse drugs
  - Naloxone 0.01mg/kg IV
  - Flumazenil 0.01 mg/kg IV

Hypothermia

- Prolonged anesthesia
- Abnormal heat production
- Abnormal temperature regulation (CNS disease, endotoxemia)

Approach to Hypothermia

- Concurrent problems? (bradycardia, hypotension, prolonged recovery)
  - Treat aggressively
- Prevention is easier that treatment
- Slow rewarming better than rapid
- Warm air blower, radiant heat lamp, warm water blanket, towels

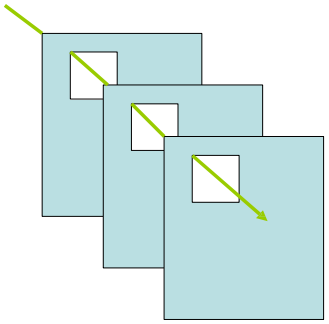
Hyperthermia

- Heat stroke (exogenous heat- OD from forced air warming, heat lamp, surgery light)
- Febrile (disease-based)
- Malignant hyperthermia

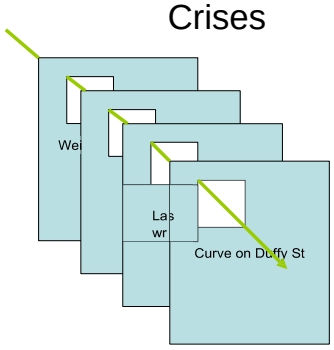
Approach to Hyperthermia

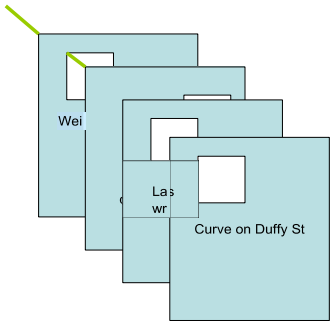
- Concurrent abrupt increase in CO_2/HR?
  - Malignant hyperthermia likely
- Turn off warming devices
- Treat underlying disease
- Active cooling if necessary (careful if under GA)

Crises

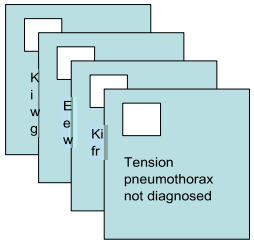


Crises





Iatrogenic Arrest



Anaesthetic Crises

- Inadvertent drug administration
- Loss of airway
- Iatrogenic cardiac arrest
  - Closed pop-off valve
- Profound or persistent hypotension
- Biting animal

Crisis Management

- DON'T PANIC
- Get help
- Figure out who's in charge
- Start to do what you can

Inadvertent Drug Admin

- Withdraw what you can
- Precipitate if possible
- Solution to pollution is dilution
- Reversal of drugs if necessary/possible
  - Naloxone 0.01mg/kg
  - Flumazenil 0.01mg/kg
  - Doxapram? 1.25 mg/kg
- Be honest with owner/team

Drug Overdose

- Step A: Determine problem exists
- Step B: Determine how severe problem is
- Step C: Drug reversible?
- Step D: Supportive care

Loss of Airway

- Give oxygen by other methods
- Attempt orotracheal intubation
  - Sylet, multiple lights, rigid ET tube
- Needle tracheotomy
- Wire stylet through trachea
- Tracheotomy

Iatrogenic Cardiac Arrest

- CHECK MACHINE
- Check syringes before injection
- Be prepared
- Begin CPCR efforts
  - ABCDE
- When in doubt, turn off gas

Profound Hypotension

- EARLY RECOGNITION
- DON'T muddle with technology
- Turn off gas
- Administer fluids, inotropes, volume expanders (hetastarch, hypertonic saline)
- Correct underlying problems (acidosis, hypocalcemia)
- Tell surgeons to hurry it up

Biting Animal

- Let animal go if possible – don't get bit!
- Get towel/rabies pole/squeeze cage
- Sedate if possible according to disease
- Protocols:
  - Morphine 0.5mg/kg, diazepam 0.5mg/kg, ketamine 10mg/kg, atropine 0.02mg/kg
  - Morphine 0.5mg/kg, medetomidine 20µg/kg

Crises Debriefing

- What happened?
- How did it happen?
- Why did it happen?
- How do we prevent it from happening again?

NOT: Whose fault?

Crisis Causes

- Rushing
- Unskilled/unknowledgeable
- Patient

Summary

- Learn common differentials
- Figure out what's going on and why
- Begin to treat appropriately
- If no response- change treatments! (see House MD)

Summary

- Crises occur as a synergy of events
- Crises can be prevented or mitigated by education, preparation, and slowing down
- In a crisis, take a deep breath, get help, figure out who's in charge, and begin

Slide 1

Communicating Anesthesia Risk to Clients

Dr. Erik Hofmeister

Slide 2

Learning Objectives

- Participants will understand risk factors for anesthesia
- Participants will know risk rate for adverse events
- Participants will understand concerns clients have
- Participants will be able to communicate with clients about the risk of anesthesia

Slide 3

Outline

- Introductions
- Literature review – anesthesia risk factors
- Client concerns
- Effectively allaying concerns
- Clinical recommendations

Slide 4

Introductions

Slide 5

Participant Poll

- What is your primary role/background?
  - Private practice veterinarian
  - Private practice technician
  - Specialty practice veterinarian
  - Specialty practice technician
  - Other

Slide 6

Participant Poll

- Has a client expressed concern to you about anesthesia in the past month?
  - Yes
  - No

Slide 7

Literature Review

- 1.7 in 1000 dogs
- 2.4 in 1000 cats
- ASA Status
  - 0.5 in 1000 dogs vs. 13 in 1000 dogs
  - 1.1 in 1000 cats vs. 14 in 1000 cats
- Most commonly postoperative

Slide 8

Literature Review

- Cats
  - Emergency
  - Smaller size <2kg
  - Larger size >6kg
  - Intubated
  - IV fluids

Slide 9

Literature Review

- Dogs
  - Emergency procedure
  - No physical exam
  - Underweight
  - Anemia
  - (Xylazine)

Slide 10

Literature Review

- Age?
  - Equivocal – some studies indicate yes, others no
  - May be due to concurrent illnesses captured by other variables (ASA)
  - Occult disease/conditions

Slide 11

Participant Poll

- What do clients express most concern with regards to anesthesia?
  - Death
  - Pain
  - Vomiting/nausea
  - "Unsettled"/behavioral
  - Drug reactions
  - Generalize/uninformed
  - Other

Slide 12

Client Concerns

- Death
  - Normal, healthy patients particularly unlikely
  - Unhealthy, emergency patients more likely (but still ~1 in 100)
  - If unexpected, can be most unsettling
- Pain
  - Clients assume patient will be comfortable
  - Some may worry about this in deciding whether to do a procedure or not
  - Postop pain often unwitnessed by client or misidentified

Slide 13

Client Concerns

- Vomiting/nausea
  - Serious problem in human anesthesia – if client had surgical procedure, may relate
  - If patient has a history of GI disease, may express concern
- Unsettled/behavioral
  - Worried about separation from owner
  - History of poor recovery from anesthesia
  - History of abnormal behavior after discharge

Slide 14

Client Concerns

- Drug reactions
  - "Sensitive" to anesthesia
  - 2008: Breeds with a sensitivity to anesthesia that were mentioned by > 30% of Web sites
  - A friend told them "x" or their breeder told them "y"
  - My concerns:
    - Sighthounds with thiobarbiturates (no longer used)
    - Boxers with acepromazine
    - MDR1 mutation dogs with acepromazine or butorphanol
    - Brachycephalic breeds
    - History of a problem

Slide 15

Client Concerns

- Generalized
  - "I just worry" or "I heard anesthesia is dangerous"
  - Asking questions may help improve understanding
  - Often worried because of unknown – providing information helpful
- Age
  - "I've heard it's not safe to anesthetize old dogs."
  - Share experience managing these cases
  - Evidence is not conclusive that this is true

Slide 16

Allaying Concerns

- Be proactive
  - Explain your process for managing pain and monitoring patient
  - Sedation, analgesia, induction, monitoring, recovery
- Solicit questions
  - Consider "what questions do you have" vs. "do you have any questions?"
  - Can ask specifically about anesthesia, "what questions do you have about anesthesia for Fluffy?"

Slide 17

Allaying Concerns

- Unsolicited questions
  - May interrupt your 'flow'
  - "I understand you have questions about anesthesia. Let's discuss the rest of Fluffy's care and we will talk about that at the end."
- What if you don't have answer?
  - "That's a good question. We don't routinely give anti-nausea medications, because we haven't experienced that as a problem. I will talk to my colleagues and get back to you about this."

Slide 18

Allaying Concerns

- Be attentive and listen
- Reflective listening
  - "I understand you're worried about Fluffy's pain."
  - "So last time she went under anesthesia, she wasn't right for 3 days."
- Can ask for clarity
  - "What are you most worried about with Fluffy's anesthesia?"

Slide 19

Allaying Concerns

- Validate, do not 'blow off'
  - "I know how worried you are about Fluffy not making it through anesthesia. I am not very worried, because she is a healthy dog and we have a technician dedicated to monitoring her throughout. Healthy dogs have a very low risk of death- only 1 in 2,000. If she has problems, we will know about them immediately and take steps to correct them."

Slide 20

Allaying Concerns

- Validate
  - "I know how worried you are about Fluffy not making it through anesthesia. It is concerning, because she has a serious disease and that makes anesthesia more challenging. With sick dogs, the chances are about 1 in 100. However, we have to proceed to try and make her better. We have a technician dedicated to monitoring her throughout. If she has problems, we will know about them immediately and take steps to correct them. Nonetheless, it is possible she will not make it despite our best efforts, but we will do everything we can for her."

Slide 21

Allaying Concerns

- More validation
  - "I understand the breeder said not to use ketamine with this line. We have other drug options available, so will be able to avoid it. I will make a note in the chart."
  - "I understand the breeder said not to use ketamine with this line. Ketamine is a routine part of our protocol and it is the one we are most comfortable and familiar with. I feel it is best to maintain our typical routine, as that will be the safest for Fluffy. If she experiences any problems with it, we will manage them. We routinely use ketamine in Fluffy's breed without problems."

Slide 22

Allaying Concerns

- Be careful to avoid jargon – use lay terms
  - "Sedate" rather than "premedicate"
  - "Transition to anesthesia" or "knock out" rather than "induce"
  - "Manage pain" rather than "analgesia"
  - "Recover from anesthesia" rather than "extubate"

Slide 23

Allaying Concerns

- Client is being too demanding or difficult
  - Reflective listening
  - Validate concerns
  - Explain rationale for choices
  - Explain need for anesthesia
  - Decide how important the issue is

Slide 24

Allaying Concerns

- Check with client to see if explanation/discussion has met their expectations
- Can appeal to 'higher power'
  - Clinic policies
    - "I'm sorry the clinic policy is to not have clients present for the start or end of anesthesia."
  - Organizations
    - "Well the American Veterinary Dental College recommends animals be under general anesthesia for cleanings, because that's the only way to do a proper dental cleaning."

Slide 25

Clinical Recommendations

- Proactively engage clients about anesthesia
- Validate feelings/concerns
- Provide information
- Call local anesthesiologist for backup

Slide 26

Learning Objectives

- Participants will understand risk factors for anesthesia
- Participants will know risk rate for adverse events
- Participants will understand concerns clients have
- Participants will be able to communicate with clients about the risk of anesthesia

Slide 27

Summary

- Participants will understand risk factors for anesthesia
  - Sick, emergency, small, +/- old
- Participants will know risk rate for adverse events
  - 1:2000 healthy, 1:100 sick
- Participants will understand concerns clients have
  - Death, pain, nausea, behavior, drug reactions, generalized
- Participants will be able to communicate with clients about the risk of anesthesia
  - Validate, explain

Medical Error

Dr. Erik Hofmeister

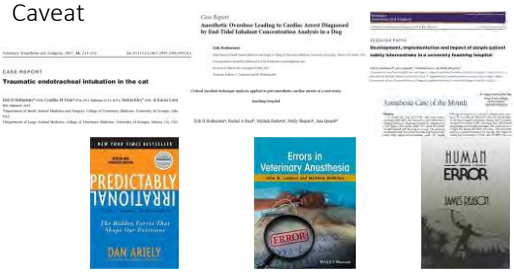
Outline

- Why errors occur
- Types of errors
- Strategies to minimize errors

Learning Objectives

- Understand why people make errors
- Describe strategies to minimize error
- Describe how to handle errors

Caveat



Why Do Errors Occur?

- To: 22333
- Text: 10257 “your text here”

Why Errors Occur

- We are not rational actors
  - Placebo effect
  - Financial motivation
  - Endowment effect
  - Cognitive biases
  - Expectations – vinegar in beer
- Excellent at pattern recognition

Et by Lion?

- On savannah, hear a rustle in the grass
- Is it a lion?
- Is it the wind?
- Do you run?
- Do you hang out?

Et by Lion

	Lion	Wind
Run	Survive	Survive
Hang Out	Die	Survive

Et by Snake?

- On savannah, see a funny object
- Is it a snake?
- Is it a stick?
- Do you run?
- Do you hang out?

Et by Snake

	Snake	Stick
Run	Survive	Survive
Hang Out	Die	Survive

Pattern Recognition Problem

- More likely to see and remember 'hits' and forget 'misses'
  - Evolved to find patterns whether meaningful or not

Ca y u rea t is?

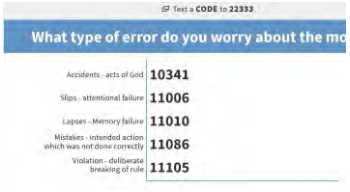
You a e not r adin g th s

Pattern Recognition Problem

- Rely on pattern recognition
- What if you have never seen the pattern before?
- Lidocaine OD
- Bird hyperthermia

Types of Errors

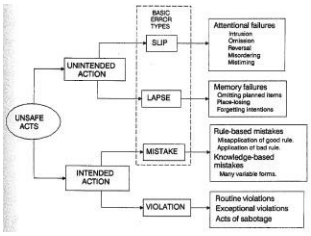
- What type of error do you worry about the most?



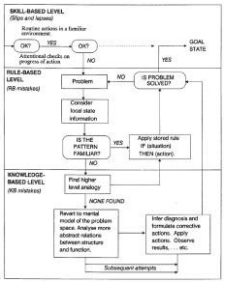
Types of Errors

- Accidents
- Unintended Actions
  - Slips
  - Lapses
- Intended Actions
  - Mistakes

Types of Errors



Types of Errors

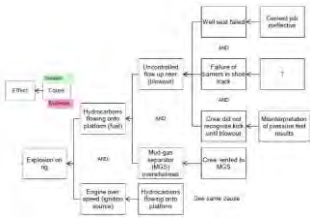


Types of Errors

- Active Errors
  - Giving wrong drug to patient
- Latent Errors
  - Drug vials of the same color

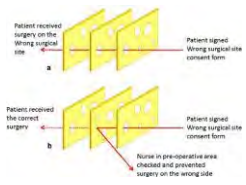
Types of Error

- System Accident
  - Highly complex
  - Tightly coupled
  - Cascading failures
  - Opaqueness

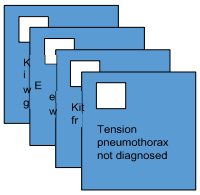


Types of Error

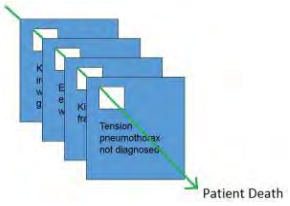
- Swiss Cheese Model
  - Complex systems have redundancy
  - Series of problems required for catastrophe
  - Organizational influences
  - Supervision
  - Preconditions
  - Specific acts



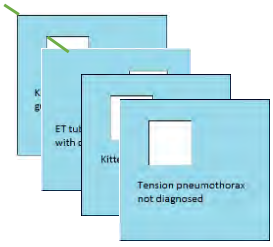
Types of Error



Types of Error



Types of Error



Types of Error

- Analysis of 13 anesthetic CPA events
  - Patient = 14
    - Looking over the shoulder of the surgeon, I could see one small area of pulmonary tissue that looked like it was normal - it was about the size of a quarter.
  - Human = 12
    - May have been preventable if more thorough monitoring peri-induction (i.e. ECG)
    - Took 2 hours of anesthesia time to cutdown to femoral, then went to CT to look for aberrant coronary vessel, which he had. Then back to IR for balloon. Took long time…
  - Procedural = 4
    - When they removed the bone due to necrosis of esophagus there was a large tear.
  - Drugs = 1
  - Equipment = 1

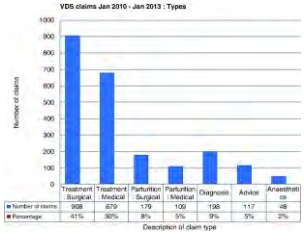
Types of Error

- Insurance Claims
  - Cognitive limitations = 51%
  - Owner contribution to error = 15%
  - Lack of technical knowledge or skill = 14%
  - Productivity = 7%
  - Failure of communication = 5%
  - Failure of leadership = 4%
  - Veterinary specific = 4%
  - Negligence = <1%

Oxtoby C, Ferguson E, White K, Mossop L. We need to talk about error: causes and types of error in veterinary practice. Vet Rec. 2015 Oct 31;177(17):438.

Types of Error

- Spay operations (n=128)
- Retained surgical items (n=76)
- Hemorrhage (n=67)



Strategies to Minimize Error

- What are strategies to minimize error?
- To: 22333
- Text: 10201 “your text here”

Strategies to Minimize Error

- First step is to identify/quantify error
  - Institution specific
  - Systems errors commonly contribute/cause problems
- Second step is to learn from error
  - M&M rounds
  - Voluntary reporting
  - Non-punitive environment
  - Confidentiality

Strategies to Minimize Error

- Critical Incident Technique
  - Qualitative analysis of what occurred
  - Useful for describing steps in detail for future remediation
- Root Cause Analysis
  - Continue to ask “why” until root cause is found

Incident 2: Transection of small intestine not observed/caught by supervising veterinarian.
Why? The supervising veterinarian had to oversee other groups.
Why? There are not enough supervisors so that each supervisor may observe up to 4 groups at a time.
-Why? The groups arrive at their operating tables at unpredictable times
-Why? Each group takes a variable amount of time with surgery and with prep.
-Why? Students are learning.
-Why? There are not enough resources to hire more supervisors.
-Why? Loss of students has reduced college revenue.
-Why? There are too many groups during each lab for the assigned number of supervisors to observe.
-Why? Groups consist of 3 students, creating ~16 groups/surgery day.
-Why? To give students the most numerous learning opportunities.

Strategies to Minimize Error

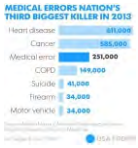
- Changing habits: "We've always done it this way"
- Accept that errors can and will occur
- Contingency plans for anticipated problems
- Prepared to seek more qualified assistance
- Do not let professional courtesy inhibit checking colleague's knowledge (pilot ice on wing example)
- Training

Strategies to Minimize Error

- Safety culture
  - Open
  - Just
  - Reporting
  - Learning
  - Informed
  - Flexible and resilient

Strategies to Minimize Error

- Cognitive Forcing
  - Rule of Three
  - Checklists
  - Mnemonics
  - Structured communication (SBAR)
- Systems walk



Strategies to Minimize Error

- What happened?
- How did it happen?
- Why did it happen?
- How do we prevent it from happening again?

NOT: Whose fault?

Strategies to Minimize Error

- Process
- Focus on system, not 'sharp' end
- Buy in – belief in improvement

Why Do Errors Occur?

- To: 22333

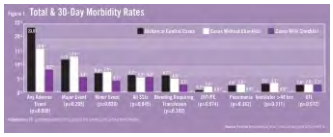
- Text: 10257 "your text here"

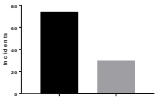
Learning Objectives

- Understand why people make errors
- Describe strategies to minimize error
- Describe how to handle errors

Conclusion

- Error is intrinsic in human activity
- Complex systems make it more likely
- Need to learn from errors and take systematic steps to improve





Newer Anesthetic Drugs

Dr. Erik Hofmeister

Purpose

- Thinking of trying new drug
- Clinicians already using them
  - Optimization
  - Should be using them?
- See what's on horizon for future

Outline

- Dexmedetomidine
- Buprenorphine/Simbadol
- Propofol/Propofol 28
- Alfaxalone
- Etomidate
- Sevoflurane
- Nocita

- Implementation

Dexmedetomidine

- Medetomidine introduced late 90s
- Used primarily in dogs/cats
- Patent was set to expire
- Pfizer switched to dexmedetomidine
- Generic medetomidine variably available

Dexmedetomidine

- Agonizes presynaptic α_2 receptor
- Inhibits release of norepi
- Decreases sympathetic outflow in CNS and periphery
- α_2:α_1 selectivity
  - xylazine 160:1
  - detomidine 260:1
  - Dex/medetomidine 1260:1

Dexmedetomidine

- Profound CV effects
  - vasoconstriction = increase SVR, increase afterload
  - Negative inotrope = decrease contractility
  - Decrease CO 30-50%
- Reflex bradycardia
- Arrhythmogenic
- Moderate respiratory depression

Dexmedetomidine

- Somewhat expensive
  - Small volume
- Moderate therapeutic index
- Arousable aggression
  - Use opioid
- Emesis, ileus
- Depresses insulin release = hyperglycemia

Dexmedetomidine

- Resedation after reversal?
- Duration shorter than medetomidine
  - 45 min vs. 60-90 min
- Sedative effects not as profound as med?

Dexmedetomidine

- Excellent sedation
  - Good for young, energetic patients
- Reversible
  - Good for short out-patient procedures
- Excellent analgesia
  - Patients still painful after opioids
  - Patients who can't tolerate opioids

Dexmedetomidine

- Use
  - 5-10 mcg/kg IM or IV
- Summary
  - Profound CV effects – bradycardia OK
  - Great sedation/analgesia
  - Reversible
  - Somewhat expensive
  - Good for healthy outpatients

Buprenorphine

- Recent research in benefits/uses
- Used in human medicine in opioid addicts
- Partial mu-agonist opioid
  - Morphine/fentanyl pure mu-agonist
  - Ceiling effect – not best for very painful patients
  - Binds mu receptor very tightly
  - Kicks morphine/fentanyl off

Buprenorphine

- Sedation with higher doses
- No real side effects
  - No panting, emesis, etc.
- Presently very expensive

Buprenorphine

- Good analgesia
  - Not as profound as morphine/fentanyl
  - Better than butorphanol
- Long lasting
  - 4-8 hours
  - Longest opioid
  - Extended release formulation (Simbadol)
- Controlled class III
  - Minimal risk of abuse (dispensing out)

Buprenorphine

- Transmucosal absorption in cats
  - Good absoprtion from mouth
  - Easy administration for clients
  - Can cause some salivation
- Maybe transmucosal in dogs
  - Make sure get in cheek
  - Ingestion not very effective

Buprenorphine

- Use
  - 0.01 mg/kg SQ/IM/transmucosal/IV
  - Q4-8h
- Summary
  - Decent analgesia (OK spay/neuter/wounds)
  - Relatively inexpensive
  - Long-lasting
  - No significant side effects

Simbadol

- Higher concentration (1.8 vs. 0.3 mg/mL)
- Higher dose (0.24 vs. 0.02 mg/kg)
- Duration ~72 hours

Propofol

- Non-barbiturate anesthetic
- Used since 1990s
- Effects at GABA, same as Thiopental
- No analgesia – need something else
- Hypotension
  - Vasodilation
  - Decrease contractility
  - Not good for CV compromised patients

Propofol

- Respiratory depression
  - Hypoventilation (increase CO_2)
  - Apnea- need to be ready to intubate
- Oxidative damage with repeat dosing in cats?
  - Evidence equivocal
- Sometimes painful given IV
  - Esp. old catheter

Propofol

- Lipid-soluble
  - Requires lipid vehicle
  - Soy-lecithin (white)
  - Promotes bacterial growth
  - Should be discarded after being open 6 hours
  - Refrigerated storage?
  - Propofol 28 contains benzyl alcohol
    - OK for induction
    - Not OK repeated inductions/CRI in cats

Propofol

- Controlled substance varies
- Requires IV access
  - Does not cause extravascular necrosis
- Can cause myoclonus
  - Not a seizure, ride it out
- Relatively narrow TI
  - Can overdose

Propofol

- Relatively expensive
  - Discarded in a day – depends on caseload
    - Propofol28 can keep for 28 days
  - More expensive than ket/val
  - Not as expensive as Telazol
  - Changeover up to caseload
    - Rec. full changeover

Propofol

- Rapidly metabolized
  - Primary indication
  - Smooth, rapid recovery
  - Liver dz, bulldogs, short procedures, neonates
  - Can be used as CRI or repeat boluses

Propofol

- Use
  - Induction 4 mg/kg
  - CRI 12-24 mg/kg/hr
- Summary
  - CV depression
  - Apnea
  - Expensive
  - Rapid/smooth recovery
  - Choice depends on caseload
    - I prefer ket/val

Alfaxalone

- Steroid anesthetic
- Part of Saffan/Althesin in 70s/80s
  - Anaphylactoid reactions in cats from carrier
  - Largely removed from veterinary market
- Now reformulated
  - Different carrier
  - Alfaxalone only (no alfadalone)
  - NO anaphylactic reactions

Propofol

- Available in the US

Alfaxalone

- Been used in Australia/NZ for while
- Controlled in the US
- Some CV depression
  - May not be as much as propofol
  - Clinical doses seems minimal
- Moderate resp depression
  - Some apnea

Alfaxalone

- Cost expensive
  - Comparable with propofol?
- No analgesia
- Recovery can be suboptimal

Alfaxalone

- Can be given IM
  - Truculent patients
  - Reptiles
- Rapidly metabolized
  - Similar to propofol
    - Not as rapid recovery
  - Can be given repeatedly
- Not labelled for stored use
- Maybe better c-sections?

Alfaxalone

- Use
  - 2-3 mg/kg IV or IM with premed
- Summary
  - Minimal CV depression
  - Some resp depression
  - Cost expensive
  - Controlled
  - Can be given IM
  - Relatively rapid/smooth induction/recovery

Etomidate

- Non-steroid non-barbiturate anesthetic
- Affects GABA as per thio and propofol
- Used in human medicine since 80s
- Universities stock and some clinics acquiring

Etomidate

- No analgesia
- Myoclonus
  - Unpleasant inductions
  - Include diazepam or midazolam
- Adrenocortical suppression
  - Esp. doses above typical induction
  - Lasts for 8+ hours
  - Bad esp. in septic patients

Etomidate

- Expensive
  - Cost per mL more than propofol
  - Volume is greater, esp. for large dogs
  - Smaller dogs may be OK
  - Can be kept over time

Etomidate

- No CV effects
  - Good for heart patients
- Decrease ICP
  - Good for head trauma
- No respiratory effects
- Wide therapeutic index
  - No CV effects so hard to overdose

Etomidate

- Not controlled
- Rapidly metabolized
  - Can give repeat boluses
  - However, be careful adrenal suppression
- Must be given IV

Etomidate

- Use
  - 0.5-1.5 mg/kg
  - Over 3mg/kg adrenocorticol suppression
- Summary
  - No CV/resp depression
  - Rough inductions
  - Expensive
  - Drug of choice for cardiac disease

Sevoflurane

- Originally synthesized 1971
- Used for long time in Japan
- Relatively recently used in animals in US
- Owned and promoted by Abbott
  - Generic is now available

Sevoflurane

- Primary differences from isoflurane
  - Solubility
  - MAC
  - Side-effects
  - Cost

Sevoflurane

- Solubility
  - Less soluble equals faster change in plasma concentration
  - Theoretical faster induction/recovery
  - Solubility difference is definite
    - Halothane 2.5
    - Isoflurane 1.33
    - Sevoflurane 0.65

Sevoflurane

- Solubility
  - Induction/Recovery Time
    - Without premed 13 vs. 10 min extubation dogs
    - With premed 11.3 vs. 10.5
    - Induction no significant difference
  - Recovery quality
    - No significant differentce immediate post-op
    - Difference later?

Sevoflurane

- MAC
  - Iso 1.3
  - Sevo 2.1
  - So, change vaporizer 0.25%
    - 0.19 x MAC Iso
    - 0.12 x MAC Sevo
  - Titrate to effect easier

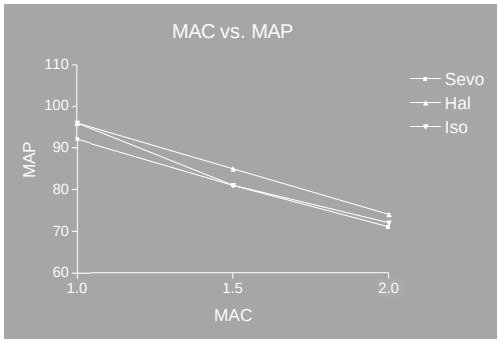
Sevoflurane

- Side-effects
  - Hypothermia as per isoflurane
  - Increased ICP as per isoflurane
  - Waste gasses similar effect
  - Slightly higher metabolism for Sevo
    - Less ideal in liver patients

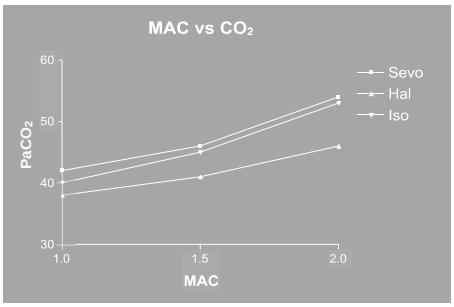
Sevoflurane

- Side-effects
  - No difference resp dep.
  - No difference CV dep.
  - Byproduct of sevo conceivably nephrotoxic
    - No clinical evidence
  - Essentially no difference from Iso

Sevoflurane



Sevoflurane



Sevoflurane

- Cost
  - Expensive to manufacture
  - Vaporizers maybe included in contract with Abbott
  - 14x cost of iso
  - Generic available cheaper but still expensive
- Less noxious

Sevoflurane

- Use
  - Vaporizer max 8%
  - Setting 3-4% typical
- Summary
  - No difference recovery times or postop quality
  - Maybe difference long-term recovery?
  - No difference side-effects
    - NO SAFER!
  - Significantly greater cost

Nocita

- Liposome-encapsulated bupivacaine
- Sustained release of local anesthetic
- Equal to epidural analgesia for stifle surgery
- Injected into peri-wound tissue
- Fairly expensive
- Other uses unsure
  - Dentals seemed to do a little better day 1-2 postop

Implementation

- Dexmedetomidine
  - Healthy dogs needing heavy sedation
  - Use bottle dose or my dose
  - Don't be afraid of bradycardia
  - Profound sedation/analgesia
    - Don't OD induction
    - Don't OD inhalant
  - Make sure to charge appropriately

Implementation

- Buprenorphine
  - Easy
  - Replace current analgesic
  - May need to increase charge
  - Educate clients for transmucosal absoprtion

Implementation

- Propofol
  - Doing a lot of bulldogs, neonates
  - Titrate to effect
  - Will need to increase charge relative to ket/val
- Propofol 28
  - No more discarding!
  - May need to increase pricing but hopefully not

Implementation

- Alfaxalone
  - Intractable patients, reptiles
  - Sick patients
  - Client charge should be appropriate

Implementation

- Etomidate
  - Very sick patients
  - Cardiac patients
  - Small patients not too expensive
  - Induction rougher
  - Charge appropriate

Implementation

- Sevoflurane
  - Picky clients
  - Edge over other guy
  - Higher vaporizer setting than iso
  - Need to adjust charging

Conclusion

- New drugs sometimes good
- Sometimes marketing ploy
- Newer not always better
- Friendly local anesthesiologist can help!

Pain Management Alternatives

Dr. Erik Hofmeister

Outline

- General Principles
- NSAIDs
- Oral Opioids
- Gabapentin
- Amantadine
- Physical
- Integrative

General Principles

- Alternatives necessary
  - Refractory acute, severe pain (surgery, trauma)
  - Patient intolerant to traditional drugs
  - Hit variety of pathways – synergy
  - Chronic, burning pain poorly responsive to traditional drugs
  - Difficult client situations

General Principles

- Chronic pain
  - Usually outpatient
  - Long-standing, established pain
  - Underlying disease not treatable
  - Strong drugs not available
  - Pain mild to severe
  - Client administration consideration
  - Treatment success frustrating
  - Try many different treatments

General Principles

- Individual responses may be due to
  - Genetic differences in receptors
  - Difference in metabolism
  - Different disease states
- Individualized patient care important
  - Incorporate client needs/values
  - Discuss goals/objectives
  - Make sure reality is discussed (similar to seizures)

General Principles

- Human pain experience
  - Significant psychological/emotional component
  - Cognitive-behavioral therapy highly effective
  - Animals don't have pain hangups/fears
  - Difficult to relate animal and human pain
- Chronic animal pain
  - Suffering
  - Immobility
  - Loss of enjoyment of activities

NSAIDs

- Historically caused significant probs
  - Esp GI
- Newer drugs dramatically safer
- Explosion of products available
  - Significant market
  - Difficult to pick out stellar performers
  - Sometimes overly excited promotion (Vioxx)

NSAIDs

- Older-style drugs
  - Ketoprofen: significant GI hemorrhage, not recommended (Banfield)
  - Buffered aspirin (Ascriptin): works fine for some patients; GI effects limit
  - Flunixin meglamine (Banamine): not usually used in dogs/cats; GI, kidney problems
  - Aspirin: GI effects
  - In general, not necessary in today's market

NSAIDs

- Newer drugs
  - More selective for COX 2
  - Cox2 responsible for inflammation
  - Cox1 responsible for side-effects, esp. GI
  - More Cox2 selective should be better!
  - …however, not so simple
  - NOT safe enough to mix
  - NEVER mix with steroids

NSAIDs

- Carprofen (Rimadyl)
  - Large number of studies
  - Proven safety in variety of situations
  - Rare liver complications (warrant monitoring?)
  - Safe in cats (off-label)
  - As effective as butorphanol for surgical pain
  - Good for chronic pain
  - Chewable tablets
  - Injectable form available

NSAIDs

- Deracoxib (Deramaxx)
  - Coxib class even more Cox2 specific
  - Not recommended for cats
  - High doses CAN cause GI effects
  - Difficult dosing small dogs
  - Rare deaths associated with use

NSAIDs

- Etodolac (EtoGesic)
  - Similar to carprofen
  - Not as much evidence
  - Generally well tolerated
  - Can have GI effects
  - Dosing can be difficult

NSAIDs

- Meloxicam (Metacam)
  - Related to piroxicam
  - Only one approved in cats
  - However, can cause renal damage (esp. longer use)
  - Good evidence of safety in variety of settings
  - Easy to dose esp. smaller patients
  - Injectable form available

NSAIDs

- Tepoxalin (Zubrin)
  - Inhibits lipoxygenase as well as Cox
  - Dual inhibitor of arachadonic acid
  - Licensed for use in dogs and cats
  - Unknown effects of anesthesia
  - Dissolvable tablet
  - GI side-effects most common

NSAIDs

- Individualized response
  - Some dogs do great with some drugs
  - Some don't tolerate side effects
  - May need to change if no success
  - Wash-out period recommended (7 days)
  - Don't give up!
- First line against chronic pain
- Can be very effective

Oral Opioids

- Classic rationale for lack of use
  - Poorly absorbed
  - Ileus
  - Dysphoria
  - Client diversion
  - Inappropriate/insufficient metabolism (codeine)
  - Perceived ineffectiveness

Oral Opioids

- Advantages
  - Good for mod-severe pain
  - Easy to administer
  - Relatively inexpensive
- Disadvantages
  - Can cause GI upset
  - Can cause dysphoria (unlikely)
  - Client diversion

Oral Opioids

- Codeine
  - Mild opioid
  - Not as divertible as morphine
  - Often marketed with NSAID- be careful!
  - Physical dependence develops
  - Maybe best reserved for severe/cancer pain
  - Cats metabolism maybe not same as dog
  - 1-2 mg/kg PO q6-8h

Oral Opioids

- Morphine
  - Highly effective analgesic
  - Can cause GI problems regularly (constipation)
  - Easily diverted
  - Bioavailability relatively low
  - Good for severe/cancer pain
  - Inexpensive
  - Can be used in cats
  - 0.5-2 mg/kg PO q6h

Oral Opioids

- Buprenorphine
  - Technically transmucosal
  - Easily administered cats
  - Dogs TM absorption also OK – put in side of mouth; large volume
  - Highly effective, minimal side effects
  - Not easily diverted
  - Administer 0.01-0.02 mg/kg PO q8h

Oral Opioids

- Tramadol
  - Mild opioid effects
  - Other effects: serotonin, norepinephrine, NMDA
  - OK for mod-severe pain
  - Dosing in dogs 2+ mg/kg PO
  - Dosing interval: q6h
  - Can be used in cats
  - GI effects (constipation, vomiting) most common
  - Do not give with psychoactive medications

Oral Opioids

- Use
  - NSAIDs insufficient
  - NSAID side-effects too problematic
- Carry own side-effects
- Consider diversion potential
- Very good analgesics
- Relatively inexpensive

Gabapentin

- GABA analog
  - Synthesized to be similar to GABA
  - However, does not act at GABA
  - Maybe affects synaptic formation
- Anti-seizure original use

Gabapentin

- Neuropathic pain analgesic
  - Unknown mechanism of action
- Not controlled
  - However, some psychotropic effects
- Xylitol sweetener in human compoundings
  - Do not use for dogs
- Fairly expensive, esp. for large dogs

Gabapentin

- In people, a third of patients with some conditions respond
- One study dogs
  - No significant benefit amputation cases
- Anecdotal evidence beneficial in chronic pain
- Maybe useful in some patients
- Adverse effects
  - Sedation, dizziness, edema (in people)

Gabapentin

- Clinical Use
  - 2-10 mg/kg PO q12h
  - Long-term treatment maybe necessary to see effect
  - Probably most useful neuropathic pain
  - Maybe useful other chronic pain
  - Worth trying if client has patience

Amantadine

- Originally designed as anti-influenza drug
- Weak NMDA antagonist
  - Dopamine and norepinephrine effects
  - Decreases central sensitization
  - However, sensitization has often already occurred
- Equivocal analgesia in people

Amantadine

- Improves client-perceived analgesia in chronic arthritis
- Unknown other disease states
- Minimal side effects
- Dose 2-5 mg/kg PO q24h x3+ weeks
  - Maybe can wean off over time
- Not expensive
- Useful in refractory cases and when not much else to do

Physical

- Nearby mechanoreceptors
  - Cross-react with nociceptors
  - Low-level stimuli of nociceptors
- Influence signal processing in spinal cord
- Gating of some signals may be occurring
  - Stimulating other neurons may result in decrease firing of nociceptors

Physical

- Increased blood flow improves healing
- Increased activity improves ROM and decreases stiffness
- Give patient something to 'do'
- Client-oriented benefits

Physical

- Massage
  - Non-painful inputs
- Physical therapy
  - Improve ROM and return to function
- No real evidence in animals yet
  - Certified canine rehabilitation
  - Cats may not tolerate well
- In people, very important field

Integrative

- Techniques that may work
  - However, mechanism unknown
  - Placebo effect
  - Not generally well accepted
  - May have role in some clinics and with some clients

Integrative

- Magnetism
- Transcutaneouos electrical nerve stimulation (TENS)
- Ultrasound
- "Cold" laser
- Acupuncture
- Chiropracty

Integrative

- Magnetism
  - Core followers/believers
  - No scientific evidence in people or animals
  - Manipulate magnetic fields to achieve effects
  - Change energy flow in patient
  - Inexpensive
  - No side-effects

Integrative

- TENS
  - Similar mechanism to physical methods
  - Evidence in people for pain relief for post-op, arthritis, musculoskeletal
  - Maybe beneficial neuropathic pain
  - Devices cost money
  - No significant adverse effects

Integrative

- Ultrasound
  - Used in human PT
  - Heats deeper tissues
  - Decrease muscle spasm
  - Increase healing/decrease scarring
  - No evidence yet
  - Units can be costly

– No significant side-effects

• Chiropracty

Integrative

- Cold laser
  - Photons from laser cause cellular effects
  - Increase healing, decrease pain sensation
  - Useful mostly for superficial pain
  - Laser light superior to normal light? Better penetration?
  - No good evidence
  - Units variable in expense
  - No real side-effects

Integrative

- Acupuncture
  - Stimulation of sites similar to gate-control theory
  - May increase endorphins
  - Some patients benefit
  - Some patients do not
  - Evidence generally positive
  - Not expensive
  - Certifying bodies
  - No side effects

Integrative

- Chiropracty
  - Physical manipulation
  - Similar to massage/PT
  - Emphasis on musculoskeletal system
  - Unknown/unproven mechanism
  - Effectiveness highly debated
  - Evidence all over
  - No real side effects

- Some patients benefit, some do not
  - Similar to NSAIDs….
- Some clients benefit
- Maybe useful to consider in practice
- No side-effects (except cost…)
- Maybe all placebo
- Can't hurt, might help

Conclusion

- Pain management complex
- Variety of options exist
  - Esp. chronic pain
  - Generally: no single best strategy
- Discussion with clients key
  - Setting expectations
- Willingness to try variety
  - Similar to internal medicine cases…
  - Cost depends on provider

Pharmacy Law

Susan Elrod, BS, PharmD, PhD

Purpose:

This course will review legal issues pertaining to medication prescribing. In particular, various controlled drug categories will be covered, with a focus on restrictions that exist for prescribing drugs from such categories. The rationale behind such restrictions will also be discussed. In order to accommodate practitioners from various states, this course will focus primarily on federal laws; individual state laws may be more restrictive. Resources for where to find state laws will be presented.

Outline:

- Introduction
- Resources
- Prescription requirements
- Controlled drug categories
    - Prescription requirements based on category
    - Partial and emergency dispensing requirements
- Electronic prescribing
- DEA numbers
- Online pharmacies
- NDC numbers

Learning objectives:

- Be able to find prescribing guidelines, both federal and for individual states
- Understand requirements for writing prescriptions
- Identify controlled drug categories and the rationale for such categories
- Understand how to write prescriptions for each drug category
- Identify prescribing considerations specific to veterinary medicine

Introduction:

- Overarching federal laws regarding prescriptions
- State-by-state laws may be more stringent
    - Requirements default to stricter law
    - If state law LESS stringent than federal, follow federal law
- Requirements for written prescriptions, prescription labels, dispensing amounts, types of drugs, types of prescribers
- Prescriptions versus orders
    - Prescription: medication dispensed to ultimate user
        - Home medications
    - Order: inpatient use

Resources:

https://www.deadiversion.usdoj.gov/index.html
https://nabp.pharmacy/boards-of-pharmacy/

Prescription requirements
    - Patient name
    - Patient address
    - Prescriber name
    - Prescriber address
    - Drug name
    - Drug strength
    - Dosage form
    - Quantity
    - Directions
    - Refills
- For controlled substances
    - Prescriber DEA number

Label requirements
- Fill date
- Pharmacy name
- Pharmacy address
- Prescription number
- Patient name
- Prescriber name
- Directions for use
- Any cautionary statements
- For CII – CIV: “CAUTION: Federal law prohibits the transfer of this drug to any person other than the patient for whom it was prescribed”.
- If filled at central pharmacy:
    - Label with retail pharmacy name, address, central pharmacy DEA registration number

Permitted prescribers:
- Controlled substances
    - Physicians
    - Dentists
    - Podiatrists
    - Veterinarians
    - Mid-level practitioners
    - Other registered practitioners
        - Authorized by state/jurisdiction

          AND
        - Registered with DEA
            - Exemptions: Public Health Service, Federal Bureau of Prisons, military practitioners

          OR
        - Employees acting under institutional DEA registration

Controlled substance schedules:

I.

- No currently acceptable medical use
- Lack accepted safety use with medical supervision
- High abuse potential

**Marijuana still in CI, despite individual state laws

II.

- Medical use
- High abuse potential
  - Severe psychological/physical dependence
- Hydromorphone, methadone, fentanyl, codeine, hydrocodone
- Non-narcotic: amphetamine, methylphenidate
  - Sometimes designated IIN
- Amobarbital, pentobarbital

III.

- Medical use
- Lower abuse potential than CI or CII
  - High psychological dependence
  - Moderate/low physical dependence
- Tylenol with codeine
  - Not more than 90 mg codeine/dose
- Buprenorphine
- Ketamine
- Testosterone

IV.

- Medical use
- Lower abuse potential than CIII
- Benzodiapezines

V.

- Medical use
- Lower abuse potential than CIV
- Drugs with limited quantities certain narcotics
  - Cough preparations: ≤ 0.2% codeine
    - ≤ 200 mg per 100 mL or 100 grams
  - Lomotil®
    - Diphenoxylate: opioid antidiarrheal
    - Atropine: prevents abuse of opioid effects

Prescriptions requirements for controlled drugs:

- Pharmacist may only dispense controlled substances to patient or member of patient's household

CII:

- Written prescription
  - Manually signed
- Electronic prescription
- May be filled at any time after being written and signed
  - Does not expire

    - Pharmacist must determine medical need
- No limit on quantity
    - 30 days per many states, insurance companies
    - Must be dispensed only for legitimate medical purpose
- Verbal order for emergency dispensing
- NO REFILLS
- May issue multiple prescriptions at once up to 90 days' supply total
    - Each must be on different prescriptions blank
    - Must have earliest date can be filled
    - No undue risk of diversion/abuse in prescriber's opinion
    - Maintain good practice
        - See patient as often as deemed necessary
        - Only issue 90 days' if warranted
- May be faxed in order to expedite filling
    - Must present original when picking up
- When fax can serve as original:
    - Compounded CII for direct IV/IM/SQ/intraspinal infusion
    - Long term care facility residents
    - Medicare/state-licensed hospice patients

Emergency dispensing

- Emergency prescription: immediate administration of drug necessary for proper treatment
    - No alternative treatment available
    - Not possible for prescriber to provide written prescription
- Telephone order allowed for CII
    - All required information written out by pharmacist with exception of prescriber signature
    - Written, signed prescription must be provided to pharmacy within seven days
        - "Authorization for Emergency Dispensing" written on prescription
        - Date of oral order
        - May be mailed: postmarked within seven days
        - Pharmacist must attach to written oral prescription
        - IF NOT PROVIDED WITHIN SEVEN DAYS:
            - Pharmacist must notify local DEA Diversion Field Office
                - If not, pharmacy loses authorization to dispense controlled substances without written prescription
                    - Includes non-CII controlled substances
- Amount prescribed limited to that needed for emergency period
    - Written order for amount beyond emergency period
- If no professional relationship between pharmacist, prescriber
    - Pharmacist must make reasonable effort to verify prescriber
        - Example: identifying clinic phone number from directory, calling back

Partial dispensing

- If pharmacist unable to supply full quantity
- Pharmacist must note amount supplied on prescription
    - Written
        - Transcribed from emergency phone order

    - Electronic
- Remainder must be dispensed within 72 hours
    - If not, pharmacist notifies prescriber
    - New prescription required for remainder after 72 hours
- Terminal illness and long-term care facility patients
    - May dispense individual dosage units
    - Document on prescription “terminally ill” or “LTCF patient”
    - Total partial fill quantity may not exceed amount prescribed
    - CII for LTCF or terminally ill patients only valid for 60 days

CIII – CV

- May be faxed/called in/e-prescribed
- No more than 5 refills
- Prescription expires after 6 months

CIII and CIV

- On back of prescription:
    - Pharmacist’s initials
    - Date refilled
    - Amount dispensed
        - If different from full amount
- May only be transferred once
    - May be transferred up to maximum refills between pharmacies sharing real-time online database
        - Non-controlled prescriptions may be transferred between pharmacies as often as requested up to total amount to be refilled
    - Must write “VOID” on face of hardcopy
        - Transfer recorded for electronic prescriptions
    - On back of transferred prescription/in electronic prescription record:
        - Name, address, DEA registration of receiving pharmacy
        - Name of receiving pharmacist
        - Name of transferring pharmacist
        - Date of transfer
    - Receiving pharmacy, whether using written/electronic prescription
        - All required original prescription information
        - Original dates
            - Written
            - Dispensed
        - Original number of refills
        - Remaining refills
        - Dates of previous refills
        - Original pharmacy’s name, address, DEA registration, prescription number
        - Name of transferring pharmacist
- Records of original, transferred prescriptions maintained for 2 years from date of last refill

Electronic prescriptions

- May be used to prescribe controlled substances
- Must meet various standards
    - System must allow for verification of prescriber
        - Documentation of verification
    - Nonrepudiation
        - Prescriber may not be able to deny prescription
    - Ensures prescription contents not altered during transmission
- Digital signature
- Pharmacy must have certification that software complies with standards
- All written prescription requirements apply
    - Must be stored by pharmacy software
- Method of restricting access to those pharmacy employees authorized to enter information relating to controlled substances
- Ability to make any notations electronically that are required for written prescriptions
- Paper or oral prescription duplicates of electronic prescriptions
    - Pharmacist must verify was not filled
    - If both received, mark one void

DEA numbers

- Issued to individuals and institutions
- Must be able to verify for controlled prescriptions
- First letters:
    - 'A' for those issued before Oct. 1, 1985
    - 'B' or 'F' thereafter
    - 'M' for mid-level practitioners
- Second letter: first letter of practitioner's last name
- Computer-generated seven-number sequence
    - How to verify:
    - Add 1^{st}, 3^{rd}, 5^{th} digits
    - Add 2^{nd}, 4^{th}, 6^{th} digits, multiply by 2
    - Above numbers
        - Last digit should be same as last number of seven-number sequence

AX5836727 ←

5 + 3 + 7 = 15

8 + 6 + 2 = 16 x 2 = 32

15 + 32 = 47 → last number is 7

- Institutional employees: may use hospital DEA number
    - Prescribing in usual course of practice
    - Permitted to do so in state
    - Hospital has verified practitioner can prescribe controlled substances in state
    - Acts only within institution
    - Hospital authorizes by designating suffix
    - Current list of individual's DEA suffixes maintained by hospital
        - Available at all times
- Exempt practitioners
    - Military practitioners

        - Army
        - Navy
        - Marine Corps
        - Air Force
        - Coast Guard
    - Public Health Service
    - Bureau of Prisons
    - May not procure or purchase controlled substances
        - May administer, dispense, prescribe
    - Government branch, service identification number required in place of DEA number
        - Public Health Service: Social Security number serves as identification number
- Mid-level practitioners
    - Physician assistants
    - Nurse anesthetists
    - Animal shelters
    - Euthanasia technicians
    - Authority to prescribe controlled substances varies according to state
        - DEA number may be issued based on state allowance
        - Pharmacist must verify prescriber is acting within laws of state where they are licensed

Online pharmacies

- Defined as person, entity, site intentionally dispensing controlled substance via the Internet
- Ryan Haight Online Pharmacy Consumer Protection Act of 2008
    - Prevention of unlawful dispensing of controlled substances via Internet
- Online pharmacies must hold modified DEA registration
- Does not include hospitals associated with:
    - U.S. government (e.g. Armed Forces)
    - American Indian tribes
- Does not apply to online refills/electronic prescriptions for registered pharmacies
- Upon application as online pharmacy; must be on homepage:
    - "In accordance with the Controlled Substances Act and the DEA regulations, this online pharmacy has made the notifications to the DEA Administrator required by 21 U.S.C. § 831 and 21 C.F.R. § 1304.40"
- Once approved, homepage must have declaration of compliance with requirements on homepage at all times
    - "This online pharmacy is obligated to comply fully with the Controlled Substances Act and DEA regulations. As part of this obligation, this online pharmacy has obtained a modified DEA registration authorizing it to operate as an online pharmacy. In addition, this online pharmacy will only dispense a controlled substance to a person who has a valid prescription issued for a legitimate medical purpose based upon a medical relationship with a prescribing practitioner. This includes at least one prior in-person medical evaluation in accordance with section 309 of the Controlled Substances Act (21 U.S.C. § 829),

or a medical evaluation via telemedicine in accordance with section 102(54) of the Controlled Substances Act (21 U.S.C. § 802(54)).”
    - Including name of pharmacy as appears on DEA registration
- Internet Pharmacy Site Disclosure Information
    - Pharmacy name, address, telephone number, e-mail address
    - Pharmacist-in-charge name, professional degree, states in which licensed, telephone number
    - States in which pharmacy licensed to dispense controlled substances
    - Certification that pharmacy is registered to dispense, deliver, or distribute controlled substances
    - Contact information for any practitioner contracted to provide medical evaluations/issue prescriptions through referrals from website
        - Name, address, telephone number, professional degree, states of licensure
- Must submit monthly report to DEA of total amount controlled substances dispensed
    - More than 100 controlled substances prescriptions dispensed
    - At least 5,000 controlled substance dosage units dispensed
    - If neither above is true, submit negative report for that month
    - May include internet transactions, mail order, or in-person
        - Means of dispensing not required in report
            - By NDC numbers
    - Due on or before 15th of following month
- Prescriptions must be for valid medical purpose
    - At least one in-person medical evaluation

NDC numbers

- National drug code
- Universal identifier for drugs
- Provided to FDA
- Three segments, 10 digits
    - Manufacturer/labeler identifier
    - Product identifier
        - Drug, strength, dosage form
    - Package size identifier

Veterinary Supplements

Susan Elrod, BS, PharmD, PhD

Purpose:
This course will cover the evidence behind the most commonly used herbal supplements and natural products in veterinary medicine. The studies referenced for these products will be provided, along with methods to check the safety and efficacy of such products. This course is provided for information only; no endorsement of any particular product is intended or should be inferred.

Outline:

- Definitions
- Common natural products
    - Omega-3 fatty acids
    - Glucosamine/chondroitin
    - SAM-E +/- sylibin
    - Milk thistle
    - Tea tree oil
    - Echinacea
    - Garlic
    - Cranberry
- Rationale for use
- Diseases/conditions
- Species
- Evidence
- Resources
- Critical evaluation

Definitions:

- Nutraceuticals: blend of “nutrition” and “pharmaceutical”
    - Coined in 1989 by Stephen DeFelice
- Supplement: addition to typical diet/treatment
- Functional food: food or part of food that imparts some benefit beyond simple nutrition

Rationale for use of nutraceuticals:

- Many clients may use as extension of trying to impart best nutrition to pet
- Veterinarians’ use in patients:
    - Due to client request
    - Preventative
    - Due to failure of conventional treatment
- Veterinarians’ concerns:
    - Safety and efficacy
    - Cost
    - Adverse effects of nutraceutical
    - Availability
    - Interactions
    - Client resolve
    - Exhaustion of alternatives

- Common conditions perceived to benefit from nutraceutical use:
    - Osteoarthritis
    - Dermatologic disease
    - Liver disease
    - Gastrointestinal disease
    - Renal disease
    - Cardiac disease
    - Cancer
    - Dental disease

Omega-3 Fatty Acids

- From fish such as tuna and salmon
    - Eicosapentaenoic and docosahexaenoic acids (EPA and DHA)
    - Small amount in beef, pork, poultry
- In flax, canola oil
    - Specifically α-linoleic acid: precursor to fatty acids in fish
- Recommended for human consumption by American Heart Association
    - Beneficial both in health individuals and those with cardiovascular disease
- Rationale for use[1]
    - Incorporated into cell membranes in all tissues[2]
        - Especially retina, brain, myocardium
        - Suggested to contribute to proper cell function
    - Compete with omega-6 fatty acids
        - Arachidonic acid
        - ➔ Cellular mediators
            - ➔ Platelet activation
            - ➔ Gastric secretion
            - ➔ Bronchoconstriction
            - ➔ Pain signaling
        - Omega-3 fatty acids prevent omega-6 inflammatory effects
    - Mechanism of benefit not fully known
    - Potential mechanisms:
        - Reduced susceptibility to arrhythmias
        - Anti-thrombogenesis
        - Lower triglycerides
        - Anti-inflammatory
        - Reduce atherosclerotic plaques
        - Increase nitric oxide-induced endothelial relaxation
        - Hypotensive
- Veterinary studies:
    - Osteoarthritis
    - Dermatitis
    - Cardiomyopathy
    - Cancer

[1] https://www.ahajournals.org/doi/full/10.1161/01.cir.0000038493.65177.94?url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org&rfr_dat=cr_pub%3Dpubmed&

[2] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2174995/

    - Reproduction
    - Inflammation
        - Exercise tolerance
- Species:
    - Dogs
    - Cats
    - Horses
    - Food animals
    - Birds
    - Mink
- Osteoarthritis
    - Dogs
        - Seven clinical studies
            - J Am Vet Med Assoc. 2010 Jan 1;236(1):59-66.
            - J Am Vet Med Assoc. 2010 Jan 1;236(1):67-73.
            - J Am Vet Med Assoc. 2010 Mar 1;236(5):535-9.
            - Can J Vet Res. 2013 Jan;77(1):66-74.
            - J Anim Physiol Anim Nutr (Berl). 2013 Oct;97(5):830-7.
            - Prostaglandins Leukot Essent Fatty Acids. 2016 Jun;109.
            - J Am Vet Med Assoc. 2018 Mar 15;252(6):701-709.
        - Certain treatments showed increase in plasma omega-3 fatty acids
        - Clients in some studies reported improved osteoarthritis symptoms
        - Appeared to be well-tolerated
        - One study showed decreased carprofen dose due to supplementation
- Dermatitis
    - Dogs
        - Four clinical studies
            - Can J Vet Res. 1997 Apr;61(2):145-53.
            - J Small Anim Pract. 2004 Jun;45(6):293-7.
            - Vet Res Commun. 2011 Dec;35(8):501-9.
            - Vet J. 2016 Apr;210:77-81.
        - One study showed atopic dogs have lower lipid levels in stratum corneum
            - Improved with supplementation
        - Improvement in some clinical measures/client assessment of dermatitis
        - One study showed cyclosporine-sparing effects
    - Horses
            - Vet Dermatol. 2019 Apr;30(2):155-e46.
            - Treated 21 horses with cream containing fish oil
                - Half of horse's body for 4 weeks, all lesions for next 4 weeks
            - Improvement with treated side in first 4 weeks
            - No improvement in pruritis, coat quality
            - Adverse effects in 5 horses
- Cardiomyopathy
    - Dogs

        - J Vet Intern Med. Mar-Apr 2007;21(2):265-73.
            - Boxers with arrhythmogenic right ventricular cardiomyopathy on no antiarrhythmics treated with 2 grams fish oil, flax oil, or placebo for 6 weeks
            - Significant decrease in ventricular premature contractions in fish oil group
    - Cats
        - J Feline Med Surg. 2014 Aug;16(8):631-6. n = 43
            - Non-supplementation study
            - Studied plasma of cats with and without hypertrophic cardiomyopathy
                - HCM associated with higher levels of plasma fatty acids
                - Authors conclude supplementation unlikely to provide benefit
- Cancer
    - Dogs
        - Cancer. 2000 Apr 15;88(8):1916-28.
            - Dogs with lymphoma
            - Fatty acids significantly increased disease-free interval and survival times
- Reproduction
    - Horses
        - Theriogenology. 2005 Mar 15;63(5):1519-27.
            - Study of effect of fatty acids on semen
            - Significantly improved semen characteristics with treatment
            - Especially beneficial for horses with marginal fertility
            - Improved freezability
- Inflammation/exercise tolerance
    - Cats
        - J Anim Physiol Anim Nutr (Berl). 2012 Aug;96(4):671-80.
            - No improvement on wound healing following molar extraction
    - Horses
        - J Vet Intern Med. Jul-Aug 2002;16(4):457-63.
        - Theriogenology. 2005 Mar 15;63(5):1519-27.
        - Equine Vet J Suppl. 2006 Aug;(36):279-84.
        - Can J Vet Res. 2007 Jan;71(1):59-65.
        - J Anim Sci. 2012 Sep;90(9):3023-31.
        - J Vet Intern Med. 2015 Jan;29(1):299-306.
            - Improvement in inflammatory response
            - Prevention of exercise-induced decrease in erythrocyte membrane fluidity
            - Fatty acids incorporated into skeletal muscle
            - Variable effects on airway disease
                - May have benefit in combination with low-dust diet

Glucosamine/Chondroitin

- Rationale for use[3]
  - Used for arthritis
  - Endogenous components of cartilage
    - Cartilage worn down in osteoarthritis
    - Theorized to preserve cartilage and prevent pain
- Species:
  - Dogs
  - Cats
  - Horses
  - Rabbits
- Dogs
  - Vet J. 2007 Jul;174(1):54-61.
    - Significant improvement
      - Slower onset compared to carprofen as positive control
  - J Am Vet Med Assoc. 2010 Jan 15;236(2):183-6.
    - No effect on glycemic control; did not cause diabetes
- Cats
  - Vet Comp Orthop Traumatol. 2014;27(1):20-6.
    - Does not appear to have effect
    - Meloxicam effective
- Horses
  - Vet Comp Orthop Traumatol. 2014;27(1):20-6.
    - No effect on gait characteristics
- Rabbits
  - Connect Tissue Res. 2011;52(4):329-39.
    - Improvement shown compared to control group following tendon rupture repair
    - In combination with sodium hyaluronate

S-adenosylmethionine

- Rationale for use[4]
  - Endogenous compound
    - Responsible for several biochemical pathways
    - Believed that supplementation allows for support and/or enhancement of those pathways
  - Improves effects and function of phospholipids in cell membranes
    - Particularly phosphatidylcholine in liver cells
    - Improves liver cell fluidity, bile flow
  - Readily converted in liver to glutathione: antioxidant
    - Many veterinary patients deficient in glutathione
    - Liver site of detoxification: especially susceptible to damage
      - Hepatoprotection
  - Also converted to methylthioadenosine

[3] https://www.nccih.nih.gov/health/glucosamine-and-chondroitin-for-osteoarthritis
[4] https://veterinarypartner.vin.com/default.aspx?pid=19239&id=4951818

        - Anti-inflammatory and analgesic compound
        - Supports development of cartilage
- Species:
    - Dogs
    - Cats
- Dogs
    - Am J Vet Res. 2005 Feb;66(2):330-41.
    - J Vet Intern Med. Jul-Aug 2011;25(4):838-45.
    - J Small Anim Pract. 2019 Sep;60(9):543-550.
        - Effects appear limited
        - Showed prevention of CCNU-induced liver toxicity
            - Significantly decreased treatment delays due to hepatotoxicity
        -
- Cats
    - J Feline Med Surg. 2003 Apr;5(2):69-75.
        - Protected erythrocytes from acetaminophen-induced oxidative damage

Milk Thistle

- Contains silybin
    - In Denamarin along with SAM-E
- Rationale for use[5]
    - Antioxidant
        - Protects against oxidative damage in liver
        - Maintains integrity of liver cells
        - Studied in humans for protection against alcohol-induced liver damage
    - Immunomodulator
    - Anti-fibrotic
    - Anti-proliferative
    - Antiviral
- Species:
    - Dogs
    - Rabbits
    - Horses
    - Cattle
    - Chickens
- Dogs
    - J Vet Pharmacol Ther. 2007 Oct;30(5):477-81.
        - Significantly decreased gentamicin-induced nephrotoxicity
        - Along and in combination with vitamin E
- Rabbits
    - Anim Reprod Sci. 2017 Sep;184:178-186.
        - Significantly improved semen quality, fertility, economic efficiency
            - Rosemary also studied, significantly improved semen quality and fertility
- Horses

[5] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3959115/

    - Am J Vet Res. 2013 Oct;74(10):1327-32.
    - Am J Vet Res. 2013 Oct;74(10):1333-9.
        - Safe, but may be poorly bioavailable
        - Limited antioxidant effects
- Cattle
    - J Vet Med A Physiol Pathol Clin Med. 2004 Mar;51(2):85-9.
        - Appears safe but not effective in dairy cows
- Chickens
    - Poult Sci. 2010 Sep;89(9):1878-86.
    - J Anim Physiol Anim Nutr (Berl). 2018 Apr;102(2):410-420.
        - No effect on gossypol toxicosis
        - Improvement in health and egg production

Tea Tree Oil

- Rationale for use[6]
    - From leaves of tea tree
    - Used by Australian aboriginal people for cuts and wounds
    - Limited research showing benefits on acne, nail fungus, etc.
    - Topical use only
- Species:
    - Dogs
    - Horses
    - Donkeys
    - Sheep
- Dogs
    - Schweiz Arch Tierheilkd. 2002 May;144(5):223-31.
    - Dtsch Tierarztl Wochenschr. 2004 Oct;111(10):408-14.
        - Significant improvement in dermatitis with 10% tea tree oil cream
            - Greater improvement than commercial cream
        - Few adverse events reported
- Horses
    - Phytomedicine. 2009 Nov;16(11):1056-8.
        - Significant improvement on ringworm infections
- Donkeys
    - Med Vet Entomol. 2013 Dec;27(4):408-13.
        - Significant reduction in lice with both tea tree oil and lavender oil
- Sheep
    - Vet Parasitol. 2012 Oct 26;189(2-4):338-43.
        - Treatment of sheep with chewing lice with 1% and 2% formulations
        - Increased with jetting
        - Reduction in wood damage
- IMPORTANT: adverse effects[7]
    - High concentrations applied topically, licked off
    - Supportive care appears to be effective in most cases

[6] https://www.nccih.nih.gov/health/tea-tree-oil
[7] Vet Hum Toxicol. 1994 Apr;36(2):139-42; J Vet Diagn Invest. 1998 Apr;10(2):208-10.

Echinacea

- Rationale for use[8]
  - Immunostimulation
    - Activates phagocytes
    - Stimulates fibroblasts
    - Enhances respiratory activity
      - Often used for upper respiratory infections
  - Various effects found in studies
    - Anti-inflammatory
    - Anti-anxiety/-depression
    - Cytotoxic
    - Antimutagenic
- Species
  - Dogs
  - Horses
  - Sheep
  - Chickens
    - No effects demonstrated on growth or benefit against E. coli or coccidia
- Dogs
  - Schweiz Arch Tierheilkd. 2003 May;145(5):223-31.
    - Administered to dogs with upper RTIs
    - Significant improvement in 92% dogs over 8 weeks
    - No control group
    - Two adverse effects
- Horses
  - Equine Vet J. 2002 May;34(3):222-7.
    - Healthy horses
    - Stimulated immunocompetence, improved blood quality
      - May improve exercise performance
- Sheep
  - Domest Anim Endocrinol. 2012 Oct;43(3):213-26.
    - Roots and flowers in feed mitigated cortisol-mediated immune and stress response
- Rabbits
  - Animal. 2016 Jul;10(7):1101-9
    - Various effects studied after feed supplementation
      - Increase in phagocytic activity only effect

Garlic

- Rationale for use[9]
  - Not fully established
  - Alliin/allicin believed to be responsible for effect
    - Appear to have HMG-CoA reductase inhibitory effects
      - Similar mechanism to ‘statins’

[8] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4441164/
[9] https://www.sigmaaldrich.com/life-science/nutrition-research/learning-center/plant-profiler/allium-sativum.html

        - Inhibits oxidation of lipoproteins
        - Inhibits platelet aggregation
        - Vasorelaxant
        - May have heptatoprotection, anti-proliferative effects
        - Possible immunostimulant, antimicrobial effects
- Species
    - Cows
    - Pigs
    - Sheep
    - Chickens
    - Horses
- Pigs
    - Pol J Vet Sci. 2009;12(3):407-14.
    - J Anim Sci. 2011 Jul;89(7):2123-31.
    - Vet Microbiol. 2012 Jan 27;154(3-4):316-24.
    - J Anim Physiol Anim Nutr (Berl). 2013 Jun;97(3):457-64.
    - J Anim Sci. 2013 Nov;91(11):5294-306.
    - J Anim Sci. 2013 Dec;91(12):5668-79.
    - J Anim Sci. 2014 Aug;92(8):3426-40.
    - Vet Rec. 2019 Mar 9;184(10):316.
        - Improved growth performance in piglets
        - Improved GI health
            - *E. coli*
            - Proliferative enteropathy
        - Improved immune response
            - Porcine reproductive and respiratory syndrome virus
            - Pluropneumoniae infection
- Cows
    - Trop Anim Health Prod. 2010 Jun;42(5):961-8.
    - J Anim Physiol Anim Nutr (Berl). 2011 Aug;95(4):449-55.
    - J Anim Physiol Anim Nutr (Berl). 2016 Aug;100(4):623-8.
        - Improved feed conversion ratio, blood metabolites
        - Improved body weight, daily intake, decrease in scours
        - Lower doses more effective
- Sheep
    - J Anim Physiol Anim Nutr (Berl). 2011 Apr;95(2):187-91.
    - Meat Sci. 2011 Jul;88(3):590-3.
        - Decreased methane production
        - Improved nutrient utilization
        - Improved taste of meat
            - Or did not adversely affect taste
- Chickens
    - Poult Sci. 2012 Sep;91(9):2148-57.
    - J Anim Sci. 2014 Sep;92(9):3945-53.
    - J Anim Sci. 2015 Jul;93(7):3410-20.
    - Poult Sci. 2015 Aug;94(8):1812-20.

    - J Anim Physiol Anim Nutr (Berl). 2017 Oct;101(5):e122-e132.
        - Improvements in GI health, immune response, growth performance/production
        - Decreased blood pressure, ascites incidence
- Horses
    - Can J Vet Res. 2007 Apr;71(2):145-51.
        - Composite of various herbs, including garlic
        - Fed to 6 horses with recurrent airway obstruction
        - Improved respiratory rate, immune response

Cranberry

- Rationale for use[10]
    - Used for urinary tract infections
    - Phytochemicals prevent bacterial adhesion in urethra
    - Veterinary clinical trials
- Other mechanisms studied (not clinical trials):
    - Anthelmintic effects in sheep (Vet Parasitol. 2018 Apr 15;253:122-129)
    - Immunomodulatory effects in chickens (Poult Sci. 2017 Feb 1;96(2):341-350)
- Species
    - Dogs
    - Cats
- Dogs
    - Am J Vet Res. 2016 Apr;77(4):421-7.
        - Prevention of UTIs in dogs administered cranberry extract
        - Prevention adhesion of *E. coli* to canine kidney cells
- Cats
    - Physiol Rep. 2018 Jun;6(12):e13737.
        - Cats with Stage II or III chronic kidney disease
        - Fed diet with various nutraceuticals including cranberry
        - Improvement in indicators of renal failure

Summary:

- Clinical trials available for many natural supplements
- Variable evidence
- Research available to assist in making informed decision regarding treatment/ recommendation

[10] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3370320/

Non-Pharmacologic Therapies

Susan Elrod, BS, PharmD, PhD

Purpose:
This course will cover the evidence behind the most commonly used complementary and alternative care methods used in veterinary medicine. The studies referenced for these products will be provided, along with methods to check the safety and efficacy of such products. This course is provided for information only; no endorsement of any particular product is intended or should be inferred.

Outline:

- Definitions
    - Complementary care
    - Alternative medicine
- Rationale for use
- Common procedures
    - Acupuncture
    - Chiropractic care
    - Low energy photon therapy
- Diseases/conditions
- Species
- Evidence
- Resources

Definitions:

- Complementary and alternative medicine (CAM): Identified by the AVMA as “a heterogeneous group of preventive, diagnostic, and therapeutic philosophies and practices” that may deviate from routine veterinary medicine
- Complementary care: Therapies (usually non-pharmacologic) used *in addition* to routine or conventional veterinary care
- Alternative medicine: Therapies (usually non-pharmacologic) used *in place of* routine or conventional veterinary care

Rationale for use:

- Overarching idea: treating patient as a whole
    - Rather than specific disease or presenting symptom
- Genetics
    - Breeds
- Nutrition
- Hygiene
- Stressors
    - Patient-specific
    - Species-specific

Acupuncture:

- Ancient Chinese technique
- Stimulation of specific points on the body
    - Thought to maintain/ restore health

- Belief in Chinese medicine that ill health results from imbalance in passive and active principles
    - Yin – yang principle
    - Acupuncture believed to relieve energy blockage due to imbalance
- Involves stimulation via various methods
    - Needles
        - Solid
        - Hypodermic
        - Bleeding
    - Electricity
    - Heat
    - Massage
    - Low power lasers
- Acupuncture points developed for human treatment transposed onto anatomy of various animal species
    - Points in palpable depressions
    - Low electrical resistance
    - High skin conductance
    - Accumulations of neurovascular bundles
- Gained awareness in the U. S. in the 1970s
- Research has been developed to explore the evidence-based rationale
    - Proposed mechanisms highly complex
    - Signal cascades
- Proposed mechanisms
    - Stimulation of sensory fibers
        - Affects motor neurons, vasculature, ligaments
    - Stimulation of afferent neurons
        - Inhibits nociception
    - Effect on nociceptive fibers
        - Inhibited by opioids
    - Effect on various neurotransmitters
        - Norepinephrine
        - Serotonin
        - Substance P
        - Acetylcholine
        - GABA
- Neuropathy
    - Affects processes that generate neuropathic pain
        - Neurotransmitters and receptors that impact central sensitization
        - Imbalance between excitatory and inhibitory processes
        - Mechanoreceptive sensory fibers that generate Substance P
- Inflammation
    - Electroacupuncture
        - Decreases lymphocytes, cytokines, T and B cells
        - Affects leukocyte migration and activity
- Methodology

- Needles embedded for 20 to 30 minutes
- Frequency
  - Intiation period: every 1 – 2 days
  - Continuation period: weekly
  - Maintenance: weekly – monthly
    - For chronic conditions such as osteoarthritis

- Techniques
  - Dry needle
    - Most common
    - May elicit hyperemia/sudden sedation
    - Length of stimulation should depend on diagnosis, etc.
  - Aqua-puncture
    - Fluids injected into acupuncture point
      - Saline
      - Vitamin B12
      - Bee venom
      - Blood
    - Prolonged effect
      - Alternative to extended dry needle stimulation
    - Pharmacoacupuncture
      - Fluid medications injected
      - Desired effect at much lower dose
  - Implantation
    - Embedding agents at acupuncture points to induce longer duration
      - Sutures
      - Staples
      - Wire
      - Gold
        - Cyanide from inflammatory cells believed to complex with gold
        - Inhibit inflammatory processes
        - Used for hip dysplasia, degenerative joint disease, epilepsy
  - Laser
    - Lasers at 630 – 960 nm wavelengths, 5 – 30 mW energy
    - Refraction in tissues
      - Best for shallow acupuncture points
    - Analgesic and anti-inflammatory
  - Electroacupuncture
    - Electrical stimulation of peripheral nerves
    - Low frequencies affect neuropathic pain
      - 2 Hz
      - Effect on NMDA receptors
    - Higher frequencies affect GABA and serotonin pathways
      - 100 Hz
      - Less effect on neuropathic pain

- Contraindications
  - Infected or inflamed areas
  - Fractures
  - Clotting abnormalities
  - Pacemakers
    - Electroacupuncture
  - Tumors
    - Especially electroacupuncture
- Precautions
  - Pregnancy
    - Avoid abdominal, certain other sites
  - Broken needles
    - Very rare
    - May migrate through tissue
- Recommending acupuncture for patients
  - Certification by American Academy of Veterinary Acupuncture and/or International Veterinary Acupuncture Society
    - www.aava.org
    - www.ivas.org
    - Searchable websites
  - Develop direct awareness of acupuncturist's proficiency
    - Certification not guarantee of competency

- Chiropractic care
  - Rationale
    - Dislocation/misalignment of joints
      - Vertebral
      - Extremities
      - Cranial sutures
    - Chiropractic care corrects via high-velocity controlled thrusts
    - Veterinary specific: analysis of physical, neurological state
      - Stance
      - Gait
      - Palpations
        - In motion
        - Static
  - Definitions
    - Subluxation: disturbance in biomechanical and/or neurologic function due to misalignment, etc.
      - Note difference from veterinary meaning
    - Vertebral subluxation complex (VSC): fixation or mobility decrease in vertabrae adjacent to subluxation
    - Motor unit: VSC and associated structures
      - Associated structures:
        - Intervertebral disc
        - Ligaments

        - Muscles
        - Structures running through vertebral foramen
            - Spinal & meningeal nerves
            - Dural extension
            - Cerebrospinal fluid
            - Blood
            - Lymph vessels
            - Connective tissues
- Indications
    - Pain in neck, legs, back, tail
        - May occur even with normal disc position
        - Improved with treatment
    - Intervertebral disc disease
    - Cervical vertebral insufficiency
    - Cauda equine syndrome
    - Hip dysplasia
        - Early intervention
    - Urinary incontinence
    - Muscle spasms
    - Nerve issues
    - Injuries during accidents or performance
    - Jaw/chewing problems
    - Post-surgery
    - Chronic care
    - Maintainace of health spine/joints
        - Certain breeds
- Stages of misalignment (subluxation)
    - Trauma/initiating cuase
    - Kinesiopathy
        - Altered joint motion persisting after inflammation/pain from initial trauma resolve
            - Hypomobility due to initial trauma
            - Hypermobility due to compensatory mechanisms
        - Manifestations
            - Intervetertebral changes
            - Soft tissues adhesions
            - Calcium depostits
            - Altered CSF flow
            - Altereted proprioception
    - Neuropathy
        - Direct: compression of spinal nerve
        - Intdirect: due to edema, changes in blood/CSF flow
            - Especially nerves flowing through intervertebral foramen
        - Hyperactivity
            - “Facilitation”

        - Increase in end organ stimulation due to initial response to stress or damage
    - Hypoactivity
        - Inhibition
        - Decreased nerve supply to end organ
- Dysfunction
    - Tissue, neurological effects
- Clinical signs
    - Lameness, pain, muscle spasm, weakness, etc.
- Degeneration
    - Tissue destruction
    - Lessened likelihood of healing
- Compensation
    - Hypermobility
    - May be at joints or areas removed from initial trauma

- Initiating treatment
    - Clinical signs may manifest late in process
        - Decreased likliehood of healing as trauma progresses
    - Prevention/early intervention may be of greatest benefit
- Patient assessment
    - Postural, gait analysis
        - Hind legs in particular
        - Pelvic tilt
    - Eye level
        - Head tilt may be due to cervial subluxations/cranial bone assymetry
    - Palpation of muscle, soft tissue
    - Spinal temperature
    - Cornerstone of chiropractice exam: motion palpation
        - Reveals hypo-/ hypermobility
        - Planes of vertebral motion
- Types of chiropractic treatment
    - Diversified/ osseous
        - Most common
        - Bone adjustments
        - Manual or via activator: hand – held instrument
        - Popping less common than in human treatment
    - Non-osseous treatment
        - No application of force
        - Structural manipulation
            - Dura mater
            - Ligaments
            - Extremity joints
            - Ribs
            - Jaw

- Treatment considerations
  - Should be well-tolerated by patient
  - Possible even in larger animals
    - Relatively little force required
    - Faster thrust compensates for smaller mass required
  - Acute cases: weekly/biweekly treatment
  - Chronic cases: weekly treatment for 2 to 4 weeks
    - Taper off
  - Maintenance treatment: monthly to yearly
- Contraindications
  - Vertebral/pelvic fracture
  - Spinal neoplasia
- Precautions
  - Disc prolapse
  - History of back surger
- Recommending chiropractice care for patients
  - Should achieve immediate, noticeable results
  - Two to three treatments often achieve lasting results
  - Certification by American Veterinary Chiropractice Association
    - http://animalchiropractic.org/
    - http://www.avcadoctors.com/search_for_avca_certified_doctor.htm

- Low-level laser therapy
  - Use of lasers to alter cell function
    - Non-thermal (sometimes called “cold laser therapy”)
    - Non-destructive
  - Class III lasers: medium-powered
    - 5 – 500 mW; typically less than 100 mW
    - 632 – 1064 nm wavelength
      - Tissue penetration at shorter wavelength
      - Deeper penetration at longer wavelength
        - Cannot reach deeper than 0.5 cm
        - No deep tissue or joint effects
  - Wavelength based on type of laser
    - Ruby
    - Inert gasses (helium-neon, argon, krypton)
    - Semiconductor
  - Proposed benefits
    - Anti-inflammatory
      - Activation of micro-circulation
      - Changes in prostaglandins
    - Lipid peroxide stabilization
      - Activation of antioxidant compounds
    - Analgesia
      - Neuronal activation of metabolism
      - Increase in endorphins

- Wound healing
    - Activation of cellular metabolism
- Immunostimulation

- Inflammation
    - *In vitro* studies: reduction in PGE2 levels, COX-2 inhibition
    - Anti-inflammatory effects when lasers directed at synovia in patiens with chronic joint disorders
        - Does not penetrate joints
    - Proposed that microcirculation activated, leading to changes in prostaglandin levels
        - Reduction in edema
    - Animal studies
        - Prevention of neutrophil migration in acute lung inflammation in mice
        - Induced repair in vocal folds, reduced inflammatory markers in pulmonary inflammation in rats
        - No studies in companion animals
    - Analgesia
        - Related to anti-inflammatory effects
            - Increased blood flow
                - Increased oxygenation, lymphatic drainage, activity of neutrophils, macrophages, fibroblasts, cell metabolism
            - Increase in synthesis, release of endorphins
                - Increase in pain threshold
            - Inhibition of nociceptive receptors
            - Possible inhibition of nerve conduction
        - Animal studies
            - Analgesia demonstrated in mice
                - Believed to act on mitochondria
            - Increased endogenous opioid levels in rats
                - One study found comparable effects to NSAIDs
    - Wound healing
        - Believe to cause ATP accumulation and activation of cellular metabolism
        - Leads to increase in proliferation of fibroblasts and other compounds
            - Improves protein and collagen synthesis
            - Capillary formation
        - Animal studies
            - Accelerated wound closure in rats
            - Improved ligament strength in rats and mice
                - Including diabetic wounds
            - Increased vascularization in tibial fracture in mice

        - Increased collagen production in Achilles tendon, increase in action potential, diminished scar tissue in peripheral nerves in rats
        - Shortened time to ambulation after hemilaminectomy in dogs
        - Anti-inflammatory, improved wound healing after dental extraction in dogs
        - Accelerated healing of pharyngeal mucosal lesions in horses
    - Neurological issues
        - Quantum light absorption changes energy levels of biological structures
        - Mitochondria photoreceptors → increase in ATP
        - Activate transcription factors
            - Anti-apoptotic, antioxidant
        - Animal studies
            - Cortical inhibition in rats
- Contraindications
    - Only established in humans, strong caution in animals due to high likelihood of crossover
    - Tumors in target area
    - Epilepsy
    - Thyroid
    - Abdominal treatment in pregnancy
    - Thrombotic issues, especially in limbs

Evidence for Cannabinoid Treatment

Susan Elrod, BS, PharmD, PhD

Purpose:

This course will cover the evidence behind the use of cannabinoid products in veterinary medicine. The current state of research into this topic will be provided, as well as information regarding currently available veterinary cannabinoid products. This course is provided for information only; no endorsement of any particular product is intended or should be inferred.

Outline:

- Definitions
    - Cannabis
    - Cannabinoids
    - CBD
    - THC
- Rationale for use
- Veterinary research/evidence
- Available products
- Resources

Definitions:

- Cannabis
    - Encompasses three different plants:
        - *Cannabis sativa*
        - *Cannabis indica*
        - *Cannabis ruderalis*
    - Plants with psychoactive properties
    - Marijuana: dried flowers
- Cannabinoids
    - Compounds contained in cannabis
    - Approximately 120
    - Cannabidiol (CBD)
    - Tetrahydrocannabinol (THC)
- Cannabidiol (CBD)
    - Non-intoxicating/non-euphoric
    - Medical effects
- Tetrahydrocannabinol (THC)
    - Main psychoactive component
    - “High”-inducing ingredient

Rationale for use[1]

- Isolate the medical benefits of CBD and minimize intoxicating effects of THC
- Legalization occurring state-by-state
- Human studies:
    - Anxiety
    - PTSD

[1] https://www.projectcbd.org/science/how-cbd-works

    - Addiction
    - Schizophrenia
    - Epilepsy
        - FDA-approval: Epidiolex
- More than 65 receptors identified as being affected by CBD
    - Low affinity for CB1 and CB2
- Receptor-independent effects
    - Delays neurotransmitter reuptake
    - Affects binding of G-protein coupled receptors
- Anxiety effects
    - Activates serotonin receptor
        - Also responsible for inhibiting nausea and vomiting
        - Effects on addiction
    - Inhibition of adenosine reuptake
    - Enhances binding affinity of GABA-A receptor
- Pain effects
    - Activates serotonin receptor
    - Binds TRPV1 receptors: mediate pain and inflammation
- Cancer effects
    - Antagonist of GPR55
        - Promotor of cancer cell proliferation; expressed in several types of cancer
    - Activates PPARs
        - Anti-proliferative effect
- Epilepsy effects
    - Antagonist of GPR55
        - Modulator of neurotransmitters
            - Epilepsy:
                - Excitatory neurons: increased NT activity
                - Inhibitory neurons: decreased NT activity
        - Inhibition of calcium release
            - Decrease in excitatory currents associated with seizure activity
    - TRPV1 desensitization
        - Increase in seizure threshold
    - Inhibition of adenosine reuptake
        - Adenosine reduces excitability, neurotransmission

Veterinary Evidence

- Species
    - Dogs
    - Cats
    - Horses
    - Cattle
- Dogs
    - Veterinarians' opinions and use
        - Front Vet Sci. 2019 Jan 10;5:338.
        - Veterinarians believe that their states do not provide sufficient guidance

        - Believe marijuana, CBD offer benefits to humans, support CBD use in animals
        - Recent graduates
            - Less comfortable discussing CBD
            - Less likely to recommend or prescribe CBD
            - Believed CBD research in dogs is need
            - Believed CBD should not remain Schedule I
        - Significantly fewer veterinarians (45.5%) comfortable discussing CBD with clients versus discussing with colleagues (61.5%)
        - Those in states with legalized recreational marijuana more likely to discuss and recommend CBD use
            - No difference in likelihood of prescribing
            - Believed CBD research in dogs is need
            - Believed CBD should not remain Schedule I
        - Potential treatment for pain, anxiety, seizures
        - Oil and edible forms most commonly used
    - Pharmacokinetics/dosage forms
        - Four studies
            - Res Vet Sci. 2019 Apr;123:26-28.
            - Animals (Basel). 2019 Oct 19;9(10):832.
            - Biomolecules. 2020 Feb 11;10(2):279.
            - J Vet Pharmacol Ther. 2020 Sep;43(5):508-511.
        - Detectable levels of CBD with oral dosing
            - 7.5 mg (~0.6 mg/kg) oromucosal spray daily
                - T_{max} shortened with 14 day dosing compared to single dose
            - 2 mg/kg hemp product twice daily
            - Undetectable at 0.037 mg/kg
        - Transdermal administration
            - Detectable at ~2 mg/kg CBD twice daily
        - No major adverse effects reported
    - Osteoarthritis
        - Front Vet Sci. 2018 Jul 23;5:165.
            - Randomized, placebo-controlled, blinded, cross-over study
                - 2 week washout
            - Significant decrease in pain at 2 mg/kg oil by mouth every 12 hours for 4 weeks
            - No adverse effects reported by clients
                - Increase in alkaline phosphatase
        - Animals (Basel). 2020 Aug 26;10(9):E1505.
            - Significantly lower pain scores, improved quality of life with 2 mg/kg oral transmucosal administration every 12 hours
    - Epilepsy
        - J Am Vet Med Assoc. 2019 Jun 1;254(11):1301-1308.
            - Randomized, placebo-controlled, blinded clinical trial
            - n = 26

- Significant reduction in seizure frequency with 2.5 mg/kg oil by mouth every 12 hours for 12 weeks
    - Similar proportion of responders between groups
- Adverse Effects
  - Vet Clin North Am Small Anim Pract. 2018 Nov;48(6):1087-1102.
    - Accidental marijuana exposure to pets increasing with greater accessibility
      - Marijuana-containing food most common type reported to Pet Poison Helpline
      - Wide safety margin, good prognosis with proper treatment
    - Synthetic cannabinoids may cause more severe symptoms, fair prognosis
      - Tremors, aggression, seizures

- Cats
  - Pharmacology/mechanism of action
    - Pharmacol Biochem Behav. 1986 Jul;25(1):89-94.
      - Depression of electrophysiological responses in motorneurons
  - Pharmacokinetics
    - Animals (Basel). 2019 Oct 19;9(10):832.
      - 2 mg/kg CBD concentration by mouth twice daily for 2 weeks
        - Lower Cmax, AUC, longer Tmax compared to dogs
        - No clinically significant changes in CBC or serum chemistry
          - One cat showed elevated ALT levels
        - Adverse effects: excessive licking, head-shaking
  - Adverse effects
    - Vet Clin North Am Small Anim Pract. 2018 Nov;48(6):1087-1102.
      - Similar to dogs, see above
- Horses
  - Aust Vet J. 2020 Jun;98(6):250-255.
    - 1% CBD in various formulations of Manuka honey applied topically
    - n = 6
    - No effect on wound healing
      - Seen with Manuka honey in other experiments
- Cattle
  - Byproducts in feed
    - Sci Rep. 2020 Jul 29;10(1):12753.
      - Industrial hemp byproducts from CBD production
      - n = 8
      - 35 g industrial hemp, 5.4 mg/kg cannabidiolic acid
        - Cannabinoids detectable in plasma
        - Need for establishment of withdrawal times for food animals exposed to industrial hemp
  - Tick treatment
    - Exp Appl Acarol. 2020 Oct;82(2):281-294.
      - Topical preparation of extracts containing cannabis and garlic

        - Significant reduction in ticks with 45% preparation 4 days after treatment
    - Believed to be due to acetylcholine esterase inhibition properties of CBD and Vitamin E
- Summary of animal studies
    - Not much evidence of efficacy
    - Very few adverse effects
        - Good prognosis with supportive care
    - Cats appear to be more sensitive than dogs

Available products:

- Various human formulations available
    - Same cautions as with pharmaceutical drugs
        - Xylitol, etc.
    - Oils (oral and topical), treats, chews, etc.
- Veterinary specific products
    - NOT AN ENDORSEMENT
    - King Kanine, HempMy Pet, ElleVet, Green Roads, Canna-Pet, cbdMD

Resources:

- DVM360
    - https://www.dvm360.com/view/cbd-forget-legal-issues-does-it-work-veterinary-patients
        - Summary of veterinarian expertise and experiences
    - Dosage
        - 0.1 – 2 mg/kg twice daily
    - Route
        - Oral
        - Caution with transdermal/inhalation
            - Bypass first-pass metabolism
    - Selection
        - National Animal Supplement Council Seal of Quality Assurance
- Canine Journal:
    - https://www.caninejournal.com/cbd-oil-for-dogs/
    - Ranks CBD products
    - Provides pros and cons
        - Pros include third-party certification
- National Animal Supplement Council
    - https://nasc.cc/news/hemp-cbd-in-pet-supplements/

Veterinary Use of Probiotics

Susan Elrod, BS, PharmD, PhD

Purpose:
This course will cover the evidence behind the use of probiotics in veterinary medicine. The current state of research into this topic will be provided, along with information regarding currently available veterinary probiotic products. This course is provided for information only; no endorsement of any particular product is intended or should be inferred.

Outline:

- Definitions
  - Probiotics
  - Prebiotics
- Rationale for use
- Veterinary research/evidence
- Available products

Definitions:

- Probiotics
  - Live bacteria, yeasts
    - *Bifidobacterium*
    - Lactic acid bacteria strains
  - Beneficial to patient, especially for GI health
    - Maintenance of "good" bacteria in body
  - In fermented foods
    - Yogurt, sauerkraut, tempeh
- Prebiotics
  - Food for probiotics
  - Type of fiber indigestible by body
  - In fiber-rich foods: fruits, vegetables, whole grains
  - Inulin, oligosaccharides, etc.
- Synbiotic: combination of prebiotics, probiotics
- Both probiotics, prebiotics promote beneficial bacteria
- Doses expressed as colony-forming units per volume

Rationale for use[1]

- GI microbiome affects the physiology of the host
  - Wide-ranging effects
  - Affected by diet, environment, exercise, genetics
- Shown to ameliorate GI diseases
  - Diarrhea
    - Due to antibiotic use
    - *Clostridium difficile*
    - Nosocomial
  - Inflammatory bowel disease
- Other conditions

[1] Adv Nutr. 2019 Jan; 10(Suppl 1): S49–S66.

    - Atopic dermatitis
    - Allergic rhinitis
    - Metabolic disorders
        - Type 2 diabetes
- Mechanism of action being elucidated
    - Normalization of disturbed intestinal microbial profile
    - Competition with pathogens, toxins
    - Modulation of enzymatic activities associated with metabolism of xenobiotics
    - Production of short-chain, branched-chain fatty acids
        - Effect on insulin sensitivity
    - Immune system modulation
    - Regulation of endocrine, neurologic function

Veterinary Evidence

- Species
    - Dogs
    - Cats
    - Rabbits
    - Horses
    - Cattle
    - Pigs
    - Sheep
    - Goats
    - Poultry
- Dogs
    - No adverse effects reported
    - Variable/limited effects
        - Vet Intern Med. May-Jun 2009;23(3):476-81
        - Vet Ther. Fall 2010;11(3):E1-14
        - Biol Trace Elem Res. 2011 Jun;141(1-3):170-83
        - Arch Anim Nutr. 2013;67(5):406-15
    - Improvement in cholesterol levels, decrease in Pseudomonas-like bacteria
        - Folia Microbiol (Praha). 2006;51(3):239-42
    - Potential effects on gastrointestinal disease
        - Decrease in time to resolution of diarrhea
            - Vet Ther. Fall 2009;10(3):121-30,
            - J Small Anim Pract. 2010 Jan;51(1):34-8.
        - May decrease need for metronidazole treatment
            - Vet Ther. Fall 2009;10(3):121-30
        - Normalization of stool consistency, maintenance of appetite
            - Rev Esp Enferm Dig. 2012 Feb;104(2):65-8.
            - Vet Microbiol. 2016 Dec 25;197:122-128.
            - J Anim Physiol Anim Nutr (Berl). 2006 Aug;90(7-8):269-77.
        - Improvement in diarrhea associated with shelter facilities
            - J Vet Intern Med. 2017 Mar;31(2):377-382.
            - Top Companion Anim Med. 2017 Sep;32(3):100-103.
    - Normalization of dysbiosis associated with inflammatory bowel syndrome

        - PLoS One. 2014 Apr 10;9(4):e94699.
        - Gut Microbes. 2017 Sep 3;8(5):451-466.
        - Benef Microbes. 2018 Feb 27;9(2):247-255.
    - Improvement of enteritis
        - Vet Rec. 2018 Mar 3;182(9):258.
    - Variable effects on atopic dermatitis
        - Clinical signs
        - Improvement on immunological indicators
            - Am J Vet Res. 2009 Jun;70(6):735-40
        - Corticosteroid-sparing effects
            - Vet Dermatol 2015 Oct;26(5):350-3, e74-5.
    - Glomerular filtration rate preserved in patients with chronic kidney disease
        - Can Vet J. 2017 Dec;58(12):1301-1305.
- Cats
    - No adverse effects reported
    - Altered GI microflora
        - Believed to lead to improvement in immunomodulatory effects, decrease in morbidity
        - Preserved GI microflora in patients treated with antibiotics
        - Am J Vet Res. 2006 Jun;67(6):1005-12.
        - J Feline Med Surg. 2009 Aug;11(8):650-4.
        - Top Companion Anim Med. 2017 Sep;32(3):104-108.
    - Decrease in shelter-associated diarrhea
        - J Vet Intern Med. Jul-Aug 2011;25(4):856-60.
    - Increased likelihood in completing course of antibiotics
        - Due to increased food intake, decrease in vomiting
        - J Vet Intern Med. 2017 Sep;31(5):1406-1413.
- Rabbits
    - Two studies, n = 6 and n = 120
        - Modification of GI microflora, potential improvement in growth performance and immune function
        - J Small Anim Pract. 2014 Sep;55(9):442-6; J Anim Physiol Anim Nutr (Berl). 2017 Oct;101(5):e1-e13.
- Horses
    - Limited effects on GI microflora, nutrient digestibility, diarrhea
        - Particular caution with foals
        - J Anim Sci. 2008 Oct;86(10):2596-608; Equine Vet J. 2016 Nov;48(6):689-696; J Am Vet Med Assoc. 2005 Jun 15;226(12):2031-4; BMC Vet Res. 2015 Feb 14;11:34; J Vet Intern Med. May-Jun 2015;29(3):925-31; Vet Rec. 2013 Feb 2;172(5):128.
    - No effect on colic
        - J Vet Intern Med. Jan-Feb 1997;11(1):36-41.
        - J Am Vet Med Assoc. 2001 Mar 1;218(5):740-8.
    - Improved exercise performance
        - Vet Res. 1994;25(4):361-70.
        - J Appl Physiol (1985). 2018 Aug 1;125(2):654-660.

    - Significant decrease in severity, duration enterocolitis
        - J Am Vet Med Assoc. 2005 Sep 15;227(6):954-9.
    - Improvement in reproduction
        - J Am Vet Med Assoc. 2007 Jul 1;231(1):107-13.
- Cattle
    - Limited effects, some benefit to inflammatory response
        - J Anim Sci. 2007 Jan;85(1):233-9; J Anim Sci. 2016 Jan;94(1):297-305; Foodborne Pathog Dis. 2016 Apr;13(4):190-5; J Vet Med Sci. 2016 Nov 1;78(10):1595-1600; J Vet Intern Med. May-Jun 2006;20(3):614-9.
    - Improvement in calves' health
        - J Dairy Sci. 2016 Oct;99(10):8081-8089; Trop Anim Health Prod. 2016 Dec;48(8):1555-1560.
    - Variable, limited effects on milk production
        - J Dairy Res. 2012 Feb;79(1):16-25; J Anim Physiol Anim Nutr (Berl). 2012 Jun;96(3):506-12; Animal. 2013 Feb;7(2):216-22; J Anim Physiol Anim Nutr (Berl). 2018 Apr;102(2):e641-e652.
    - Improved immune response
        - J Dairy Sci. 2010 Dec;93(12):5851-5; Res Vet Sci. 2012 Jun;92(3):427-34.
    - Infectious disease
        - Significant decrease in infection of paratuberculosis, salmonella
            - J Dairy Sci. 2013 Oct;96(10):6535-8; Zoonoses Public Health. 2015 Dec;62(8):599-608.
- Pigs
    - Significant research on benefit against diarrhea in piglets
        - Res Vet Sci. 1999 Dec;67(3):223-8; Arch Anim Nutr. 2005 Dec;59(6):405-17; J Anim Physiol Anim Nutr (Berl). 2006 Feb;90(1-2):25-31; Pol J Vet Sci. 2011;14(1):117-25; J Anim Sci. 2014 Apr;92(4):1496-503; PLoS One. 2015 Jan 24;10(1):e0116635; Benef Microbes. 2015 Mar;6(1):41-4; J Appl Microbiol. 2015 Mar;118(3):727-38; Animal. 2015 Nov;9(11):1756-9; Animal. 2017 Jan;11(1):33-44; BMC Vet Res. 2017 Nov 15;13(1):335.
        - Improved daily intake, gain
        - Similar or improved effects compared to antibiotic administration
            - J Anim Sci. 2010 Dec;88(12):3880-6; J Anim Sci. 2017 Jun;95(6):2627-2639.
    - Improved performance
        - J Anim Sci. 2011 Jun;89(6):1795-804; J Anim Sci. 2015 Jan;93(1):405-13; J Anim Sci. 2017 Jan;95(1):308-319
        - Greater weaning weights, improvement in litter average daily gain for litters from sows supplemented with probiotics
            - J Anim Sci. 2013 Jul;91(7):3390-9.
        - Improved weight gain, carcass quality
            - J Vet Med A Physiol Pathol Clin Med. 2004 Aug;51(6):306-12; BMC Vet Res. 2012 Jun 25;8:89; Res Vet Sci. 2018 Apr;117:60-64.

        - Decreased need for antibiotics
            - J Anim Sci. 2012 Dec;90 Suppl 4:4-6
        - More diverse fecal microbiota in caesarean-delivered piglets
            - J Anim Sci. 2012 Dec;90 Suppl 4:433-5.
    - Improvement in immune response
            - Pol J Vet Sci. 2005;8(1):29-35; Anim Sci. 2011 Jan;89(1):52-8; Vet Res. 2012 Jul 27;43(1):58; Pol J Vet Sci. 2014;17(1):61-9; Vet Immunol Immunopathol. 2015 Mar 15;164(1-2):40-50; J Anim Sci. 2015 Nov;93(11):5313-26; J Anim Sci. 2018 Jun 4;96(6):2342-2351
        - May have increased immune response in vaccinated piglets
            - Vet Immunol Immunopathol. 2007 Jul 15;118(1-2):1-11.
        - Improvement in piglets before weaning when administered to both sows and piglets
            - Vet Immunol Immunopathol. 2007 Dec 15;120(3-4):136-47.
        - Prebiotics, probiotics have distinct effects
            - May be antagonistic when combined
            - Arch Anim Nutr. 2010 Aug;64(4):304-21.
            - J Anim Physiol Anim Nutr (Berl). 2010 Oct;94(5):e164-77.
        - Did not appear to overcome stress associated with crowding, enterotoxigenic E. coli
            - J Anim Sci. 2014 May;92(5):2017-29.
- Sheep
    - Two studies
        - Modification of ruminal methane emissions
            - Minor changes in other parameters
            - J Anim Sci. 2016 Feb;94(2):739-50.
        - Grape pomace significantly increased growth of probiotic bacteria, inhibition of pathogens in lambs
            - Along with antioxidant benefits
            - J Anim Physiol Anim Nutr (Berl). 2017 Oct;101(5):e108-e121.
- Goats
    - Two publications by same research group
        - J Anim Physiol Anim Nutr (Berl). 2014 Oct;98(5):879-85; Trop Anim Health Prod. 2016 Oct;48(7):1513-6.
        - Goats fed on oak leaves supplemented with *Streptococcus gallolyticus*
        - Improved growth performance, limited improvement in meat quality
            - No adverse effects on meat quality
- Poultry
    - 114 clinical trials
        - Gathered into review studies
    - May reduce salmonella infections when added to feed
        - Animal. 2012 Apr;6(4):557-64.
    - Improvement in growth performance, feed efficiency
        - Vet Immunol Immunopathol. 2018 Jul;201:1-11.

- Poultry-specific mechanisms proposed
  - Production of antibacterial substances
  - Immune system effects
  - Competition for adhesion receptor in epithelium
    - Attach to intestinal mucosa and prevent binding of pathogenic bacteria
    - Anim Health Res Rev. 2005 Jun;6(1):105-18.
  - Improvement of GI health, food safety
    - Appl Environ Microbiol. 2020 Jun 17;86(13):e00600-20.
  - Sparing of antibiotics as growth promoters
    - May not be more economically viable than antibiotics
    - Poult Sci. 2018 Nov 1;97(11):3807-3815.
- Inulin as a prebiotic
  - Variable/contradictory results
    - 5 – 10 g/kg feed seems beneficial
  - Increased proliferation of Bifidobacterium, Lactobacillus
  - Inhibition of pathogens
  - Improvement in immune response
  - Metabolism of minerals and lipids
  - J Anim Physiol Anim Nutr (Berl). 2016 Dec;100(6):1015-1022.
- In ovo supplementation
  - Beneficial effects of pre-/pro-/synbiotics
  - Improvement of immune and digestive systems
  - J Sci Food Agric. 2019 Jun;99(8):3727-3739.
- No adverse effects
  - Environ Sci Pollut Res Int. 2018 Nov;25(32):31971-31986.

Available Products

- Various probiotics, prebiotics, synbiotics
- Strains used in above studies
  - *Bifidobacterium, Lactobacillus*
- Products used in above studies
  - Fortiflora®
    - *Enterococcus faecium*
  - Bioracing®, Bovamine®
- Selection[2]
  - Host origin
  - Non-pathogenic
  - Stable during storage

Summary

- Unlikely to cause adverse effects
- May have immune, GI benefit
- Antibiotic-sparing for food animals

[2] 2003 Poultry Science 82:627–631

Back to the Basics: Approaching Poisonings Correctly, Right from the Start!

Renee D. Schmid, DVM, Pet Poison Helpline, Bloomington, MN

Proper Patient and Toxicity Assessment

When presented with a patient that has had a potential toxin exposure, it is important to make a full assessment of the patient and the toxin before starting treatment. Who, what, when, where, why and how are questions to ask in every toxicity situation. Common things to consider include:

Is the patient stable?

Assessing the patient's vitals and overall health status is often a key factor that is overlooked when presented with a panicked owner and patient. However, many steps that follow are critical in knowing the current condition of the patient.

What is the signalment? (species, breed, age, weight)

When dealing with dogs, certain breeds are at a higher risk for MDR-1 mutation which changes the toxicity severity for numerous medications including ivermectin. Washington State has an excellent website that discusses susceptible breeds and drugs that can be affected by the mutation, http://vcpl.vetmed.wsu.edu/problem-drugs.

Breed conformation may affect the type of decontamination that is recommended or appropriate. Very young or very old animals may have altered toxicity effects. An accurate weight is critical in assessing the degree of toxicity for most ingestions.

What is the current and past medical history including current medications?

Knowing the patient history and current status will help determine what type of therapy the patient can safely receive. If a patient has a history of CHF, administering 2-3x maint. IV fluid rates for a potential renal toxin would likely be inappropriate. Certain medications may interact or worsen toxicity signs depending on the exposure and should be known before starting therapy.

What was the toxin and route of exposure?

Knowing the specific toxin is crucial in many cases. Having a patient present to you that has ingested mouse bait is not a straightforward treatment of vitamin K1. Bromethalin, cholecalciferol and corn cellulose are not treated with vitamin K1. Also, different LAACs will have a different margin of safety and amount required to be ingested before toxicity would be expected.

The route of exposure important in determining the toxicity risk and severity. Was skin exposed to a liquid substance and then the animal licked it off or was it rolled in, walked through, etc.? Is there granular material stuck in the patient's haircoat that may pose a risk if ingested or allowed to remain the haircoat? Many toxins have a different degree of toxicity or treatment needs based on the route of exposure.

How much was the animal exposed to?

Not every toxin exposure will result in signs. It is true that "the dose makes the poison" and jumping to a conclusion that any exposure will require therapy often results in mismanagement and unnecessary stress to the patient, owner and treating veterinarian. While it is also true that there are a few toxins which may result in extremely significant signs at very low doses, i.e. mycotoxins, in general, there is a margin of safety for everything.

What is the time since exposure?

Knowing the time frame plays a large role in determining the type of decontamination that can be done or needs to be done. It also allows you to assess whether or not signs are likely to develop. If an animal has been exposed to a toxin 4 hours prior that would undoubtedly cause signs within a 1-2 hour timeframe yet remains asymptomatic, there is a strong likelihood that a toxic exposure did not occur.

Where did the exposure occur?

Knowing the location of the exposure is helpful at determining other potential confounding factors as well as the potential severity of a product. An animal that has been exposed to a fertilizer used on corn fields will likely have different needs than an animal exposed to a fertilizer placed on a residential yard.

What other factors are involved?

It is important to get the full story, or as much as one is able, in order to put together the most accurate picture as possible. Was the exposure accidental or malicious? Many times, instances arise where a toxin is suspected due to an incident and that becomes the focus instead of the big picture. An example of this is when an owner fertilized the yard 4 days ago and today their dog has started to vomit. The owner is focused on the fertilizer being the culprit but also forgot to mention that he ate the leftover pizza or that there has been company at the house and the dog is often stressed with increased anxiety when this occurs.

Staff safety in handling symptomatic patients

In the midst of urgency when presented with a potential toxicity situation, it is important to remember the safety of staff and take special precautions where needed. Dermal toxins that require decontamination should involve use of personal protective equipment including gloves, glasses or goggles and gown or long sleeves. While this may not be critical for bathing a cat that has had a permethrin product applied, it is very important when dealing with corrosives and hydrocarbons which may be damaging to the skin. Phosphide pesticides produce phosphine gas which, when inhaled, produces asthmatic-like symptoms as well as nausea and headache. The phosphine gas often smells like rotten eggs or garlic. Zinc phosphide used in gopher bait (“peanuts”) is a commonly seen form that is ingested in animals. When inducing emesis, precaution should be taken to ensure that the animal is either outside or in a well-ventilated area of the clinic to minimize inhalation of the gas. It is also important to instruct owners to ensure that the home or vehicle is well-ventilated in the event that the animal has spontaneous vomiting. Animals that have neurologic signs may be more likely to show aggression which may make treatment difficult. Even when animals appear to be stable, caution should be used when handling any animal that may develop neurologic signs as the development of signs may be sudden and unprovoked.

Ideal drugs for toxicity management

While the list of potential toxins to animals is long and covers a wide range of sources, medical management of cases can often be narrowed down to more commonly used drugs. The following are recommended drugs that are ideal to stock in your clinic and always have on hand.

Decontamination needs

Specific discussion of decontamination needs can be found in the Decontamination: It's More than Vomiting!" proceedings.

Ocular

If an animal has been exposed to a chemical that is an irritant or corrosive to the eye, ocular decontamination is warranted. As ocular decontamination should be started as soon as possible, owners should be encouraged to flush the eye at home with tap water. Eye drops should be avoided. Eye was can be used in the clinic in addition to tap water, with saline being the least preferred method.

Dermal

Rinsing product for 15 minutes and bathing with a degreasing dish soap 2-3 times will help to remove product. Burn/wound management should be used as needed.

Inhalation

For minor irritants, fresh air is generally sufficient treatment. Animals with underlying respiratory disease may require more intensive treatment, however. Oxygen therapy is often required for smoke inhalation, carbon monoxide and cyanide toxicity.

Gastrointestinal

Inducing emesis in dogs is performed by using apomorphine or 3% hydrogen peroxide. Apomorphine is a synthetic opioid that stimulates dopamine receptors in the CRTZ and can be given at 0.03mg/kg IV, 0.04mg/kg IM or by crushing ½ tablet for small dogs and 1 tablet for large dogs and placing in the conjunctival sac. Naloxone may be used if excessive sedation occurs without reversing vomiting. 3% hydrogen peroxide is given orally at a dose of 1-2ml/kg (1-2 tsp per 10 pounds). This should be fresh, bubbly and non-expired for effectiveness. Hydrogen peroxide is a gastric irritant and exceeding recommended amounts may result in gastritis with gastric bleeding. Ropinirole has recently been marketed as an emetic in dogs in the form of eye drops under the trade name Clevor®, to be given at a dose of Dose 2.7-5.4 mg/m^2 (average dose 3.75 mg/m^2).

Inducing emesis in cats is best performed by using an *a*-2 adrenergic receptor such as xylazine at 0.44mg/kg IM or dexmedetomidine at 7mcg/kg IM or IV. The sedative effects can be reversed with yohimbine or Anti-Sedan. Hydromorphone at 0.1mg/kg IM or SQ has also shown to have a good response in cats and is more cardiac sparing than an *a*-2 adrenergic agonist. Cats are more sensitive to developing hemorrhagic gastritis with hydrogen peroxide and is often not effective. Apomorphine is also not very effective in cats as the cat vomiting center is mediated by alpha receptors and not dopamine receptors. Apomorphine may cause dysphoria and agitation ("morphine mania") in cats.

The standard dosing of activated charcoal is 1-2g/kg, with 1g/kg being ideal in most situations. A cathartic is recommended to be given with the initial dose to help increase the rate of intestinal evacuation, and is included in many activated charcoal suspensions. Giving repeated doses of activated charcoal without sorbitol is valid for products that undergo enterohepatic recirculation or medications that have extended release properties. Doses are typically repeated every 6-8 hours for up to 24 hours, depending on the toxin. Sodium levels should be monitored and IV or SQ fluids given to minimize the risk of hypernatremia.

Neurologic management

Many toxins cause neurologic signs including agitation, hyperactivity, tremors, seizures, severe CNS depression and obtundation. In most cases, acepromazine at 0.05-0.1mg/kg is ideal for treating most situations of agitation and hyperactivity. This can be given IM or SQ. If given IV, ½ of the dose IV and the other ½ IM or SQ is common. However, there are animals with severe signs where giving the full dose IV would be appropriate. Butorphanol at 0.2-0.4mg/kg IM or SQ is ideal for treating agitation in an animal that may have a low normal blood pressure, be hypotensive, geriatric or where hypotension may develop, as is the case for albuterol and certain marijuana toxicities. Benzodiazepines are RARELY the treatment of choice for agitation and hyperactivity. These are typically contraindicated as the signs are often exacerbated.

Methocarbamol is the drug of choice for tremors that are a result of any toxin. The dosing range is wide at 55-220mg/kg. The preferred route of administration is IV. However, if only tablets are available, they can be crushed, mixed with saline and given rectally. The onset of action will be delayed compared to IV, but remains an acceptable option. Ideally, having a bottle of injectable methocarbamol on hand would be most preferred as signs can be more easily controlled and dosing can be titrated as needed. Benzodiazepines are typically not effective at tremor control and is not recommended.

Seizure control can often require more than one drug of choice. For instances where signs include agitation or hyperactivity, phenobarbital or Keppra is preferred over benzodiazepines as they often exacerbate agitation. In refractory seizure control, propofol or general anesthesia may be needed.

Cerebral edema may occur in cases of bromethalin ingestion and generally requires mannitol therapy in symptomatic cases. Hypoglycemia may develop in numerous instances of toxicity, with the most common being xylitol cases. 50% dextrose solution is imperative to have available for adequate management and control of hypoglycemia.

Renal management

Intravenous fluids may be beneficial for toxins that undergo renal excretion by increasing diuresis of the toxicant. Instances where this is helpful include toxicity of phenobarbital, amphetamines, salicylate, lithium and bromides. Due to NSAIDs being highly protein bound, fluid diuresis serves as a renal protectant.

Specific drugs for renal management needs are rare. In isolated cases, furosemide at 2-4mg/kg IV and mannitol at 1-2g/kg IV have been used to improve diuresis. In general, IV fluids are the treatment of choice for renal management with potential nephrotoxicities including grapes/raisins, NSAIDs and lilies (cats).

Hepatic management

Hepatoprotectants are often needed for toxins that may cause hepatic damage or necrosis. SAM-e containing supplements such as Denosyl® and Denamarin® are generally started after the toxic insult and continued for 2-4 weeks. N-Acetylcysteine is a hepatic detoxifier and used for the acute phase of toxicity.

Cardiac management

Cardiac signs in the poisoned patient typically include changes in heart rate, blood pressure and rhythm. Standard therapies used in other instances of cardiac changes are often used in instances of poisonings as well. It is recommended to review Plumb's or another formulary for more specific dosing instructions, but generally include:

Tachycardia:

Acepromazine: 0.05-0.1mg/kg IV, IM, SQ, used in conjunction w/ agitation +/- hypertension

Butorphanol: 0.2-0.4mg/kg IV, IM, SQ, used in conjunction w/ agitation +/- hypertension

Beta blockers:

Esmolol: 0.25-0.5mg/kg IV over 1-2 mins followed by CRI at 0.01-0.2mg/kg/min

Propranolol: 0.02mg/kg IV over 2-3 mins. Repeat in 20 mins until effect is seen, up to 0.1mg/kg

Metoprolol: 5-50mg TOTAL PO divided q8-12 hours

Bradycardia:

Atropine: 0.02-0.04mg/kg IV

Calcium Gluconate 10%: Reserved for calcium channel blocker toxicities. 0.5-1.5ml/kg IV slowly followed by 0.25-0.35ml/kg/hr CRI. Monitor ECG during administration

Hypertension: Systolic ≥160mmHg (Normal 120mmHg), MAP >130 (Normal 100mmHg)

Acepromazine: 0.02-0.1mg/kg IV, IM, SQ

Amlodipine (arterial vasodilator): Dogs 0.1-0.5mg/kg PO q 12-24 hrs. Cats 0.625-0.125mg PER cat q 12-24 hrs. May take several hours for full effect. DO NOT give if bradycardic.

Beta blockers:

Esmolol 0.25-0.5mg/kg IV over 2-5 mins followed by 10-200mcg/kg/min CRI

Propranolol: 0.02-0.1mg/kg IV q 8-12 hrs. Oral Dogs 0.1-1.0mg/kg q 8-12hrs, Cats 0.25mg PER cat PO q 8-12 hrs

Others include hydralazine, ACE inhibitors, isoflurane

Hypotension: Systolic ≤90mmHg or MAP ≤60mmHg. Normal 80mmHg or MAP 80mmHg

IV fluids: Crystalloids 20ml/kg bolus over 10-15 mins. Repeat 2-3x as needed

Colloids (VetStarch®) 5ml/kg bolus over 15 mins. Repeat 2-3x as needed

Vasopressors: Dopamine, norepinephrine, dobutamine, vasopressin, epinephrine, digoxin

Gastrointestinal management

Toxins that result in gastrointestinal signs are numerous. An anti-emetic such as metoclopramide (Reglan®) at 0.2-.0.5mg/kg IM, SQ, PO, maropitant (Cerenia®) at 1mg/kg SQ or ondansetron (Zofran®) at 0.1-0.2mg/kg SQ, IM, IV is a mainstay for general veterinary care and widely used for treating emesis resulting from GI toxicants. Anti-diarrheals including probiotics, metronidazole and high fiber sources are often needed for treating diarrhea caused by GI toxicants as well. In toxicities where ileus is a concern such as loperamide, metoclopramide has a dual benefit with its anti-emetic and pro-kinetic properties. If GI protectants are needed for potential GI ulceration, a

proton pump inhibitor (PPI) is generally preferred with an H2 blocker being used if a PPI is not available. Sucralfate and a PGE1 analog, such as misoprostol, are frequently used in situations where GI ulceration is of higher risk of occurrence.

Patient management and general follow-up needs

Patient management focuses on symptomatic and supportive care as well as minimizing toxin absorption. Once appropriate decontamination has been performed, home monitoring or continued in hospital care will need to be instituted. For toxins that may cause organ damage such as renal toxins and hepatic toxins, follow-up lab work is generally required following hospitalization or home management. Ingestions that result in hypercalcemia including vitamin D_3, cholecalciferol and calcipotriene often require follow-up therapy and lab work that may last for weeks to months in refractory cases. Thorough and consistent communication with the pet owner is imperative for successful outcomes and should be done for any toxin exposure even if additional hospitalization is not required.

Suggested reading:

Marks S, Kook P, Papick M, Tolbert M, Willard M. ACVIM consensus statement: Support for rational administration of gastrointestinal protectants to dogs and cats. J Vet Intern Med 2018:1-18.
Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology 2nd edition.
Peterson and Talcott Small Animal Toxicology 3rd edition.

Decontamination: It's More than Vomiting!

Renee D Schmid, DVM – Pet Poison Helpline Bloomington, MN

The goal of decontamination is to inhibit or minimize toxin absorption and to promote excretion or elimination from the body. It also allows us to remove or dilute topical irritants or corrosives. Consider the proper patient assessment as described above to determine if the benefits of decontamination outweigh the risk and whether the exposure will harm the patient. If decontamination is deemed to be warranted, selecting the appropriate method will help ensure successful management of the patient. Types of decontamination include:

Ocular

If an animal has been exposed to a chemical that is considered an irritant or corrosive to the eye, ocular decontamination is warranted. The pH will indicate if it is acidic or alkaline and there are often key words on the bottle such as caution or danger. As ocular decontamination should be started as soon as possible, owners should be encouraged to flush the eye at home with tap water. Eye drops should be avoided. Once in the clinic, a labeled eye wash is ideal, followed by tap water. Saline has not been shown to be beneficial in cases of alkali ocular burns.

If the product is an irritant the eyes should be irrigated for 10-15 minutes at home and monitored for signs of irritation including redness, lacrimation, pawing or rubbing at the eye, squinting or edema. The eyes should be irrigated for 15-20 minutes at home if a corrosive. Corrosive products should also have an additional 15-20 minutes of irrigation performed by a veterinarian followed by a fluorescein stain, topical antibiotic ointment or drops and use of an Elizabethan collar.

Dermal

Dermal decontamination is indicated for exposure to corrosives or irritants, glues or adhesives, gasoline/hydrocarbons and systemically absorbed toxins. This will help prevent oral exposure by self-grooming, remove unwanted substances, minimize paresthesia and reduce the risk of burns.

Irritant products generally have a caution statement on the label and result in mild redness and irritation. Rinsing product off or bathing with a degreasing dish soap is generally effective treatment. Vitamin E oil may also provide relief in situations where paresthesia is present. Corrosive products are alkaline or acidic in nature and generally have a danger statement on the label. These products result in chemical burns to the skin. Rinsing product for 15 minutes and bathing with a degreasing dish soap 2-3 times will help to remove product. Burn/wound management should be used as needed.

Glues and adhesives are typically non-toxic. They adhere to the eyelid, teeth, skin and fur. Certain types can be loosened with oil. The affected fur can be clipped if the animal is bothered to help avoid self-mutilation. Otherwise, if the affected area is not problematic, no therapy is generally necessary, and the product should wear off with time.

Gasoline and hydrocarbons are typically not seriously toxic. They may cause defatting of the skin resulting in cracking, secondary infections and chemical irritant contact dermatitis. Bathing 2-3 times with a degreasing dish soap is generally adequate therapy. There is a small risk for aspiration if oral exposure occurs and if inhaled, CNS depression may develop.

Systemically absorbed toxins do not generally cause dermal damage, however, result in systemic signs. These include tea tree oil, topical pain creams, estrogen creams, pyrethrin products (cats), psoriasis cream and 5-FU. Bathing 2-3 times in a degreasing dish soap will help to minimize absorption depending on the timing since exposure.

Inhalation

Toxins that may require respiratory decontamination include concentrates or corrosives, including bleach and ammonia mixtures, as well as smoke inhalation and carbon monoxide. A simple yet important aspect of inhalation decontamination is to remove the animal from the source of exposure. For minor irritants, fresh air is generally sufficient treatment. Animals with underlying respiratory disease may

require more intensive treatment.Oxygen therapy is often required for smoke inhalation, carbon monoxide, and cyanide toxicity.

Birds are very sensitive to inhalants, and fragrances, Teflon, and regular respiratory irritants may cause significant concern. The animal should be removed from the source, be given humidified oxygen, and offered heat support and fluids.

Gastrointestinal

Emesis

Emesis is by far the most common method of gastrointestinal decontamination. Approximately 49% (range of 9-75%) of stomach contents are recovered less than 30 minutes after ingestion. 17-62% is recovered 1 hour after ingestion. These ranges often make emesis success or failure difficult to assess, particularly when the ingestion is suspected but not known or when the number of items ingested is unknown as is often the case when an animal chews up a bottle of medication or eats a handful of raisins. If emesis is unproductive, it does not guarantee that the ingestion did not happen as emesis does not fully empty the stomach of its contents.

In many cases, there is a window of opportunity of only 1-2 hours for a positive return on emesis. However, there are certain toxins that can have successful emesis for up to 6 hours post ingestion. These include grapes, raisins, chocolate, xylitol containing gum, bezoars, massive ingestions and drugs that decrease gastric emptying (opioids, salicylates, anticholinergics and tricyclic antidepressants).

Inducing emesis in dogs is performed by using apomorphine or 3% hydrogen peroxide. Apomorphine is a dopaminergic receptor agonist drug that stimulates dopamine-2 receptors in the CRTZ and can be given at 0.03mg/kg IV, 0.04mg/kg IM or by crushing ½ tablet for small dogs and 1 tablet for large dogs and placing in the conjunctival sac. Naloxone may be used if excessive sedation occurs without reversing vomiting. 3% hydrogen peroxide is given orally at a dose of 1-2ml/kg (1-2 tsp per 10 pounds). This should be fresh, bubbly and non-expired for effectiveness. Hydrogen peroxide is a gastric irritant and exceeding recommended amounts may result in gastritis with gastric bleeding. Ropinirole has recently been marketed as an emetic in dogs in the form of eye drops under the trade name Clevor®, to be given at a dose of Dose 2.7-5.4 mg/m^2 (average dose 3.75 mg/m^2).

Inducing emesis in cats is best performed by using an *a*-2 adrenergic receptor agonist drug such as xylazine at 0.44mg/kg IM or dexmedetomidine at 7mcg/kg IM or IV. The sedative effects can be reversed with yohimbine or atipamezole. These drugs are approx. 50% effective in cats, may cause excessive sedation and, in rare cases, cause cardiovascular collapse. These are not generally recommended for older or disease compromised cats. Hyrdomorphone at 0.1mg/kg IM or SQ has also shown to have a good response in cats and is more cardiac sparing than an *a*-2 adrenergic agonist. Cats are more sensitive to developing hemorrhagic gastritis with hydrogen peroxide and is often not effective, therefore, not recommended. Apomorphine is also not very effective in cats as the cat CRTZ is mediated by alpha receptors and not dopamine receptors. Apomorphine may cause dysphoria and agitation ("morphine mania") in cats.

Products used in the past that should NOT be used to induce emesis include salt, syrup of ipecac, digital manipulation, liquid dish soap, raw eggs, Tabasco, or mustard. Salt toxicity, gastric irritation, nerve damage or aspiration may occur when other methods are used.

Emesis should not be induced in symptomatic animals, those that have already vomited to bile/clear, or those with a history of aspiration pneumonia or at risk for such due to laryngeal paralysis or megaesophagus. Examples of toxicity ingestions that should not have emesis induced include sharp/dangerous objects that may cause more trauma to the esophagus or enter the soft palate, corrosive agents (alkaline batteries, disc batteries, alkaline substances with a pH >11, acidic substances with a pH < 3) that may cause chemical burns to the esophagus and GIT, or hydrocarbons (gasoline, kerosene, motor oil) that present a moderate aspiration risk.

Caution should be taken if inducing emesis in brachycephalic breeds, young animals (less than 3 months of age), geriatric pets (greater than 10-12 years of age), animals with a history of heart disease,

seizures, recent surgery or those that have a non-toxic ingestion. Species that do not vomit include rabbits, ruminants (sheep, cattle, llama, goat), horses, birds and several rodents including chinchillas, rats and gerbils. Other decontamination methods will be needed for these species.

Gastric lavage

Gastric lavage may or may not be more effective at removing gastric contents. Often the more forceful contractions of the gastric muscles during emesis are more effective at removing contents than passive flow from lavage. This is a viable option for those species that do not vomit, symptomatic patients with a large ingestion, a large amount of stomach contents or where emesis was unsuccessful. It also may be helpful with potentially fatal ingestions including calcium channel blockers, beta blockers, baclofen, and metaldehyde.

Safe performance of gastric lavage requires sedation, intubation, and endotracheal insufflation. The animal should be in right lateral recumbency with the head tilted down at an approximately 20-degree angle. The stomach tube should be measured to the last rib, passed in to the stomach, and flushed with 5-10ml/kg warm water. The stomach should be agitated and then aspirated or allow for gravity to drain stomach contents. Once adequate removal of stomach contents is achieved, activated charcoal can be given. Caution should be used, however, as it is not uncommon for regurgitation to occur and the risk of aspiration is high. If activated charcoal is given, an anti-emetic, head elevation and continued intubation for as long as animal will tolerate until they can protect their airway should also be done.

Risks that are associated with gastric lavage include aspiration pneumonia, the need for general anesthesia, esophageal or gastric rupture and electrolyte imbalances. There are numerous contraindications to performing gastric lavage. These include hydrocarbon ingestions due to the high aspiration risk, corrosives, recent surgery (pending location), unstable patients, and those at a risk for bleeding or injury.

Activated charcoal and cathartics

Activated charcoal binds to many toxins in the GI tract by physical contact and weak covalent forces. Charcoal is a black powder composed of partially decomposed cellulose of soft wood. Activated charcoal is produced by heating common charcoal in the presence of a gas which creates numerous internal pores to trap chemicals within the activated charcoal. This process results in a highly porous material with an enormous surface area relative to its weight. The adsorptive capacity of activated charcoal is a function of its binding surface area. There is limited data regarding the benefit of activated charcoal with many toxins and one must weigh the risk vs. benefit when considering its use.

Benefits of activated charcoal include that it is readily available, relatively inexpensive, decreased absorption of 25-30% when administration is delayed, and beneficial use a wide number of toxins. Activated charcoal can be given with food to aid in administration without decreasing effectiveness. Drawbacks of activated charcoal use include difficulty of administration, potential vomiting after administration, potential diarrhea, binding to therapeutic medications, the unknown benefit with many toxins, and most importantly, the risk of hypernatremia. Hypernatremia may occur with any dose of activated charcoal, with an increasing risk as the number of doses increase.

There are numerous situations where activated charcoal use should be avoided based on the status of the animal. These include animals that are symptomatic, particularly with neurologic signs as aspiration risks are increased, animals with dehydration, current hypernatremia, hypovolemic shock, decreased GI motility/ileus, recent GI surgery and protracted vomiting. Activated charcoal should be avoided in instances where the risk of aspiration pneumonia is higher, including an unprotected airway, decreased level of consciousness, excessive sedation or agitation and when having to force feed. Activated charcoal should also be avoided in situations where endoscopy or abdominal surgery of the GI tract may be needed, concerns of a gastric or intestinal obstruction and ingestions where there is an increased risk of aspiration pneumonia, such as with caustic substances and hydrocarbons. Contraindications to activated charcoal use in general include exposures that occurred > 2 hours after ingestion unless enterohepatic recirculation occurs, or extended release formula medication was

ingested, alcohols (ethanol and ethylene glycol), xylitol, heavy metals, salt, paintball and non-toxic ingestions.

Cathartics are helpful at gastrointestinal decontamination for numerous reasons including accelerated speed of drug transit through the GIT, decreased time for toxin absorption, and decreased time for desorption of toxin from the activated charcoal. Sorbitol, a hexahydric sugar alcohol, is frequently combined in activated charcoal formulations at a dose of 3ml/kg PO of a 70% solution. Magnesium based cathartics (magnesium hydroxide, magnesium oxide, magnesium sulfate) should be used with caution in cats due to their increased risk of serum and brain magnesium levels. Hypermagnesemia displayed as hypotonia, ECG changes, altered mental status and respiratory failure may occur. It is also recommended to avoid magnesium-based cathartics in bromethalin toxicities due to potential similar clinical signs if hypermagnesemia were to occur. Magnesium hydroxide is often used in cases of mild iron toxicity due to its ability to precipitate binding of iron in the GIT to insoluble iron hydroxide.

The standard dosing of activated charcoal is 1-2g/kg, with 1g/kg being ideal in most situations. A cathartic is recommended to be given with the initial dose to help increase the rate of intestinal evacuation. Repeated doses of activated charcoal without sorbitol is valid for products that undergo enterohepatic recirculation or medications that have extended release properties. Doses are typically repeated every 6-8 hours for up to 24 hours, depending on the toxin. Sodium levels should be monitored and IV or SQ fluids given to minimize the risk of hypernatremia.

Whole bowel irrigation

Whole bowel irrigation (WBI) is rarely used in veterinary medicine. Situations when WBI may be helpful include enteric coated medications, iron ingestion, sustained or extended release medications and ingestions of packets of medications. WBI is performed using a nasoesophageal or nasogastric tube and administering 25-40ml/kg PEG-ES solution orally followed by a continuous oral infusion of 0.5ml/kg per hour until there is radiographic clearance or clear feces are present. Contraindications for WBI are like that of activated charcoal administration.

Endoscopy and surgical removal

Endoscopy may be indicated for ingestions of objects in situations where emesis would not be safe either due to the object size/shape or risk of oral/esophageal injury, such as ingestions of coins, non-leaking batteries, patches (fentanyl, lidocaine), bottles/plastic and metals. Endoscopy may also be warranted in evaluating injury to the esophagus and stomach. Negative aspects to endoscopy include the status of the animal if symptomatic, cost, equipment access and the need for general anesthesia.

When an animal is unable to vomit or if an object is not able to be removed endoscopically, surgery may be necessary for a successful outcome. Examples of this include leaking batteries, bread dough, a large number of objects and medication bezoars. Occasionally, surgery is required for removal of substances that do not pose a toxicity concern, however, a foreign body/obstruction concern. Sharp objects and large foreign bodies may require surgical removal. Gorilla Glue® has expansive properties and while toxicity is not seen, can form a hard, rock-like substance that encompasses the diameter of the stomach.

Suggested Reading:

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology 2nd edition.

Peterson and Talcott Small Animal Toxicology 3rd edition.

Niedzwecki, AH, et al. Effects of oral 3% hydrogen peroxide used as an emetic on the gastroduodenal mucosa of healthy dogs. JVECC, 2016; 0:1-7.

Willey, JL, et. Al. Evaluation and comparison of xylazine hydrochloride and dexmedetomidine hydrochloride for the induction of emesis in cats: 47 cases (2007-2013). JAVMA, 2016; 248:923-928.

Thawley, VJ. Assessment of dexmedetomidine and other agents for emesis induction in cats: 43 cases (2009-2014). JAVMA, 2015; 247:1415-1418.

FROM THE PLANTER TO THE CARPET: TOXIC PLANTS

Renee D Schmid, DVM, DABVT, DABT - Pet Poison Helpline Bloomington, MN

INTRODUCTION

For curious dogs or cats, it's difficult to resist the allure of plants. While many plants are simple gastrointestinal irritants, some have the potential to cause life-threatening toxicosis. Therefore, it is essential for technicians and veterinarians to be familiar with common plants that have the potential to cause severe toxicosis in dogs and cats.

LILIES

Plant name: Lily (*Lilium spp.* and *Hemerocallis spp*.)
Other common name(s): Easter lily, tiger lily, Japanese show lily, stargazer lily, rubrum lily, day lily
Species of concern: Cats only
Toxic dose: 1-2 leaves or petals, ingestion of the pollen from the fur or water from the vase
Toxic portion of plant: All, even the pollen
Onset/duration of clinical signs: Usually 6-12 hours post exposure.
Clinical signs: Early onset vomiting, depression, anorexia. Acute anuric renal failure in 1-3 days. Azotemia, epithelial cats (12-18 hrs post ingestion) proteinuria, and glucosuria. Pancreatitis is a rare sequela.
Treatment: No antidote. Induce emesis within 1-2 hrs after ingestion (xylazine is preferred in cats) followed by one dose of activated charcoal. IV crystalloid therapy at 2-3 times maintenance for 48 hrs. If oliguric, a furosemide CRI (1-2 mg/kg/hr) and mannitol bolus (1-2 g/kg) in well hydrated patients may be used. Gastrointestinal protectants as needed. Peritoneal or hemodialysis has been successful.
Prognosis: Good if treated early and aggressively; poor if treatment is delayed 18-24 hr or anuria has developed.

Asiatic hybrid lily (*Lillium spp.)* Photo courtesy of Tyne K Hovda, Pet Poison Helpline.

INSOLUBLE CALCIUM OXALATES

Plant names: Anthurium, flamingo flower (*Anthurium* spp.), Arrowhead vine (*Syngonium* spp.), Caladium (*Caladium* spp.), Calla lily (*Zantedeschia* spp.), Dumbcane (*Dieffenbachia* spp.), Peace lily (*Spathiphyllum* spp.), Philodendron, sweetheart vine (*Philodendron* spp.), Pothos, Hunter's robe, Devil's ivy (*Epipremnum* spp.), Umbrella plant (*Schefflera actinophylla*)
Other common name(s): Calla lily, Philodendron, Peace lily
Species of concern: All
Toxic dose: Varies with the plant. In general, a small ingestion can cause clinical signs.
Toxic portion of plant: All
Onset/duration of clinical signs: Onset rapid to within a few hours, lasting up to 24 hrs after ingestion.
Clinical signs/MOA: These plants contain insoluble calcium oxalate crystals (raphides) that are released from the plant (idioblasts) when chewed. Signs include oral irritation (salivation, redness, pawing), v/d, anorexia. Oropharyngeal swelling and dermal/eye irritation can occur, though rare.
Treatment: Dilute with water or milk, antiemetic, SQ fluids, GI protectants as needed.

SOLUBLE OXALATES CONTAINING PLANTS
Plant names: Common or garden rhubarb (*Rheum rhabarbarum*), Shamrock plant (*Oxalis* spp.), Sour Star Fruit (*Averrhoa carambola*)
Other common name(s): Rhubarb, Shamrock, Star Fruit
Species of concern: All
Toxic dose: Varies with the plant.
Toxic portion of plant: Varies. Leaves of rhubarb are toxic but not the stalk. All parts of shamrock and sour star fruit are toxic.
Onset/duration of clinical signs: Onset rapid to within a few hours, lasting several days after ingestion.
Clinical signs/MOA: Oxalic acid/oxalate salts cause GI irritation, bind Ca which leads to hypocalcemia & CaOx renal damage
Clinical signs: Lethargy, vomiting, anorexia, hypocalcemia, tetany, CaOx crystalluria, ARF
Treatment: Emesis, activated charcoal w/sorbitol, IV fluids for 48hrs, antiemetic, monitor renal enzymes/UA daily for 72hrs & hospitalize on IV fluids for 48hrs

CARDIAC GLYCOSIDES
Plant names: Foxglove (*Digitalis lannata and D. purpuea),* Oleander (*Nerium oleander)*, Lily of the Valley (*Convallaria majalis),* Kalanchoe (*Kalanchoe spp.)*
Other common name(s): Yellow Oleander, Lucky Nut, Be-Still Tree
Species of concern: All
Toxic dose: Varies with the plant. In general, just a few seeds or leaves (fresh or dried) are concerning.
Toxic portion of plant: All
Onset/duration of clinical signs: 45 min to a few hours after ingestion.
Clinical signs/MOA: These plants contain cardiac glycosides, similar to digoxin, and interfere with the Na/K pump mediated by ATPase. This results in an increase in intracellular sodium and a decrease in intracellular potassium with a resultant loss of myocardial function due to a decreased resting membrane potential. Common signs include salivation, vomiting, diarrhea and abdominal pain within 45 min as well as hyperkalemia and bradycardia with 1st, 2nd or 3rd degree AV block, ventricular arrhythmias, and asystole. Weakness and depression often precede cardiac abnormalities. Some, such as kalanchoe, may also cause neurological signs and mydriasis.
Treatment: Early emesis and activated charcoal (repeated q 4-6 hrs for 2-3 doses). ECG monitoring x 24 hrs if symptomatic. Judicious use of IV crystalloids. Atropine (0.02-0.04 mg/kg IV, IM, SQ) for bradycardia. Antiemetics such as maropitant, 1 mg/kg SQ q 24 h (not labeled for cats). Lidocaine or procainamide for persistent tachycardia (rare). A temporary pacemaker and digoxin-specific Fab fragments (Digibind®, 1-2 vials needed, ~$600/vial) have been used successfully in dogs.
Prognosis: Poor unless treated early and aggressively.

Foxglove (*Digitalis purpuea).* Photo courtesy of Tyne K Hovda, Pet Poison Helpline.

GRAYANOTOXINS
Plant name: Rhododendrons and azaleas (*Rhododendron spp.)*, Laurels (*Kalmia spp.)*
Other common name(s): Mountain Laurel, Sheep Laurel
Species of concern: All
Toxic dose: Ingestion of 0.2 % of animal's body weight. An adult human can eat 3 leaves/flowers without developing clinical signs.
Toxic portion of plant: All.
Onset/duration of clinical signs: Rapid (1-2 hrs), rarely delayed up to 12 hrs.

Clinical signs: These plants contain grayanotoxin glycosides which bind to sodium channels, increase their permeability and result in prolonged depolarization of cardiac muscle. Gastrointestinal signs such as hypersalivation, vomiting, diarrhea and abdominal pain predominate. Cardiac signs such as bradycardia or tachycardia, arrythymias, and hypotension may also occur. Rarely, CNS signs such as tremors, seizures, and coma can occur.
Treatment: No antidote. Early emesis and activated charcoal (repeated q 4-6 hrs for 2-3 doses). ECG and blood pressure monitoring x 24 hrs if symptomatic. Judicious use of IV crystalloids. Atropine (0.02-0.04 mg/kg IV, IM, SQ) for bradycardia. Antiemetics such as maropitant, 1 mg/kg SQ q 24 h (not labeled for cats). Lidocaine or procainamide for persistent tachycardia (rare).
Prognosis: Good with early intervention.

SAGO PALM
Plant name: Sago or cycad palm (*Cycas spp., Macrozamia spp, Zamias spp.)*
Other common name(s): Leatherleaf palm and Japanese fern palm. These are not true palms. Grown outdoors in warm areas, houseplants in cool areas.
Species of concern: Dogs
Toxic dose: 1-2 seeds are fatal in a medium sized dog. A “few bites” can cause poisoning.
Toxic portion of plant: All, especially the seeds, contain cycasin and other toxins.
Onset/duration of clinical signs: Variable (hours to days)
Clinical signs: Common signs include vomiting and diarrhea (+/- blood), lethargy, depression within hours. Acute, severe hepatic necrosis develops in -2-3 days. CNS signs (weakness, coma, seizures) are possible and may be related to liver failure.
Treatment: Induction of emesis and multiple doses of activated charcoal (q 4-6 hrs for 2-3 doses), IV fluids with dextrose, B vitamins and colloids as needed. Hepato protectants such as SAMe (load at 40 mg/kg/day PO for 3-4 days, then 20 mg/kg/day x 2 weeks) and/or silymarin (20-50 mg/kg daily PO). Broad spectrum antibiotics if liver necrosis. Diazepam (0.25-0.5 mg/kg IV PRN) for seizures. GI protectants as needed.
Prognosis: Good if treated prior to the onset of liver failure.

Suggested reading:

Toxic Plants of North America, 2nd ed. Burrows/Tyrl (2013)

Blackwell’s Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology 2nd edition.

Peterson and Talcott Small Animal Toxicology 3rd edition.

Handbook of Toxic Plants of North America, Burrows/Tyrl (2006)

A Guide to Poisonous House and Garden Plants, Knight (2006)

Common Psychiatric Pharmaceutical Poisonings

Renee D Schmid, DVM, DABVT, DABT – Pet Poison Helpline Bloomington, MN

Nearly half of the exposures managed by Pet Poison Helpline are to human drugs. Here we will discuss the most common prescription psychiatric exposures: Anti-depressants, ADHD treatments, and more. For each of these, we will review clinical signs of toxicity, diagnostics, and treatment.

Amphetamines and Methylphenidate

Prescription stimulants are commonly prescribed for treatment of attention deficit disorder in children and adults, narcolepsy, and sometimes weight loss. Common amphetamine drug brands include Adderall®, Dexedrine®, Desoxyn®, Dyanavel®, Dextrostat®, Evekeo® and Vyvanse®. Methylphenidate (Ritalin® and Concerta®) and dexmethylphenidate (Focalin®) are also commonly prescribed CNS stimulants. Novel formulations of these drugs are appearing on the market. Adzenys® XR-ODT is an extended release amphetamine in an orally disintegrating tablet designed to make administration easier for children or other people who have difficulty swallowing pills, and Daytrana® is a transdermal patch designed to deliver methylphenidate throughout the day. Fruit-flavored chews and solutions are also available. These stimulant drugs may come in immediate-release or an extended or sustained release formulation, as indicated by the letters XR, SR, ER, LA, or CD in the trade name.

While the range of toxicity varies amongst these drugs, clinical signs typically begin near a dosage of 1 mg/kg in dogs. Immediate-release drugs are rapidly absorbed, and clinical signs can develop 20-30 minutes after ingestion. Sustained-release products and transdermal patches (if swallowed whole) may result in a slower onset of action as well as a prolonged duration of clinical signs. Signs of intoxication involve CNS over-stimulation and excessive sympathomimetic effects such as agitation, vocalization, hyperactivity, hypertension, head bobbing, mydriasis, hyperthermia, tachycardia, tremors, and seizures.

Treatment is primarily symptomatic and supportive. Emesis should only be performed on asymptomatic animals and needs to be done promptly due to the rapid onset of clinical signs. Activated charcoal may be helpful, especially with sustained release products. Ingested patches may need to be retrieved by emesis, gastric lavage, or endoscopy. Maintaining control of agitation, hyperthermia, tachycardia, and tremors are key elements in these cases. Acepromazine (0.05-0.1+ mg/kg IV, IM, SQ) or chlorpromazine (0.5-1 mg/kg IV or IM) can be successfully used to achieve sedation and can be additionally beneficial in reducing heart rate, temperature, and blood pressure in agitated patients. It is recommended to start at the low end of the dosage range for sedatives and increase as needed. Some animals may require larger dosages than are listed here. Additionally, serotonin syndrome may occur and can be treated with cyproheptadine 1.1 mg/kg in dogs or 2-4 mgs total per cat orally or crushed into a slurry and delivered rectally. Benzodiazepines are typically avoided as they tend to result in paradoxical increased CNS excitement in these patients. Other commonly used interventions include injectable methocarbamol for tremors, injectable beta-blockers for tachycardia refractory to sedation, and IV fluids. Prognosis is generally good with prompt and aggressive treatment, though prolonged care may be needed, especially in large overdoses of extended release drugs.

Guanfacine and Clonidine

Guanfacine is a centrally active drug with alpha 2-adrenergic agonist properties used for treatment of ADHD. It is not a stimulant, and the mechanism of action in treating ADHD is unknown. It can be used alone for treatment or in combination with other stimulants. Common brand names for this drug are Intuniv®, an extended release formulation, and Tenex®, an immediate release form of the drug. Clonidine is another similar alpha 2-adrenergic agonist drug sometimes used off-label to treat ADHD, autism, and Tourette's Syndrome. Both clonidine and guanfacine were originally used as antihypertensive agents, and clonidine is also sometimes prescribed as a sleep aid, especially for children with sleep disturbances associated with ADHD or the stimulants prescribed to treat ADHD. Clonidine is now being used in dogs for certain behavioral conditions including phobias and separation anxiety.
Overdose of guanfacine and clonidine can result in clinical signs including depression, sedation, ataxia, vomiting, bradycardia, hypotension, and potentially seizures and tremors. Signs can develop at low

doses, and these drugs have a narrow margin of safety in pets. Signs are expected within 4 hours of exposure and can last 24-72 hours.
Asymptomatic patients may be induced to vomit and then given one dose of activated charcoal. In symptomatic patients, atipamezole, while not a true antidote, can be used to reverse signs of toxicity with these drugs and is typically dosed at 50 mcg/kg IM. Atipamezole will need to be re-dosed frequently as it typically lasts only 2-3 hours, while the effects of clonidine and guanfacine can have a duration of 24 hours or longer. IV fluids are warranted to maintain hydration, increase perfusion, and support blood pressure. Vital signs, especially heart rate and blood pressure, should be monitored frequently. If seizures occur, they can be treated with standard anticonvulsants.

Atomoxetine
Atomoxetine (Strattera™) is a non-stimulant SNRI (selective norepinephrine reuptake inhibitor) used as a second line treatment drug for ADHD. At low doses, signs of anorexia, sedation or agitation have been reported with potential for hypertension, tachycardia, and possibly tremors at higher doses. Signs usually develop within a few hours and can last 12-24 h. Cats and pets with liver disease are thought to be more sensitive to the effects of this drug.

With recent ingestion, induce emesis and then give one dose of activated charcoal. Treatment is symptomatic and supportive if signs develop with anti-emetics for nausea/vomiting, sedation for agitation, beta blockers if persistent tachycardia develops, and methocarbamol for tremors.

SSRI Antidepressants
Prescription antidepressants drugs routinely rank amongst the most commonly prescribed medications in the US and are increasingly used in veterinary medicine for a variety of behavioral disorders, including separation anxiety, storm phobias, inappropriate urine marking, stereotypic behaviors, and psychogenic alopecia. Common SSRIs include fluoxetine, citalopram (Celexa®), escitalopram (Lexapro®), paroxetine (Paxil®), and sertraline (Zoloft®). These drugs may come as either an immediate release or extended release formulation.

Selective serotonin reuptake inhibitors block the reuptake of serotonin in the presynaptic membrane, which results in an increased concentration of serotonin in the CNS. The range of toxicity varies depending on the drug and species. Small overdoses of SSRIs typically result in sedation or agitation, hypersalivation, vomiting, mydriasis, and tremors. Larger overdoses may cause tremors, seizures, nystagmus, dysphoria, vocalization, aggressive behavior, ataxia, and, bradycardia. As the degree of overdose increases, so does the risk for the development of serotonin syndrome, a toxidrome characterized by central nervous, autonomic, and neurobehavioral signs including agitation, vocalization, vomiting, diarrhea, muscle rigidity, increased reflexes, tremors, hyperthermia, hypertension, and transient blindness.

Treatment of SSRI overdoses is largely supportive and symptomatic. Appropriate decontamination with early emesis and activated charcoal is recommended if aspiration risk is low. Cyproheptadine, a serotonin antagonist, is useful in reducing the severity of signs, especially vocalization and dysphoria and is dosed at 1.1 mg/kg in dogs and 2-4 mg total dose per cat q 4-6 hours either orally or crushed into a slurry and delivered rectally. Agitation may be treated with acepromazine (0.05-0.2 mg/kg, IV, IM, or SQ PRN) or chlorpromazine (0.5-1 mg/kg, IV or IM PRN) starting at the low end of the dosage range and increasing as needed. Some animals may require larger dosages than are listed here. Benzodiazepines are typically avoided as they tend to result in paradoxical increased CNS excitement in these patients. Additional treatments include methocarbamol for tremors (55-220 mg/kg, IV slowly and to effect), IV fluids for thermal cooling and to maintain hydration and adequate perfusion, and beta-blockers (e.g., propranolol 0.02-0.06 mg/kg, slowly IV) for tachycardia and hypertension that is not corrected following appropriate sedation.

Overdoses of other antidepressants such as duloxetine (Cymbalta®), a SNRI, and venlafaxine (Effexor®), a bicyclic antidepressant, are clinically similar to SSRI overdoses. Cats seem particularly drawn to Effexor capsules and will readily ingest them. Treatment is similar to SSRI overdoses but more focus on sedation may be needed.

Tricyclic Antidepressants
Tricyclic antidepressants are another class of antidepressants used in human medicine as well as veterinary medicine for separation anxiety, other behavior conditions, excessive grooming or feather plucking, urinary conditions, pruritus, and neuropathic pain. Common tricyclic antidepressants include amitriptyline, clomipramine (Clomicalm®), nortriptyline, and doxepin.

These drugs have a narrow margin of safety, and anticholinergic effects can develop with overdose. Signs of toxicity may include constipation, urine retention, mydriasis, sedation vs agitation, disorientation, ataxia, arrhythmias, tachycardia, hypertension, vomiting, serotonin signs, and seizures. Treatment is similar to SSRI overdoses with decontamination, IV fluids, sedation if agitation develops, cyproheptadine if serotonin syndrome develops, antiemetics for vomiting and nausea, methocarbamol for tremors, and anticonvulsants if seizures develop. Close monitoring of vital signs, especially cardiovascular monitoring, is warranted Many tricyclic antidepressants are fat soluble, so treatment with intravenous lipids may be helpful in cases of severe toxicity.

Benzodiazepines and Non-Benzodiazepine Sleep Aids
Benzodiazepines are commonly used as anxiolytics, anticonvulsants, muscle relaxants and sedatives/hypnotics. Non-benzodiazepine hypnotics are typically used as sleep aids in human medicine. Although the two groups have different pharmacological profiles, both exert their effects through the inhibitory neurotransmitter gamma-amino butyric acid (GABA) and have similar clinical effects and treatment regimens. Common benzodiazepines used in veterinary medicine include alprazolam (Xanax®), diazepam (Valium®), lorazepam (Ativan®), midazolam (Versed®), and zolazepam found in combination with tiletamine as the dissociative agent (Telazol®). Other benzodiazepines used in human medicine include clonazepam (Klonopin®), oxazepam (Serax®), and temazepam (Restoril®). Common Non-benzodiazepine hypnotics include zolpidem (Ambien®), eszopiclone (Lunesta®), and zaleplon (Sonata®).

Both families of drugs have a relatively wide margin of safety, and fatality is unlikely to occur with acute overdose. Chronic oral use of diazepam in cats, however, can result in hepatic failure and should be avoided. Following ingestion, clinical signs of acute intoxication typically develop rapidly within 30-60 minutes and commonly include sedation vs paradoxical CNS stimulation (agitation), ataxia, confusion, and vomiting.

Treatment of acute ingestions consists of appropriate decontamination and supportive care. If necessary, the reversal agent or antidote, flumazenil, can be used but is rarely needed as these drugs are typically well tolerated. In symptomatic animals, monitor the body temperature and blood pressure and provide thermoregulation. IV crystalloids can be used as needed to maintain perfusion, treat hypotension, and correct dehydration. In cases of paradoxical stimulatory signs, additional benzodiazepines should *not* be administered as they will exacerbate the clinical signs. Instead, acepromazine (0.05-0.2 mg/kg IV, IM or SQ PRN) or butorphanol (0.1-0.5 mg/kg IV, IM, or SQ PRN) can be used. The reversal agent flumazenil (0.01 mg/kg, IV to effect PRN) is the antidote for benzodiazepine overdoses but is only necessary in rare cases of severe CNS or respiratory depression.

Lithium
Lithium carbonate and lithium citrate are used to treat bipolar disorder and as an adjunct to other antidepressants in humans, and its mechanism of action is not well understood. It has recently been tried as a treatment of anemia and neutropenia in dogs with bone marrow suppression, though with questionable efficacy. Lithium is a cation that competes with sodium, potassium, calcium, and magnesium at cellular sites, so animals with renal disease, dehydration, and sodium depletion can be more sensitive to this drug.
Acute overdoses of this drug are typically well tolerated with only mild vomiting, anorexia, and lethargy expected. Chronic overdose, which occurs rarely in pets, can be more serious, and signs may include lethargy, muscle rigidity, tremors, seizures, hypotension, arrhythmias, and bradycardia.
Emesis may be induced with recent ingestion. Activated charcoal is not effective at binding lithium. IV fluids can increase elimination of lithium, and 0.9% NaCl may be more effective at enhancing renal

excretion. Treatment is otherwise symptomatic and supportive with antiemetics for vomiting, anticonvulsants for seizures, and methocarbamol for tremors and muscle rigidity.

Lamotrigine

Lamotrigine (Lamictal®) is a phenyltriazine anticonvulsant that is also used to treat bipolar disorder in humans. Overdose of this drug can result in vomiting, lethargy vs hyperactivity, ataxia, weakness, tremors, seizures, hypokalemia, and acidosis. Arrhythmias, hypotension, and rare hepatotoxicity are also possible. This drug is rapidly absorbed with onset of action in most cases within 4 hours, and signs can last 24-48 hours.

Treatment of acute ingestions consists of appropriate decontamination and supportive care. Intravenous fluid may aid elimination and is also used for hydration and perfusion. Close monitoring of heart rate, EKG, and blood pressure are warranted. Ventricular arrhythmias may be treated with lidocaine or procainamide if needed. Antiemetics are used as needed for vomiting, and diazepam and/or phenobarbital if seizure activity develops. Very depressed or comatose patients may need monitoring of respirations and blood gas, and some dogs require ventilatory support.

Antipsychotics

Antipsychotic drugs are used in human medicine to treat bipolar disorder, schizophrenia, and other psychiatric and neurologic conditions in humans. Common drugs in this class include olanzapine (Zyprexa®), risperidone (Risperdal®), aripiprazole (Abilify®), and ziprasidone (Geodon®). Signs of toxicity with these drugs include agitation or lethargy, hyperesthesia, vomiting, diarrhea, hypotension, tachycardia, ataxia, vocalization and aggression, serotonin syndrome, and arrhythmias. Olanzapine can cause fluctuations between sedation and agitation. Animal studies of risperidone have shown that induction of emesis with apomorphine can be inhibited by this drug and may not be productive. It is also important to note that the “discmelt” version of Abilify® may contain xylitol.

These drugs are quickly absorbed with rapid onset of signs, and signs typically last approximately 24 hours in dogs. Treatment of acute ingestions consists of appropriate decontamination with emesis only in asymptomatic patients and activated charcoal only if low risk of aspiration. IV fluids are used for hydration and perfusion. Treatment is supportive with close monitoring of vital signs and blood pressure in symptomatic pets. Sedation is warranted in agitated pets, and if serotonin syndrome develops, cyproheptadine 1.1 mg/kg orally or rectally may be administered every 6-8 hours as needed. Tremors can be treated with methocarbamol and seizures with standard anticonvulsants.

Suggested Reading

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Know When to Feast and When to Fret - Foods Toxic to Pets

Renee D Schmid, DVM, DABVT, DABT – Pet Poison Helpline Bloomington, MN

Chocolate

The main toxic components of chocolate are two types of methylxanthines: theobromine and caffeine. Methylxanthines cause an increase in cAMP due to inhibition of cellular phosphodiesterase, stimulate the release of catecholamines from the adrenal medulla, cause competitive inhibition of adenosine, and increase intracellular calcium levels, which ultimately results in cerebral vasoconstriction, increased cardiac muscle contractility, and CNS stimulation. Theobromine is slowly absorbed by dogs and may take up to 10 hours before peak plasma levels are reached. The half-life of theobromine is also longer in dogs at 17.5 hours.

When assessing the toxicity of chocolate, it is important to remember that darker chocolates such as cocoa powder, unsweetened chocolate, and higher cocoa content dark chocolates contain more theobromine and are therefore more toxic to pets. Milk chocolate contains less theobromine and is better tolerated, and white chocolate contains minimal theobromine with minimal risk of toxicity. With chocolate ingestions, it is also important to get a good history to rule-out other potentially toxic co-ingestions such as marijuana, xylitol, macadamia nuts, alcohol, and raisins.

Theobromine toxicity will typically result in mild GI upset and excitement starting around 20mg/kg, moderate signs with potential for tachycardia at 40mg/kg, and potential for tremors and seizures at 60mg/kg. Due to the slow absorption of chocolate, induction of emesis is often rewarding even 6 hours after ingestion. Due to enterohepatic recirculation, multiple doses of activated charcoal are warranted, and IV fluids can help with excretion, as well as hydration and perfusion. Frequent walks or urinary bladder catheterization can keep the bladder empty to prevent reabsorption of metabolites in the urine. Treatment is otherwise symptomatic and supportive with close monitoring of vital signs, antiemetics for vomiting, sedation for agitation, beta blockers for sustained tachycardia, antiarrhythmics as needed, and anticonvulsants if seizures occur. Due to the prolonged half-life of theobromine, severely affected animals may remain symptomatic up to several days after ingestion.

Caffeine

Caffeine toxicity is possible when pets consume foods or drinks high in caffeine such as coffee, tea, and energy drinks; when they ingest caffeine pills or supplements that contain caffeine; and sometimes in novel forms, such as caffeine gum for alertness. Caffeine is rapidly absorbed, reaches peak plasma levels within 30-60 minutes, and has a half-life of 4 hours. Most common signs with overdose of caffeine include vomiting, diarrhea, CNS excitation, cardiac stimulation, hypokalemia, and hypertension. Mild signs are expected at 15 mg/kg of caffeine, moderate signs at 25 mg/kg, and severe CNS and cardiovascular signs at 50 mg/kg.

Treatment of caffeine toxicity is similar to chocolate toxicity. As caffeine is rapidly absorbed, emesis is generally not recommended in symptomatic patients, especially those as risk of aspiration. Due to enterohepatic recirculation of caffeine, multiple doses of activated charcoal can be useful, and IV fluids can help with excretion, as well as hydration and perfusion. Frequent walks or urinary bladder catheterization can keep the bladder empty to prevent reabsorption of metabolites in the urine. Treatment is otherwise symptomatic and supportive with close monitoring of vital signs, antiemetics for vomiting, sedation for agitation, beta blockers for sustained tachycardia, antiarrhythmics as needed, and anticonvulsants if seizures occur. Potassium should be monitored in cases of caffeine toxicity and may need to be supplemented in IV fluids if hypokalemia develops.

Xylitol

Xylitol is an increasingly popular, naturally-occurring sugar alcohol used as a sugar free sweetener in low calorie, sugar free, diabetic, and diet foods. It may be used alone as a sweetener in foods or in combination with other sweeteners such as aspartame, sugar, and/or other sugar alcohols. Xylitol is most

commonly ingested as an ingredient in sugar free gums and other premade foods but is also available in a powdered form for baking at home.

Xylitol is known to increase insulin release from the pancreas of dogs, which can result in hypoglycemia if ingested at doses of 0.1 g/kg and above. Elevated liver enzymes and liver necrosis may occur with large ingestions of > 0.5 g/kg xylitol. Cats are not considered to be sensitive to xylitol.

Xylitol is rapidly absorbed by the stomach and upper GI tract when ingested by dogs. It is safest to verify normoglycemia before inducing emesis, and activated charcoal is not indicated as it does not reliably bind to xylitol. If a hypoglycemic dose has been ingested, monitor blood glucose for at least 8 hours with the plan to supplement 2.5-5% dextrose as needed if hypoglycemia develops.

In dogs that have ingested a potentially hepatotoxic dose of xylitol, preemptive dextrose supplementation for 24-48 hours is thought to help mitigate liver toxicity. Treatment with liver protectants, such as SAM-e and N-acetylcysteine, are warranted in patients consuming hepatotoxic doses of xylitol. Liver enzymes are monitored every 12-24 hours for at least 48-72 hours or until they have normalized or at least plateaued. In cases of hepatic necrosis, secondary coagulopathy is possible, so coagulation monitoring and transfusion may needed.

Grapes and Raisins

Grapes, raisins, sultanas, and *Vitus spp.* currants (marketed as Zante currants) have been shown to cause renal failure in dogs. *Ribus spp.* currants are in the gooseberry family and do not cause renal toxicity in dogs. The toxicity risk to cats and ferrets is unknown, however considered lower risk with only rare anecdotal reports of toxicity. The mechanism of toxicity is unknown and is considered to be idiosyncratic. Grape seed extract appears to be safe and has not been shown to cause renal injury.

Vomiting is often an early sign of grape and raisin toxicity in dogs and usually develops within 24 hours of ingestion. 24-48 hours after ingestion, signs progress to lethargy, continued vomiting, inappetence and possible abdominal pain. Azotemia may occur within 24 hours of ingestion. Untreated, anuria and oliguria may be seen within 48-72 hours of ingestion. Decontamination is central in prevention of grape and raisin toxicity in dogs. As grapes are slowly absorbed, emesis can be performed up to 6 hours after ingestion followed by one dose of activated charcoal. Baseline lab work to assess current renal function, anti-emetics, and IV fluids for 48 hours are important components to therapy. Renal function should be monitored daily during hospitalization, and if azotemia occurs, IV fluids are continued for 24-48 hours after values have either returned to normal or stabilized. Prognosis is good in patients that are treated early and aggressively.

Onions and Garlic

Foods from the *Allium* species include onions, garlic, shallots, chives, scallions, and leeks, and if ingested in sufficient quantity by cats and dogs, they can result in oxidative hemolysis and anemia. For onions, ingestions > 5 g/kg in cats and 15-30 g/kg in dogs are considered toxic. Garlic is more potent, and doses of >1 g/kg is considered toxic for cats and > 5 g/kg is considered toxic to dogs. Clinical signs of toxicity include vomiting, diarrhea, lethargy, depression, abdominal pain, diarrhea, pallor, tachycardia, tachypnea, and icterus. Signs may develop within 1-2 days with a large acute exposure or up to several days with smaller exposures.

When toxic quantities of onions or garlic are consumed, treatment involves early induction of emesis and then one dose of activated charcoal. Baseline blood work should be checked with the plan to continue to monitor the PCV/CBC until it has normalized if anemia develops. Anemia typically resolves 10-14 days after ingestion. Any gastrointestinal signs can be treated supportively with fluids, anti-emetics, anti-diarrheals, and bland diet as needed. Antioxidants such as ascorbic acid, Vitamin E, and NAC have not been proven to provide any protective effects.

Macadamia Nuts

Ingestion of macadamia nuts by dogs can cause signs including vomiting, diarrhea, pancreatitis, lethargy, hyperthermia, weakness, tremors, lameness, and ataxia. Approximately 1 nut/kg body weight is considered toxic to dogs. Signs are usually self-limiting and typically resolve within 24-48 hours. This is a poorly understood toxicity, and dogs seem to be the only species sensitive to macadamia nuts.

Treatment involves early decontamination by inducing emesis and then one dose of activated charcoal for patients that have recently ingested a toxic amount of these nuts. For symptomatic patients, treatment is supportive with antiemetics if vomiting or nausea develop, fluids as needed for hydration, nursing care, supportive treatment if pancreatitis develops, thermoregulation, analgesia for lameness or joint stiffness, and methocarbamol if tremors develop. Prognosis is excellent with this toxicity and a full recovery is expected.

Unbaked Yeast Bread Dough

Rising, uncooked yeast dough is another category of potentially toxic food and would include any yeast dough to make bread, rolls, buns, or pizza. Dogs will readily eat entire loaves of rising bread dough, and unfortunately a dog's warm, dark stomach is an ideal environment for yeast to ferment, which results in production of both gas and ethanol (alcohol). When ingested, the dough rapidly expands in the stomach, gas is produced by the yeast, and dangerous distension of the dog's stomach can result. The alcohol produced by the yeast can also cause dogs to develop ethanol toxicity with signs including hypoglycemia, ataxia, weakness, and lethargy.

With recent ingestion in an asymptomatic patient, emesis can be induced to remove the dough. If the dough is not recovered with emesis, gastric lavage with cold water can also be attempted. Activated charcoal is not indicated and is not considered helpful for this toxicity.

Symptomatic patients should be hospitalized for monitoring and may need serial radiographs to assess passage of dough. In rare cases, surgical removal of the dough may be necessary. Intravenous fluids can be given for hydration and perfusion, and dextrose supplementation may be needed if hypoglycemia from ethanol toxicity develops. Vitals should be monitored closely and heat support may be needed for hypothermic patients. Metabolic acidosis can develop and is treated with IV fluids and potentially sodium bicarbonate if severe.

Alcohol

Pets, especially dogs, will readily drink unattended glasses of beer, wine, and other alcoholic beverages if given the opportunity. Dogs can also develop alcohol toxicity after eating alcohol-soaked desserts, like rum balls or cakes soaked in rum or bourbon. Ethanol can cause depression, weakness, ataxia, GI signs, and potentially hypoglycemia, hypothermia, acidosis with higher doses. Onset of signs is rapid as ethanol is rapidly absorbed.

Ethanol is rapidly absorbed, so emesis induction is rarely rewarding. Induction of vomiting should be avoided in symptomatic patients due to risk of aspiration. Activated charcoal does not bind to alcohol and is not indicated. Treatment is symptomatic and supportive with intravenous fluids for hydration and perfusion with dextrose supplementation if hypoglycemia develops. Vitals should be monitored closely and heat support may be needed for hypothermic patients. Metabolic acidosis can develop and is treated with IV fluids and potentially sodium bicarbonate if severe. Naloxone has been used with variable results to reverse CNS depression and may be redosed as needed if effective. Antiemetics may be given for nausea and vomiting. Prognosis is good, especially with early and appropriate treatment.

Avocado

Avocado is primarily toxic to birds, and all parts of *Persea americana* (fruit, pits, leaves, and stems) are considered toxic in affected species. Signs of avocado toxicity in birds include agitation, feather-pulling, fluffed feathers, lethargy, anorexia respiratory distress, pulmonary edema, respiratory collapse, pericardial

effusion, cardiac arrhythmias, and sudden death. Avocado toxicity has also been reported in rabbits with cases of non-infectious mastitis and agalactia reported in nursing rabbits, and cardiac arrhythmias, submandibular edema, respiratory distress, and sudden death developing in rabbits after ingestion of leaves. In dogs and cats, avocado may cause mild GI upset, rare pancreatitis, and if the pit is ingested whole, there may be concern for gastrointestinal obstruction.

Immediate care is indicated for birds that ingest avocado. Crop lavage followed by one dose of activated charcoal can be administered to asymptomatic birds. Recently exposed and asymptomatic rabbits can also be given one dose of activated charcoal (they cannot be induced to vomit). Treatment is symptomatic and supportive with oxygen, nutritional support, and diuretics for pulmonary edema. If mastitis develops, it is non-infectious, so antibiotics are not typically required. Warm compresses and analgesia can be helpful. Prognosis is poor once cardiac or respiratory symptoms occur in birds and rabbits.

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LETHAL LEPTOSPIROSIS: A PREVENTABLE DISEASE

Jane Sykes, BVSc(Hons), PhD, MBA, DACVIM (SAIM)
School of Veterinary Medicine – Dept. of Medicine & Epidemiology
University of California – Davis, Davis, CA, USA

INTRODUCTION

Leptospirosis is caused by several pathogenic species of the spirochete *Leptospira*. Organisms are transmitted by direct contact with infected urine, bite wounds or predation of infected wildlife, or indirectly, through contact with infected water, soil, food or bedding. Leptospires can survive several weeks in the environment when conditions are optimal, such as where there is a source of water (standing or rapidly moving) and temperatures between 0 and 25°C. The seasonality of the disease is variable depending on local rainfall patterns. In areas with year-round rainfall, the disease may occur throughout the year.

There are hundreds of pathogenic serovars, which are grouped into antigenically-related serogroups. In addition, classification of leptospires is gradually moving from serovar-based classification to genotype-based classification. Each serovar (and more accurately, each genotype) is adapted to a one or more mammalian host species (maintenance hosts). Other hosts act as incidental hosts. Disease in incidental hosts tends to be more severe and the duration of shedding is generally shorter. Maintenance hosts include dogs (Canicola); rats (Icterohaemorrhagiae); small wildlife mammalian species such as voles, skunks, and raccoons (Grippotyphosa); cattle (Hardjo); and mice (Ballum). The prevalence of infection with a serovar/genotype in dogs depends on the degree of contact between the dog population and the maintenance host for that serovar/genotype. In truth, the actual serovars causing disease in dogs worldwide still remain poorly characterized, but recent studies using culture and PCR have begun to shed more light on the true infecting serovars, and what domestic and wildlife reservoir hosts might be infected. Other factors, such as the density of reservoir hosts, the concentration of organisms in their urine, and specific leptospiral strains may also be important in determining whether disease occurs in dogs and humans.

Pathogenic leptospires penetrate abraded skin or mucous membranes and multiply rapidly, causing renal failure, hepatic injury and vasculitis. The disease is multisystemic and may also involve the pancreas (pancreatitis), gastrointestinal tract (gastroenteritis), eye (uveitis) and lungs (leptospiral pulmonary hemorrhage syndrome, or LPHS).

CLINICAL MANIFESTATIONS

Most infections are subclinical. Younger, large breed, outdoor adult dogs are commonly affected, but one study showed an increase in the percentage of small breed dogs diagnosed with leptospirosis between 1970 and 2009 (Lee et al, 2014), possibly because they are less often vaccinated. Younger animals tend to be more severely affected. We have seen an increase in the number of small breed dogs with leptospirosis that have not had a history of vaccination for leptospirosis.

Lethargy, anorexia, vomiting, pyrexia, dehydration, abdominal pain and increased thirst and urination are common signs of acute leptospirosis. Reluctance to move due to myositis or pancreatitis; icterus; punctate retinal hemorrhages and uveitis may be noted. Respiratory difficulty may result from pulmonary hemorrhage, which is often associated with the development of moderate anemia.

LABORATORY FINDINGS

Leukocytosis, thrombocytopenia, azotemia, hypoalbuminemia and mild to moderately elevated liver enzyme activities are common. Although hyperkalemia has been reported, normokalemia or hypokalemia are more common. Urinalysis may reveal isosthenuria, proteinuria, glucosuria and casts. Proteinuria is typically low-level (urine protein:creatinine ratio < 5), in contrast to dogs with Lyme nephritis, which have glomerular disease and higher ratios. Thoracic radiography may reveal a focal or diffuse interstitial to bronchointerstitial pattern; alveolar patterns may represent pulmonary hemorrhage. Hepatomegaly, splenomegaly, renomegaly and/or peritoneal effusion may be evident from abdominal radiography. Hyperechoic renal cortices and mild renal pelvis dilation are occasionally seen on ultrasound.

DIAGNOSIS

Identification of leptospirosis requires a high clinical suspicion for the disease. Currently available diagnostic tests include PCR, serology using the *microscopic agglutination test (MAT)*, and in-clinic serologic assays that detect IgG/IgM (SNAP Lepto, IDEXX Laboratories), or IgM (WITNESS Lepto, Zoetis). In the *MAT*, respective titers are provided for each of several different serovars in order to increase the chance of antibody detection. The MAT does not accurately predict the infecting serovar, and therefore *should not* be used for this purpose. Titers with any serologic test may be negative in the first week of illness. Positive titers early in the course of an illness may reflect residual post-vaccination titers or prior subclinical infection. *Demonstration of a fourfold rise in titer is required over a 1-2 week interval. In acutely ill dogs (< 1 week of illness), it is the author's opinion that leptospirosis serology should only be performed in a paired fashion or not at all, because of the limited utility of a single positive titer, regardless of its magnitude.* Postvaccinal titers against Icterohaemorrhagiae, Canicola, Grippotyphosa and Pomona occasionally rise as high as 1:6400 for a few months after vaccination, and these can interfere with interpretation. Use of a laboratory with a high level of quality control is recommended, or a laboratory that participates in the International Leptospirosis Society's proficiency testing scheme.

In-clinic serologic assays are useful for screening dogs for the presence or absence of antibodies. Should these kits yield negative results, it may be too early for the animal to have developed antibodies (as can occur with the MAT). Another test should be performed one week later to see if the animal seroconverts. The IDEXX assay detects IgG and IgM, and the WITNESS test detects IgM. Should these kits yield positive results, then the clinician should consider whether previous vaccination has occurred. Previous subclinical exposure should also be considered as a reason for positive results. Although the WITNESS test is less likely to be influenced by previous exposure or vaccination, some dogs can still be positive several weeks after vaccination. Clinicians should consider reflex testing with MAT in order to obtain a quantitative titer if positive results occur using in-clinic serologic tests, followed by convalescent serology 1-2 weeks later in order to document a change in titer. Additional clinical validation of these assays in different regions of the United States would be helpful to confirm their sensitivity and specificity.

The sensitivity and specificity of PCR may vary geographically depending on the serovars present and shedding patterns that occur for those serovars. The sensitivity may also be higher very early in the course of illness and in dogs that have not received any treatment with antimicrobials. PCR assays are best performed on blood AND urine concurrently because urinary shedding begins 10 days after the onset of infection.

TREATMENT

Treatment involves use of parenteral penicillin derivatives for leptospiremia, such as ampicillin (20 mg/kg IV q6-8h, adjusting dose down if severe azotemia is present) for up to 14 days or as long as the patient is vomiting. Treatment should then be changed to doxycycline (5 mg/kg PO q12h) for 2 weeks, in order to eliminate organisms in the kidney. Supportive therapy is also indicated for acute renal failure. The use of hemodialysis can improve survival in dogs with severe renal failure.

PREVENTION

In North America, vaccines are available for serovars Canicola, Icterohaemorrhagiae, Pomona and Grippotyphosa. The vaccines are safe and efficacious and several recent studies indicate they provide at least a 1-year duration of immunity.

Although it was prevalent when the two-way (Canicola and Icterohaemorrhagiae) vaccines were in widespread use, vaccine failure appears to be rare with the current 4-serovar vaccines (Hennebelle et al, 2013). With improvement in vaccines, the incidence of adverse reactions approaches that for distemper-hepatitis-parvovirus vaccines, even in small breed dogs. Vaccination against pathogenic leptospires is strongly recommended for dogs living in areas where leptospirosis occurs (ie. throughout the US), and are recommended even for small breed dogs that are confined to urban backyards, because of the possibility of infection as a result of rodent exposure. Minimizing access to rodents, farm animals and other wild animals also should help to prevent infection.

PUBLIC HEALTH RISK

Leptospirosis remains an important zoonosis, although most documented human leptospirosis in North America results from recreational activities that involve water, rather than contact with dogs. There are anecdotal reports of leptospirosis in staff that work in veterinary hospitals. Warnings should be placed on cages, and gloves should be worn while handling these dogs. At the author's hospital, contact precautions are lifted after 72 hours of specific antimicrobial therapy.

SUMMARY INCLUDING 5 KEY "TAKE HOME" POINTS

1. Leptospirosis can be transmitted through contact with infected urine, urine-contaminated water sources or predation.
2. Most humans in the United States contract leptospirosis through recreational activities involving water.
3. Leptospirosis is increasingly a problem in small breed, unvaccinated dogs.
4. New in-clinic diagnostic tests for leptospirosis are antibody tests that may be negative early in the course of illness or positive following recent subclinical exposure or vaccination.
5. Current 4-serovar vaccines have improved safety when compared with older vaccines and are recommended on an annual basis for prevention of the disease in all breeds of dogs.

References/Suggested Reading

References available on request

A Practitioner's Approach to Culture and Susceptibility Panel Interpretation

Jane Sykes, BVSc(Hons), PhD, MBA, DACVIM (SAIM)
School of Veterinary Medicine – Dept. of Medicine & Epidemiology
University of California – Davis, Davis, CA, USA

Introduction

In recent years, there has been a frightening rise in the prevalence of multidrug resistant bacteria in dogs and cats. Because of this, whenever possible veterinarians should make attempts to confirm a suspected bacterial infection by requesting microscopic evaluation of direct smears, culture and susceptibility by a laboratory before the choice is made to administer an antimicrobial drug. A Gram stain prepared from the specimen can permit the rapid preliminary diagnosis of infection, and provide information regarding whether the organism(s) present are gram-positive or gram-negative. This helps guide the clinician to select an appropriate empiric therapy, if necessary, while awaiting the results of culture and susceptibility testing.

Important terms used to describe resistant bacterial infections are as follows:

- Beta lactam: antimicrobial drug that includes a beta-lactam ring (all penicillins, cephalosporins and carbapenems such as meropenem). These bind to penicillin binding proteins (PBPs) (bacterial enzymes that catalyze bacterial cell wall formation) and cause bacterial lysis.
- Beta lactamases: bacterial enzymes that destroy the beta lactam ring (associated with resistance to beta-lactams). These include a variety of penicillinases. Beta lactamase inhibitors are drugs that inhibit these enzymes and include clavulanic acid and sulbactam.
- ESBLs: extended spectrum beta lactamases. These are bacterial enzymes that destroy critical beta-lactam drugs needed for treatment of resistant bacterial infections in humans (by definition, third generation cephalosporins such as cefuroxime, cefotaxime, ceftazidime). They are generally expressed by gram-negative enteric bacteria such as *E. coli* and *Klebsiella*.
- MRS: methicillin resistant staphylococcus. These organisms express an altered penicillin binding protein (PBP2a) that does not bind beta-lactam drugs. Therefore they are resistant to penicillins, cephalosporins and carpapenems.
- MDR: multidrug resistance. By definition, this is resistance to *3 or more CLASSES* of antimicrobial drugs (e.g., cephalosporins, fluoroquinolones, and aminoglycosides).

Methods Of Susceptibility Testing

Clinical microbiology laboratories will perform susceptibility testing for most aerobic bacteria, with the exception of streptococci. Streptococci from dogs and cats are almost always susceptible to penicillins. Most laboratories also do not routinely perform susceptibility testing on anaerobes, which also mostly have predictable susceptibilities, although resistance in anaerobes is increasing and some anaerobes, such as *Bacteroides fragilis*, have a high prevalence of β-lactamase enzyme production.

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Institute (CLSI) that specify medium composition and pH, inoculum size (determined on the basis of turbidity measurements), inoculation procedures, agar depth and incubation conditions, as well as quality control requirements. Because failure to comply with these protocols can lead to erroneous results, veterinarians should always attempt to use laboratories that follow CLSI or EUCAST protocols.

The most widely used dilution method is broth microdilution, whereby 2-fold dilutions of antimicrobials are made in a broth media in a microtiter plate. Pre-prepared frozen or freeze-dried plates are available commercially for inoculation (e.g., Sensititre® plates, TREK Diagnostic Systems). The results can be determined using visual examination of the plates for the inhibition of bacterial growth, or by the use of semi-automated or automated instrumentation. The MIC for each antimicrobial drug tested against the organism is reported to the clinician on the susceptibility panel. It is the lowest concentration of antibiotic (usually in μg/mL) that inhibits growth of the organism in vitro, and the lower the MIC, the more potent the antimicrobial is at inhibiting bacterial growth.

Diffusion methods include gradient diffusion (also known as Etest®) and disk diffusion. The *Etest* involves use of a plastic strip coated with an antimicrobial gradient on one side and an MIC interpretive scale on the other side. An agar plate is inoculated with the organism of interest so that subsequent growth of the organism will form a "lawn", rather than individual colonies. The strips are applied to the surface of the plate, with the lowest concentration towards the center. The antimicrobial drug diffuses into the medium, which results in an elliptical zone of growth inhibition around the strip. The MIC is read at the point of intersection of the ellipse with the MIC scale on the strip. Although the strips are expensive, Etests have the advantage of being adaptable to use with fastidious organisms and anaerobes if susceptibility testing of these organisms is deemed necessary. *Disk diffusion* involves application of commercially available drug-impregnated filter paper disks to the surface of an agar plate that has been inoculated to confluence with the organism of interest, and is also known as Kirby-Bauer antibiotic testing. Commercially available, mechanical disk-dispensing devices can be used to apply several disks simultaneously to the surface of the agar. The drug diffuses radially through the agar, the concentration of the drug decreasing logarithmically as the distance from the disk increases. This results in a circular zone of growth inhibition around the disk, the diameter of which is inversely proportional to the MIC. The zone diameters are interpreted on the basis of guidelines published by CLSI and the organisms are reported as susceptible, intermediate or resistant.

Breakpoints And Definition Of Susceptible Vs. Resistant Organisms

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Once a positive culture has been obtained, the veterinarian must consider the significance of the positive test result, even if susceptibility test results are reported. The detection of bacterial organisms within a sample does not always imply that the organism is causing the animal's clinical signs. Contamination is the most common cause of false positive cultures. Isolation of only one or two colonies of coagulase-negative staphylococci, *Bacillus* spp., *Corynebacterium* spp., and propionibacteria commonly suggest contamination. Isolation of large numbers of a single type of bacteria from a normally sterile site is generally clinically significant, especially when supported by cytologic examination of a stained smear that demonstrates the presence of bacteria within leukocytes.

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    a. *Will they achieve adequate concentrations at the site of infection?*
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4. *Is the antimicrobial drug currently being administered the most appropriate for the infection I am trying to treat?*

Because antimicrobial susceptibility testing results are generally not available until 2-3 days after submission of a specimen for culture, in animals that are critically ill, antimicrobial therapy may already have been initiated by the time those results are available. The susceptibility results may show that the organism is resistant to a drug being used, in which case the drug should be changed to one that the organism is susceptible to. The susceptibility pattern can also aid in choosing an alternate drug when the patient does not tolerate the initial drug prescribed. Susceptibility testing may indicate that the organism is susceptible to a more narrow-spectrum (and generally less expensive) antimicrobial drug than the drug initially prescribed, in which case the treatment should be changed to minimize the development of antimicrobial resistance.

5. Can I shorten the duration of therapy?

Currently there is a trend to try to shorten the duration of therapy to minimize selection pressure on bacterial populations. This differs from previous recommendations to 'finish a course' of antimicrobial drugs. In general, surgical prophylaxis should involve administration of antimicrobials only in the immediate peri-operative period (immediately before and during surgery).

Addressing the Antibiotic Resistance Problem: Which Antibiotic, When And For How Long?

Jane E. Sykes, BVSc(Hons) PhD DACVIM
University of California, Davis, CA, USA

Introduction

In recent years, there has been a frightening rise in the prevalence of multidrug resistant bacteria in dogs and cats. Because of this, whenever possible veterinarians should make attempts to confirm a suspected bacterial infection by requesting microscopic evaluation of direct smears, culture and susceptibility by a laboratory before the choice is made to administer an antimicrobial drug. A Gram stain prepared from the specimen can permit the rapid preliminary diagnosis of infection, and provide information regarding whether the organism(s) present are gram-positive or gram-negative. This helps guide the clinician to select an appropriate empiric therapy, if necessary, while awaiting the results of culture and susceptibility testing.

Important terms used to describe resistant bacterial infections are as follows:

- Beta lactam: antimicrobial drug that includes a beta-lactam ring (all penicillins, cephalosporins and carbapenems such as meropenem). These bind to penicillin binding proteins (PBPs) (bacterial enzymes that catalyze bacterial cell wall formation) and cause bacterial lysis.
- Beta lactamases: bacterial enzymes that destroy the beta lactam ring (associated with resistance to beta-lactams). These include a variety of penicillinases. Beta lactamase inhibitors are drugs that inhibit these enzymes and include clavulanic acid and sulbactam.
- ESBLs: extended spectrum beta lactamases. These are bacterial enzymes that destroy critical beta-lactam drugs needed for treatment of resistant bacterial infections in humans (by definition, third generation cephalosporins such as cefuroxime, cefotaxime, ceftazidime). They are generally expressed by gram-negative enteric bacteria such as *E. coli* and *Klebsiella*.
- MRS: methicillin resistant staphylococcus. These organisms express an altered penicillin binding protein (PBP2a) that does not bind beta-lactam drugs. Therefore they are resistant to penicillins, cephalosporins and carpapenems.
- MDR: multidrug resistance. By definition, this is resistance to *3 or more CLASSES* of antimicrobial drugs (e.g., cephalosporins, fluoroquinolones, and aminoglycosides).

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Guidelines for Treatment of Urinary Tract Infections in Dogs and Cats

The International Society for Companion Animal Infectious Diseases (ISCAID) Antimicrobial Guidelines Working Group was formed to develop guidelines for antimicrobial drug use in dogs and cats, because of concerns that antimicrobial drug resistance has dramatically increased in prevalence among isolates from dogs and cats in the last decade. The founding members of the ISCAID Working Group are Scott Weese, Joseph Blondeau, Dawn Boothe, Edward Breitschwerdt, Luca Guardabassi, Andrew Hillier, Michael Lappin, David Lloyd, Mark Papich, Shelley Rankin, Jane Sykes, and John Turnidge. Input has also been obtained from panels of Diplomates of relevant specialty groups. It should be noted that members of the working group receive support from a variety of industry groups that provide funding for honoraria and research. Guidelines for treatment of urinary tract disease in dogs and cats were initially published in 2011 as open access documents (www.iscaid.org). During the course of guideline development, it became clear that there is a significant lack of objective, published information. Accordingly, recommendations have been based on available data, whenever present, along with expert opinion, considering principles of infectious diseases, antimicrobial treatment, antimicrobial resistance, pharmacology, and internal medicine. Since the first publication of the urinary guidelines, the Guidelines Working Group has revised and updated them based on new evidence available in veterinary and human medicine. The information below does not represent the final recommendations of the Working Group, but provides an update for practitioners on the current perspective of the author as part of the Guidelines Working Group.

Sporadic Cystitis

Sporadic cystitis is a sporadic bacterial infection of the bladder in an otherwise healthy individual with normal urinary tract anatomy and function. A clinically significant urinary tract *infection* (UTI) implies the presence of clinical signs of lower urinary tract disease - in other words, dysuria, hematuria, pollakiuria, and/or stranguria. Complete urinalysis should be performed for all cases and quantitative aerobic culture and susceptibility (C&S) testing is encouraged. Free-catch samples should be avoided due to the potential for contamination by commensal bacteria. Recommendations for initial treatment are amoxicillin (11 – 15 mg/kg PO q12h) or trimethoprim-sulfonamide (15 mg/kg PO q12h). If culture and susceptibility testing reveals a resistant isolate and there is a lack of clinical response, treatment should be changed to an appropriate antimicrobial drug. Although treatment has been recommended in the past for 7 to 14 days, recent research suggests 3-5 days may be more appropriate. There is no evidence that intra- or post-treatment urinalysis or urine culture is indicated in the absence of ongoing clinical signs of UTI.

Recurrent Bacterial UTI

Recurrent bacterial UTIs are defined by the presence of 3 or more episodes of UTI during a 12-month period or 2 or more infections within a 6-month period. When recurrent UTIs are diagnosed, efforts should be made to identify the underlying cause. Primary care veterinarians should consider referral for a work-up that might include abdominal ultrasound and

cystoscopy. Treatment should be based on the results of C&S testing, with initial empiric therapy following the recommendations for Sporadic Bacterial Cystitis.

Although 4 weeks has been recommended for treatment, shorter durations are likely to be recommended in the future, with a focus on *clinical cure* (resolution of clinical signs of lower urinary tract disease) rather than microbiological cure (negative bacterial urine cultures). There is insufficient evidence to recommend "pulse" or chronic low-dose treatment, urinary antiseptics, and nutritional supplements such as cranberry juice extract for prevention of UTIs.

Subclinical Bacteriuria

Subclinical bacteriuria is the presence of bacteria in the urine as determined by positive bacterial culture, in the absence of clinical signs of UTI. Treatment of subclinical bacteriuria is generally not recommended, but could be considered if there is a high risk of ascending or systemic infection (eg. patients with renal disease).

Urinary Catheters

Proper aseptic placement and maintenance of urinary catheters is critical. Open collection systems should be avoided. If clinical signs of lower UTI (gross hematuria or pyuria) or pyelonephritis (fever and leukocytosis with or without azotemia) are absent, then no culture or treatment is indicated. The duration of catheterization should be as short as possible. Catheter removal is not necessary in the presence of subclinical bacteriuria. There is no indication for routine use of prophylactic antimicrobials after the catheter is removed. If clinical signs of a UTI or fever are present, then perform a urine culture by collecting urine after replacement of the urinary catheter with a new catheter. Several milliliters of urine should be removed to clear the new catheter first before a specimen is obtained for culture. Alternatively, the catheter can be removed, the urinary bladder allowed to fill, and a cystocentesis performed. Culture from the collection bag, and culture of the catheter tip after removal are not recommended. Treatment should follow the guidelines for sporadic bacterial cystitis, and is more likely to be successful after catheter removal.

Pyelonephritis

For animals with suspected pyelonephritis, C&S testing should always be performed. Treatment should be initiated while awaiting culture results, using antimicrobials effective against Gram-negative *Enterobacteriaceae*. A fluoroquinolone is a reasonable first choice, after which treatment should be based on C&S results. If combination treatment was used initially and C&S results indicate that both drugs are not required, the spectrum should be narrowed. Treatment for 4 weeks has been recommended, but it is likely that shorter durations of treatment (10-14 days) may be effective. Culture is recommended 1-2 weeks after treatment is discontinued, together with a physical examination and assessment of azotemia, but the possibility of subclinical bacteriuria should be considered when interpreting culture results.

Medical dissolution of struvite urolithiasis

Urine culture should be performed in all animals with urolithiasis. Culture of surgically-removed uroliths can be considered, but the clinical relevance of the results may be unclear. If evidence of bacterial cystitis is present, antimicrobial drug selection should be approached as for Sporadic Cystitis. Seven days of treatment is recommended for animals with urolithiasis and concurrent bacterial cystitis. The need for further treatment requires further study. Urine culture after completion of medical urolith dissolution is not recommended in the absence of clinical signs of lower urinary tract disease. Confirmation of elimination of uroliths through diagnostic imaging and investigation of predisposing factors for cystitis (and subsequent struvite urolith risk) is important.

Bacterial prostatitis

Empirical treatment of bacterial prostatitis should target gram-negative *Enterobacteriaceae*. Administration of a veterinary fluoroquinolone such as enrofloxacin should be considered while awaiting culture and susceptibility testing results. Use of trimethoprim-sulfonamide can be considered but is not recommended where a fluoroquinolone can be used because of the greater risk of adverse effects with the typical duration of treatment. Limited data are available to guide duration of treatment. Four weeks is typically recommended for acute prostatitis, with 4-6 weeks for chronic disease. Shorter durations might be effective in acute cases that are castrated and where there is a rapid clinical response to therapy. A longer duration of treatment may be required in some chronic cases, particularly when abscessation is present or when castration is not performed. Castration should be recommended in dogs that are not intended for breeding. Poor initial response to therapy should lead to re-assessment of the diagnosis. If prostatitis is still suspected, consideration of collection of ultrasound-guided fine needle aspirate of prostatic cyst fluid or prostatic tissue core biopsy for culture and cytology or histopathology. Prostatic abscesses should be drained because of the low likelihood of resolution with medical treatment alone. If necessary, surgical drainage should be performed after culture results are available, whenever possible, to facilitate proper peri-operative antimicrobial therapy.

Urological surgery, minimally invasive urological procedures and urologic implants

Bacterial culture of urine collected by cystocentesis is indicated before cystoscopic procedures or laparoscopic or open urologic surgery. If bacteriuria is identified, treatment based on susceptibility result is recommended for 3-5 days immediately before the procedure to reduce bacterial counts. Peri-operative antimicrobial prophylaxis should be considered for procedures that involve stone manipulation or open surgical procedures that involve the urinary tract, although this is controversial. When antimicrobial prophylaxis is indicated, the antimicrobial(s) should be administered intravenously within 60 minutes of the procedure and be re-dosed intra-operatively after 2 half-lives of the drug have passed (when applicable), in order to target the time that bacterial invasion is most likely to occur. Typically, this is until wound closure or completion of an endoscopic procedure. In the absence of complicating factors or infection, peri-operative prophylaxis should not continue for greater than 24h.

TAKE HOME POINTS

1. UTI is associated with clinical signs of lower urinary tract disease; subclinical bacteriuria is bacteriuria without clinical signs of lower urinary tract disease
2. Consider withholding antimicrobial treatment for animals with subclinical bacteriuria, provided there is no suspicion for pyelonephritis
3. Aim to identify and treat the underlying cause when recurrent cystitis is present
4. Shorter courses of treatment than previously recommended are likely effective for UTI (3-5 days) and pyelonephritis (10-14 days)
5. Whether antimicrobial therapy is needed throughout the dissolution period for animals with struvite urolithiasis is controversial and requires further study.

The Microbial Cocktail: Update on Canine and Feline Infectious Respiratory Infections

Jane Sykes, BVSc(Hons), PhD, MBA, DACVIM (SAIM)
School of Veterinary Medicine – Dept. of Medicine & Epidemiology
University of California – Davis, Davis, CA, USA

INTRODUCTION

Infectious respiratory tract disease (IRD) remains a major problem in shelter, breeding and boarding kennel environments, despite widespread vaccination against the pathogens that contribute to disease. As a result of improvements in diagnostic testing, there is increasing awareness of mixed infections in affected animals. In environments such as shelters, co-infections with a variety of different viruses and bacteria may be more common that infections with a single pathogen, especially in dogs. In addition, several pathogens have emerged in recent years as important contributors to canine infectious respiratory disease complex (CIRDC) in kennel and shelter situations.

Pathogens causing IRD can help each other to infect the host. For example, canine distemper virus causes profound immunosuppression, which predisposes dogs to infection with other respiratory viruses and bacteria. Severe disease is more likely to be associated with co-infections. Single infections may be present in some animals that show no signs of illness. Similar findings have been reported in children with community-acquired pneumonia.

CANINE INFECTIOUS RESPIRATORY DISEASE DIFFERENTIAL DIAGNOSIS

Understanding the differential diagnosis for CIRD is important because it aids selection of appropriate diagnostic tests, the design of rational therapy, and permits institution of proper preventative measures for CIRD. There are now over 10 organisms known to play a role in canine infectious respiratory disease.

Bacterial causes of canine infectious respiratory disease include *Bordetella bronchiseptica*, *Streptococcus equi* subspecies *zooepidemicus*, and *Mycoplasma* spp. Viral causes of canine infectious respiratory disease include influenza viruses, canine distemper virus, canine respiratory coronavirus, canine parainfluenza virus, canine adenovirus (especially canine adenovirus-2), canine pneumovirus, and canine herpesvirus.

Establishment of a specific etiologic diagnosis is generally not necessary in dogs that are otherwise healthy but just have the characteristic, ‘honking’ cough of the kennel cough syndrome. The vast majority of these dogs will have self-limiting infections, with clinical signs generally resolving within 5-7 days without antimicrobial therapy. Some dogs may require a short course of antimicrobial therapy, but it is recommended that antibiotic treatment be withheld if uncomplicated infection is present and clinical signs have been present for less 10 days. A cough suppressant such as hydrocodone could be considered in this situation, but cough suppression is contraindicated in dogs with complicated disease (moist cough, pulmonary infiltrates, fever, lethargy, inappetence).

Diagnostic testing is indicated if

- An outbreak has occurred.
- Affected dogs are systemically unwell.
- The cough is persisting despite treatment.

Establishment of a diagnosis can help with control and prevention in kennel situations, and appropriate antimicrobial therapy for dogs with bacterial infections, e.g., *Bordetella bronchiseptica* infections. Some *B. bronchiseptica* infections can be refractory to treatment with systemic antimicrobial drugs. This may result from antimicrobial resistance or inadequate drug penetration to the site of infection.

Clinical signs are not useful for diagnosis of a specific infectious agent, because the signs are overlapping and non-specific, and mixed infections are commonly present. Diagnostic tests available for diagnosis of canine infectious respiratory disease include culture for bacteria and mycoplasmas, blood tests (serology) for antibody against canine influenza virus and canine distemper virus, and polymerase chain reaction testing of throat swabs or respiratory lavage specimens for the DNA and RNA of respiratory viruses and bacteria. Many laboratories offer canine respiratory disease PCR *panels*. This has led to increased detection of canine infectious respiratory pathogens and an increasing awareness of co-infections. Virus isolation in culture is cumbersome and is not widely offered for routine diagnostic purposes. Sometimes a diagnosis is best obtained by combining multiple different diagnostic modalities.

Culture remains a useful test for bacteria such as *Bordetella bronchiseptica* and mycoplasmas, although the growth of mycoplasmas can be slow and unreliable. Culture also allows susceptibility testing for *B. bronchiseptica*, as some strains may demonstrate antibiotic resistance.

The results of serologic testing may be difficult to interpret as a result of prior vaccination. Vaccination can help to reduce the severity of disease but does not prevent infection.

EMERGING AND RE-EMERGING RESPIRATORY PATHOGENS OF DOGS

Bordetella bronchiseptica

Bordetella bronchiseptica is the most common bacterial agent causing CIRD, and tends to cause moderate signs of CIRD. Infection is best diagnosed via transtracheal washing or bronchoalveolar lavage, but occasionally throat swabs or nasal washings/swabs will be positive. Both culture and PCR assays are available for detection of *Bordetella bronchiseptica*. Parenteral, intranasal or oral vaccines are available to help to prevent bordetellosis, but the relative efficacy of these vaccines STILL remains unclear. Dogs vaccinated with parenteral vaccines require two doses given 4 weeks apart for initial protection, and protection does not become effective until one week after the second dose. Only a single dose of an intranasal vaccine is required. Annual boosters are indicated thereafter for both parenteral and intranasal vaccines. Inadvertent administration of intranasal or oral *Bordetella* vaccines can lead to cutaneous abscesses or life-threatening systemic infections and death, so it is particularly important to pay attention to the vaccine type and the proper route of administration. If inadvertent administration of these vaccines occurs, immediate treatment with doxycycline is indicated; immediate subcutaneous administration of gentamicin and crystalloids at the site of inoculation has also been advocated. *Bordetella bronchiseptica* has the potential to cause respiratory disease in immunocompromised humans, but there is no clear evidence that the organisms in canine avirulent live vaccines are capable of contributing to human illness.

Streptococcus spp.

Streptococcus equi subspecies *zooepidemicus* is a beta-hemolytic streptococcus that has caused outbreaks of acute suppurative or necrotizing hemorrhagic pneumonia in shelter situations.

Streptococcus canis can be found in the lungs of both healthy dogs and dogs with kennel cough, whereas *S. equi* is rarely found in healthy dogs. Whether it acts as a primary pathogen or secondary invader is not clear, but in a recent outbreak from California, the consistent presence of co-infection was not documented. It is rarely isolated from household pets. No vaccine is available.

Mycoplasmas

Mycoplasmas are normal flora in the respiratory tract of dogs, but are occasionally isolated from dogs with infectious respiratory disease without evidence of coinfection. The primary mycoplasma associated with lower respiratory disease in dogs may be *Mycoplasma cynos*. Other mycoplasmas have been isolated from the respiratory tract of dogs, but these have not been definitively associated with lower respiratory disease. Molecular techniques have improved our ability to detect mycoplasmas, but we still have trouble knowing whether a positive result is associated with disease. No vaccine is available.

Influenza viruses

Influenza viruses are enveloped viruses with segmented single-stranded RNA genomes that belong to the family *Orthomyxoviridae*. Influenza viruses that cause disease in domestic animals belong to the genus *Influenzavirus A.* Influenza A viruses are classified based on the composition of their hemagglutinin (H) and neuraminidase (N) genes. To date, 18 H types and 11 N types have been identified, each of which are antigenically distinct. Genomic rearrangements that occur within influenza A viruses allow for occasional cross-species transmission. These occur when two different viruses simultaneously infect a host, with subsequent genetic reassortment. Occasionally, cross-species transmission occurs without alteration of the viral genome. The names of influenza viruses are specified as follows: influenza genus (A, B or C)/host/geographic origin/strain number/year of isolation and, in parentheses, H and N type. For example, A/canine/Florida/43/2004 (H3N8).

In the USA, canine influenza virus (CIV) emerged in racing greyhounds in Florida in 2003 and 2004, where it caused hemorrhagic pneumonia and a high mortality. Serological evidence of infection in the greyhound dog population dates back to 1999. Infections spread slowly and have subsequently been reported in racing greyhounds and non-greyhounds in at least 38 US states. Outbreaks continued to occur in shelter situations for nearly a decade after the virus was discovered, but now the virus appears to be destined for extinction, if it is not extinct already. The virus that has circulated in the USA is an H3N8 virus that resembles an equine influenza virus, which suggested that an interspecies jump occurred without genetic reassortment. Instead, accumulation of point mutations with minor amino acid changes occurred, followed by sustained transmission among dogs. The most significant outbreaks of disease due to CIV have occurred in Florida, New England, Colorado, Wyoming, and Texas. In other states, sustained transmission of the virus from one dog to another has not occurred. The most significant risk factor for infection has been indoor housing. Virtually all cases to date have involved dogs in kennels, animal shelters or dog day care facilities. Dogs of all ages and breeds are susceptible, but to date severe hemorrhagic pneumonia has only occurred in greyhounds. The virus is shed for up to 7 to 10 days, but is typically shed for just a few days. In some dogs, shedding may have ceased when clinical signs are most apparent. CIV can still infect horses, but horses develop only mild disease or no clinical signs.

Canine influenza virus H3N2 was first detected in March of 2015 in Illinois and Michigan and was likely imported from Korea. Additional importation events have occurred since and contributed to outbreaks in California. Disease caused by H3N2 is more severe than that caused by H3N8 and the shedding period appears to be longer (21 days).

Although infections with influenza viruses may be more likely to produce signs of fever and lethargy than dogs infected with other respiratory pathogens (e.g., *Bordetella bronchiseptica*, canine respiratory coronavirus, canine distemper virus, canine herpesvirus, canine adenovirus 2, canine parainfluenza virus), it is not possible to diagnose influenza virus infections in dogs based on clinical signs alone. The high prevalence of co-infections and increased severity of disease when multiple pathogens are present further complicates diagnosis. A history of exposure to other animals with respiratory disease can raise suspicion for the diagnosis.

When outbreaks occur, attempts to make a diagnosis are indicated. Collection of multiple specimen types (oropharyngeal swabs, nasal swabs, and if possible transtracheal or bronchoalveolar lavage specimens) from several dogs with and without clinical signs can facilitate diagnosis and allow interpretation of the significance of positive test results. Organism detection methods, such as PCR, are likely to be of highest yield early in the course of illness (e.g., the first 1 to 3 days), or in exposed dogs that have not yet developed clinical signs. Using a combination of serology and organism detection methods (culture or PCR) may also facilitate diagnosis. Necropsies can provide valuable information, and should be performed as soon as possible after death or euthanasia occurs by a veterinary pathologist. Tissues should be submitted for histopathology (in formalin), bacterial and virus cultures (fresh tissue), and/or PCR for respiratory viruses and bacteria. Despite the increased availability of molecular diagnostic assays, virus isolation is still offered to veterinarians for routine diagnostic purposes by some veterinary diagnostic laboratories that specialize in virology (e.g., the Animal Health Diagnostic Laboratory at Cornell University in the USA).

Panels of real-time PCR assays that detect respiratory pathogens may include assays for CIV. Unfortunately, false negative PCR results are common because of transient or low-level shedding of many respiratory viruses. In addition, because influenza viruses are RNA viruses, false negatives may result from degradation of viral RNA during specimen transport.

Point-of-care assays are available for detection of nucleoprotein antigen to human influenza A viruses. Unfortunately, such assays have limited sensitivity and specificity for diagnosis of influenza virus infections in dogs.

Serological assays for CIV exposure are based on serum neutralization or hemagglutination-inhibition. Serology is of limited use for diagnosis, because of vaccine titer interference in regions where vaccination is performed, and the high prevalence of subclinical exposure in regions where infection is endemic. Titers may be negative in the first 10 days of illness. Despite these limitations, serological assays have been key to identification of outbreaks of disease caused by CIV, when the disease is not endemic and widespread immunization has not yet been performed. Analysis of paired serum specimens collected 2 weeks apart can be used to document recent infection. In some dogs, no other diagnostic test may be useful for antemortem diagnosis because virus shedding is so transient and difficult to detect. Assays for CIV that use equine influenza virus antigen for antibody detection have suboptimal sensitivity.

Treatment of influenza virus infections is supportive. The efficacy and optimal dosage of neuraminidase inhibitors like oseltamivir is unknown, and because oseltamivir is a first line treatment for pandemic influenza in humans, it should not be used to treat dogs with respiratory disease, even when CIV infection is known to be present. In the United States, inactivated,

parenteral vaccines are available for reduction of disease caused by H3N8 CIV and H3N2 CIV. Their use has been recommended for dogs that may contact other dogs in regions where CIV is endemic. Vaccination against CIV is also required for importation of North American dogs to Australia. The initial vaccine may be given as early as 6 weeks of age. Because CIV vaccines are inactivated, 2 initial doses are required 3 to 4 weeks apart, and maximum immunity does not occur until 1 week after the second dose. As a result, CIV vaccines may not protect dogs that enter shelters with endemic canine influenza.

There is currently no evidence of zoonotic transmission of CIV. However, a recent study revealed that a variety of human influenza viruses infect the canine trachea, and that reassortment of these viruses with CIV results in viable viruses. Thus dogs have the potential to be sources of novel viruses that could lead to influenza virus pandemics in humans.

Canine Respiratory Coronavirus

Canine respiratory coronavirus is a more recently discovered virus that represents another cause of respiratory disease in dogs worldwide. It has similarity to a cow coronavirus but is distinct from canine enteric coronavirus (for which vaccines are available). Its presence tends to correlate with mild disease, but it has been detected in outbreaks of severe respiratory tract disease. Infection with canine respiratory coronavirus may predispose to other bacterial and viral infections, but may also potentially be a primary pathogen. It can be detected using PCR on transtracheal or bronchoalveolar lavage specimens, or throat swabs. Currently, no vaccines are available to prevent this infection.

Canine Distemper Virus

Canine distemper virus is another important cause of kennel cough, and it can also cause neurologic or gastrointestinal signs. However, many dogs with distemper lack neurologic or gastrointestinal signs. It is probably vastly undiagnosed as a cause of kennel cough in dogs. Canine distemper virus can be detected using PCR on respiratory specimens. It can also be detected using PCR on whole blood or conjunctival scrapings. Canine distemper virus vaccines are part of the core vaccine series. Three initial doses (6-8 weeks, 10-12 weeks, and 16-18 weeks) are required, after which an annual booster is indicated followed by boosters every 3 years. Recently, increasing numbers of distemper cases have been described in adult, previously vaccinated dogs, including in outbreak situations. The reason for this remains unclear, but careful handling and storage of vaccines that contain canine distemper virus is important to preserve their efficacy. Administration of the third puppy dose earlier than 16 weeks of age may also contribute to vaccination failure due to interference by maternal antibody.

Canine Parainfluenza

Canine parainfluenza virus remains the most important viral cause of CIRD in dogs, and intranasal and parenteral non-core vaccines are available and in widespread use for prevention of infection. Again, the relative efficacy of these types of vaccines is not well understood. Other viral pathogens include canine adenovirus (for which vaccination is available and used as a core vaccine for prevention of infectious canine hepatitis), and canine herpesvirus. Canine herpesvirus is also a cause of conjunctivitis and keratitis in dogs.

Canine Pneumovirus

First described in the United States in 2010, there is growing evidence that canine pneumovirus is a significant cause/contributor to CIRD worldwide.

FELINE UPPER RESPIRATORY DISEASE UPDATE

The usual pathogens continue to be a problem in feline upper respiratory tract disease – feline herpesvirus 1, feline calicivirus and *Chlamydia psittaci*, which *C. psittaci* primarily causing problems in multiple cat households where there are cats under a year of age; *Mycoplasma felis* and *Bordetella bronchiseptica* may also be involved in some situations, but these organisms can also be found in healthy cats. The availability of oral famciclovir and topical cidofovir has improved treatment of cats with severe herpesvirus infections. There are no antiviral drugs available for treatment of calicivirus infections. For sick cats with mucopurulent discharges, the ISCAID Antimicrobial Working Group recommends the first choice antibiotic as doxycycline (see accompanying notes on antimicrobial choices for respiratory disease).

SUMMARY

In conclusion, an increasing number of pathogens have been recognized as causes of CIRD in dogs, and co-infections with multiple pathogens are commonly present in both cats and dogs. It is important not to overlook the possibility of coinfections, which may contribute to severe disease or result in a failure to respond as expected to therapy. Prevention is assisted by proper attention to hygiene and quarantine, minimizing overcrowding within kennels, multiple cat households and shelters, and use of vaccines. Because of growing concerns about antibiotic resistance, antibiotic treatment should be withheld unless dogs and cats are systemically unwell and show signs of mucopurulent nasal discharge, lethargy, or have evidence of secondary bacterial pneumonia. Dogs and cats with mild signs of respiratory disease typically recover without treatment over 1-4 weeks. Cough suppression may be indicated to help the dog (and the owner) sleep at night. If antibiotics are deemed indicated, doxycycline should be considered as a first-line treatment because of its activity against *Bordetella* and mycoplasmas.

AFAST® Introduction and Its Target Organ Approach – Everyday Extension of Your Physical Exam

Emerald Coast Veterinary Conference, Sandestin, FL, USA
Gregory R. Lisciandro, DVM, Dipl. ABVP, Dipl. ACVECC
Hill Country Veterinary Specialists & FASTVet.com, Spicewood, Texas USA
Email LearnGlobalFAST@gmail.com
Cell 210.260.5576
Website FASTVet.com
Textbook Point-of-care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2021

Introduction

The clinical utility of AFAST®, its target-organ approach and its applied fluid scoring system may be used in virtually all subsets of patients including trauma, triage (non-trauma) and tracking (monitoring) cases in the emergent and critical care settings. The previously published T^3 designation encompasses these 3 subsets, Trauma, Triage (non-trauma), and Tracking (monitoring) and avoids the onslaught of confusing acronyms and terms in human medicine and now veterinary medicine.

However, AFAST® has become a universal acronym and now used "an extension of the physical examination" and thus the T^3 designation is unnecessary as more and more veterinarians understand its daily applications to nearly all patients. Most importantly, AFAST® has *exact* clarity to its standardized 5-acoustic windows or views so colleagues know exactly what ultrasound study is being performed (Lisciandro et al. 2009; Lisciandro 2011, 2014, 2016, 2020; Boysen and Lisciandro 2013; McMurray et al. 2016).

The AFAST® examination carries greater potential to positively guide clinical course and improve patient outcome by detecting conditions and complications otherwise missed or delayed based on traditional first line evaluation of physical examination, laboratory testing, and radiographic finding. AFAST® findings are made more clinically relevant for the clinician, client, and referring veterinarian by using its standardized ultrasound format, and by recording AFAST® findings on standardized goal-directed templates for medical records (see below).

The mindset for those using AFAST® is one of a *ruling in* and *ruling out* test (highly specific and highly sensitive) for the presence or absence of free fluid, and a *ruling in* test for soft tissue abnormalities of its target-organ (highly specific and variably [user-dependent] sensitive). In other words, AFAST® serves as a screening test for obvious abnormalities of its target-organs. In other words, if you see an abnormality it's likely real; however, if you don't see an abnormality, then it may have been missed, being user-dependent keeping in mind our training is not the same as a radiologist or cardiologist. Thus, AFAST® is not intended to replace a complete detailed abdominal ultrasound.

AFAST® can answer many clinical questions within its 5-view framework. AFAST® has an applied abdominal fluid scoring system that helps semi-quantify effusions and help with decision-making regarding medical versus surgical cases including the need for blood transfusion(s), exploratory surgery, and other interventions in both bleeding and non-hemorrhaging patients. Moreover, the AFAST® Cysto-Colic View urinary bladder volume estimation formula provides a means to estimate urine volume and thus over time a means to non-invasively estimate urine output (Lisciandro and Fosgate 2017). Pneumoperitoneum, gastrointestinal peristalsis, renal perfusion, volume status and intrathoracic abnormalities are added AFAST® clinical information gained without any additional views.

The standardization and clarity of Global FAST®, the term used for combining AFAST®, TFAST® and Vet BLUE®, is the author's recommended approach for using FAST and point-of-care ultrasound (POCUS) because it avoids "selective imaging" and "satisfaction of search error." "Selective imaging" leads to "confirmation bias error", searching for evidence to fulfill the clinician's preconceived bias for the diagnosis. For example, you wouldn't only palpate the abdomen in a vomiting patient, thus you shouldn't selectively image.

"Satisfaction of search error" is common in radiology and occurs when the evaluator stops at the first abnormality carrying the potential to miss other important findings. Advantageously, the Global FAST® Approach provides exact clarity to an unbiased set of 15 data imaging points of the abdomen and thorax, including heart and lung (Lisciandro 2011, 2012, 2014, 2020); and should preempt all other POCUS examinations. *The bottom line, POCUS examinations should be considered as an add-on to Global FAST®, or the 2 approaches should be used together to avoid such errors.*

Finally, the Global FAST® Approach, better ensures that more traditional complete ultrasound studies are ordered for the *correct* cavity and that it is safe to restrain the patient especially for dorsal recumbency.
The Global FAST® Approach is our 3rd standardized veterinary ultrasound examination, unique because it screens both cavities, and should be a first line extension of the physical exam in most if not all patients (Lisciandro 2020).

Distinguishing Global FAST® from Flashing and POCUS
Global FAST®. Global FAST® is the combination of AFAST® and its Target-organ Approach and its Abdominal Fluid/Hemorrhage Scoring System and urinary bladder volume estimation formula, TFAST® for the detection of pleural and pericardial effusion, pneumothorax, and its 4 TFAST® echo views, and Vet BLUE®, the veterinary brief lung ultrasound exam, a regional, pattern-based approach with its B-line Scoring System, and its Visual Lung Language. Each of these 3 ultrasound formats has exact clarity to its respective acoustic windows (views) and findings (patient data) are recorded in goal-directed templates. Without this disciplined approach, accurate tracking patients and measuring your overall point-of-care ultrasound program quality is impossible. Moreover, the veterinary radiologist and cardiologist perform

their studies in the exact same manner every time for good reasons, to better know where to expect anatomy, and better recognize deviations from what is expected, and thus to minimize missing abnormalities. The Global FAST® sonographer's baseline skill set is to be able to recognize free fluid and merely deviations from the expected at its respective target-organs.

Flash exams. The "Flash Approach" is a term applied to a desultory sweep (no organized direction, no defined acoustic windows, no clarity) of the abdomen, thorax, and now lung answering a simple binary question of fluid positive or fluid negative within the abdomen and thorax; and the presence or absence of B-lines (also called lung rockets). The "Flash mentality" should be likened to performing an incomplete physical examination. For most veterinarians, we know the risk of missing important clinical information by doing so.

Point-of-care Ultrasound (POCUS). Point-of-care ultrasound (POCUS), which includes FAST (focused assessment with sonography for trauma, triage and tracking) examinations, is defined by the author as a goal-directed ultrasound examination(s) performed by a healthcare provider point-of-care (cageside) to answer a specific diagnostic question(s) or guide performance of an invasive procedure(s).

**The Global FAST® Approach is not a "Flash exam." AFAST®, TFAST®, Vet BLUE®, and Global FAST® should never be used interchangeably with the "Flash approach." These terms are erroneously and misleadingly used by our colleagues.*

**The Global FAST® Approach should be used as a baseline set of unbiased data imaging points surveying both cavities and then POCUS or Focused Exams as add-on evaluations to prevent "satisfaction of search error", "selective imaging" and "confirmation bias error"; and thus for increasing the probability of an accurate assessment through integration of global clinical findings.*

Patient Positioning, Preparation, Probe Type, Preset, Probe Maneuver

Positioning. Standing (sternal) and lateral recumbency are used. Right lateral recumbency is preferred over left lateral because of it is advantageous for echocardiography, electrocardiography, and imaging the caudal vena cava, however, the fluid scoring system is validated in *either* lateral positioning. Generally, if a patient is standing, AFAST® and Global FAST® are performed in standing. In AFAST®- negative for fluid standing (sternal) patients, lateral recumbency is unnecessary. If AFAST® is positive for free fluid, then follow the "AFAST® 3-minute fluid scoring rule" of moving to lateral recumbency and waiting 3-minutes to allow free fluid to redistribute for an accurate abdominal fluid score. Right lateral recumbency is generally only added to a standing AFAST®- Global FAST® when there is free fluid in the abdomen, TFAST® echo views and characterization of the caudal vena cava and its associated hepatic veins are unsatisfactory, or changes in positioning are warranted to better interrogate target-organs. *Dorsal recumbency is never used because it is too risky for hemodynamically fragile or unstable patients especially with intrathoracic problems including cardiac and pulmonary conditions and pleural space disease.*

Preparation. Fur is not shaved but rather parted with minimal amounts of isopropyl alcohol followed by alcohol-based hand sanitizer (HS) because HS couples as well as commercially available gel with the advantage of evaporating off the patient. Alcohol-based HS is also less noxious and less cooling than isopropyl alcohol; and less gooey (hand sanitizer evaporates) than

acoustic coupling gel. Isopropyl alcohol should not be used if electrical defibrillation is anticipated (fire/burn hazard).
**Make every attempt to part the fur and place the probe as directly as possible on skin to maximize the image quality and minimize "air-trapping" between the probe head and the skin.*
Probe Type. The microconvex (curvilinear) probe is used for the entire Global FAST®. A phased-array (sector) cardiac probe and linear probe may be used but each are unnecessary for most patients only adding more time to the study and are generally reserved for more complete detailed examinations.
Preset. The entire AFAST® (and Global FAST®) is performed with the abdominal preset. Preset may be changed, but by doing so, generally only adds time and changing presets may be reserved for more complete detailed examinations. Of note, cardiac presets reverse the orientation used for abdomen and lung, which becomes spatially challenging.
Probe Maneuver. The probe maneuvering is standardized. The probe is fanned, rocked cranially, and returned to the starting point at each AFAST® view. We premise this probe maneuvering on the original study that showed when comparing longitudinal to transverse views, they matched 397/400 times for the detection of free fluid. *All AFAST® views are imaged by fanning, rocking cranially, and returning to the starting point.*

The AFAST®

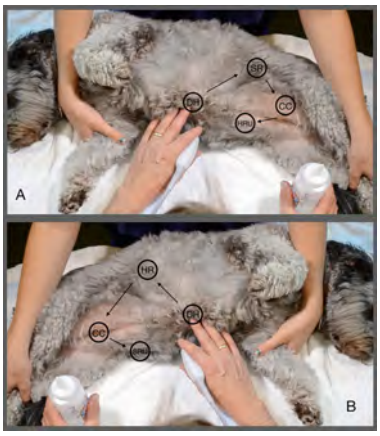

Figure. The AFAST® views used for abdominal fluid scoring are shown on a dog and analogous for cats (and non-human primates and exotic companion mammals). Note not shown is the Hepato-Renal 5th Bonus view when in right lateral and the Spleno-Renal 5th Bonus view when in left lateral recumbency. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

AFAST® Order. The AFAST® regardless of positioning (standing/sternal, right lateral recumbency) is always performed in the same order beginning at the Diaphragmatico-Hepatic (DH) view, followed by the least gravity dependent Spleno-Renal (SR) view, then the Cysto-Colic (CC) view, completing the AFAST® at the most gravity dependent Hepato-Renal Umbilical (HRU) view, where abdominocentesis is performed in most fluid-positive patients. The spleen is generally identified in this region (HRU) and then followed performing a Focused Spleen. In left lateral recumbency the order is analogous with the Hepato-Renal (HR) view replacing the

Spleno-Renal view and the Spleno-Renal Umbilical (SRU) view replacing the Hepato-Renal Umbilical (HRU) view. As with right lateral recumbency, a Focused Spleen is performed *immediately after the umbilical view* and completing the 4 views of the AFAST® fluid scoring system (DH, SR, CC, HRU).

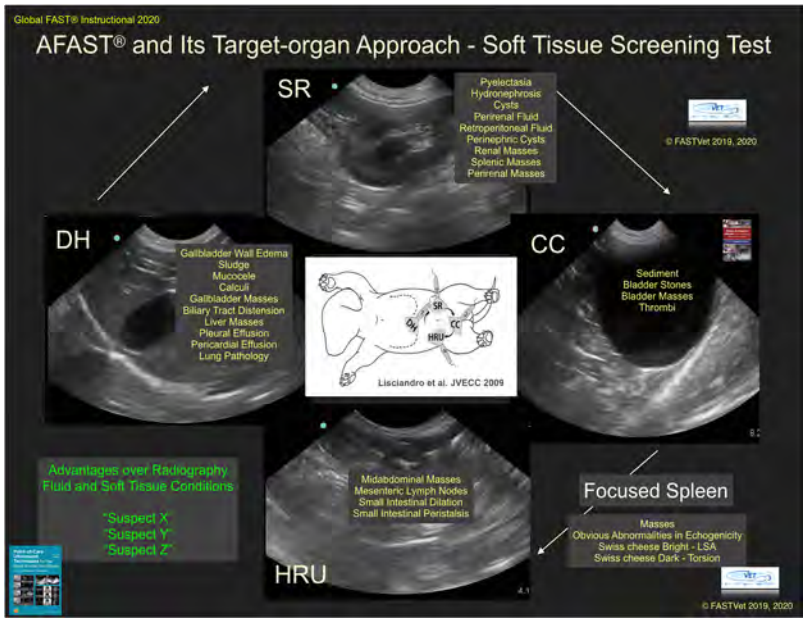

Figure. The AFAST® views used for abdominal fluid scoring are shown on a dog and analogous for cats (and non-human primates and exotic companion mammals) with actual ultrasound images along with obvious soft tissue abnormalities possible. Note not shown is the Hepato-Renal 5th Bonus view when in right lateral and the Spleno-Renal 5th Bonus view when in left lateral recumbency. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

Diaphragmatico-Hepatic (DH) view. Target-organs are liver, gallbladder and the heart, lung, and pleural cavity looking beyond (cranial to) the diaphragm and the caudal vena cava and its associated hepatic veins as it traverses the diaphragm. They are imaged in longitudinal planes with fanning, rocking cranially, and returning to your starting point.

Soft Tissue Abnormalities Screened for at the DH View:

- Gallbladder sediment and sludge
- Gallbladder mucoceles
- Choleliths
- Gallbladder wall edema
- Gallbladder wall masses
- Biliary tract distension
- Liver masses and cysts
- Liver and heterogenous echogenicity

- Dirofilariasis
- Pericardial effusion
- Pleural effusion
- Lung lesions along the pulmonary-diaphragmatic interface

- **Add-on:** Caudal vena cava characterization (and hepatic venous characterization)

Spleno-Renal (SR) view. Least gravity-dependent view. Target-organs are left kidney and spleen where the spleen is attached to the greater curvature of the stomach via the short gastric vessels. They are imaged in longitudinal planes with fanning, rocking cranially, and returning to your starting point. The stomach and colon are deep to the target-organs and often air-filled shadowing through the far field. This view would be used for the detection of pneumoperitoneum (air would rise).

Soft Tissue Abnormalities Screened for at the SR (and HR) View:

- Retroperitoneal effusion
- Pyelectasia
- Hydronephrosis
- Perirenal fluid
- Perinephric cysts
- Cortical cysts
- Cortical infarction
- Renal masses
- Splenic masses
- Spleen and heterogenous echogenicity
- Perirenal and retroperitoneal masses
- Ureteral distension

- **Add-on:** Pneumoperitoneum and renal perfusion

Cysto-Colic (CC) view. The target-organ is the urinary bladder with the acknowledgement of the colon that when air-filled obscures imaging. Probe (scanning plane) is directed into the view's most gravity-dependent "CC Pouch." They are imaged in longitudinal planes with fanning, rocking cranially, and returning to your starting point. The thigh is often seen through the far field. In predominately intact species, such as non-human primates and exotic companion mammals, the sex organs, especially the uterus, should be part of this view.

Soft Tissue Abnormalities Screened for at the CC View:

- Urinary bladder sediment
- Urinary bladder calculi
- Urinary bladder wall masses
- Urinary bladder thrombi
- Urinary bladder wall irregularities

- Pregnancy
- Uterine abnormalities

Hepato-Renal Umbilical (HRU) view. Most-gravity dependent. Misnomer. The view previously designated Hepato-Renal (HR) view is now considered the "Hepato-Renal Umbilical (HRU) view." Target-organs are really the spleen and intestine. Neither the right kidney nor the right liver is imaged. The probe is placed at the level of the umbilicus and imaged its scanning plane into the most gravity-dependent "HR Umbilical Pouch." In standing or sternal, the probe as placed on the umbilicus. Fanning, rocking cranially, and returning to the starting point is the same probe maneuver at all AFAST® views. In predominately intact species, such as non-human primates and exotic companion mammals, the sex organs, especially the uterus, should be part of this view. This view really should be renamed as the "Spleno-Intestino Umbilical view" (and likely will be).

Soft Tissue Abnormalities Screened for at the HR-Umbilical View:

- Heterogenous echogenicity of the splenic parenchyma
    - Bright Swiss Cheese R/O lymphoma
    - Dark Swiss Cheese R/O splenic torsion
- Splenomegaly
- Myelolipomas
- Splenic masses
- Mid abdominal masses
- Abnormalities of the splenic hilar vessels
- Small intestinal peristalsis
- Pregnancy
- Uterine abnormalities

Hepato-Renal 5th Bonus View: Not part of the abdominal fluid scoring system. Most often performed standing immediately as the final view of Global FAST®. Target-organs are right kidney and adjacent right liver. They are imaged in longitudinal planes with fanning, rocking cranially, and returning to your starting point.

Soft Tissue Abnormalities Screened for at the HR5th Bonus (SR5th Bonus) View:

- Retroperitoneal effusion
- Pyelectasia
- Hydronephrosis
- Perirenal fluid
- Perinephric cysts
- Cortical cysts
- Cortical infarction
- Renal masses
- Liver masses
- Liver and heterogenous echogenicity

- Perirenal and retroperitoneal masses
- Ureteral distension

- **Add-on:** Portal vein interrogation, pancreas, duodenum

GOAL-DIRECTED TEMPLATE FOR AFAST®

Patient positioning: right or left lateral recumbency or standing or sternal
Gallbladder: present or absent, contour, wall, content, unremarkable or abnormal
Urinary bladder: present or absent, contour, wall, content, unremarkable or abnormal

Positive of negative at the 4-views (0 negative, 1 positive)

Diaphragmatico-Hepatic (DH) site:	0 or 1/2 or 1
Spleno-Renal (SR) site:	0 or 1/2 or 1
Cysto-Colic (CC) site:	0 or 1/2 or 1
Hepato-Renal Umbilical (HRU) site:	0 or 1/2 or 1

Total Abdominal Fluid Score (0-4): _____

HR5th Bonus View: 0 or 1/2 or 1 **or Indeterminate or Not Assessed (NA)**
Focused Spleen (*add-on after completing the* AFAST® HR Umbilical View):
_____

DH View:
Pleural effusion: absent, present (mild, moderate, severe) or indeterminate or NA
Pericardial effusion: absent, present (mild, moderate, severe) or indeterminate or NA
[$]**Hepatic venous distension:** unremarkable or present (Tree Trunk Sign) or indeterminate or NA
[&]**Caudal vena cava characterization:** bounce (unremarkable) or FAT or flat or indeterminate or NA
[#]**Vet BLUE:** B-lines: 0, 1, 2, 3, >3, or ∞ and if Shred__cm, Tissue__cm, Nodule__cm, Wedge__cm

Comments:_____
_____
Note: The AFAST® is a rapid ultrasound examination used to detect the presence of free abdominal fluid and obvious soft tissue abnormalities as a screening test in order to better direct resuscitation efforts and diagnostics, detect complications, and manage patients. AFAST® is not intended to replace a complete detailed abdominal ultrasound exam.

[$]**The hepatic veins** should *not* be apparent in both dogs and cats placed in lateral recumbency. When imaged the branching has been referred to by the author as the "Tree Trunk Sign."
[&]**The caudal vena cava** can be alternatively referred to as a bounce = fluid responsive cava (~35-50% diameter change); FAT = fluid intolerant cava (distended with maximum height > 1 cm

in dogs < 9kg and > 1.5 cm in dogs > 9kg with little height change [< 10%]); flat = hypovolemic cava (small with maximum height < 3 mm in dogs < 9 kg, < 5 mm in dogs > 9 kg with little height change [< 10%]).
[#]**Vet BLUE** screens for lung abnormalities along the Pulmonary-Diaphragmatic Interface.

Template provided by Dr. Gregory Lisciandro, DVM, DABVP, DACVECC, FASTVet.com and Hill Country Veterinary Specialists Copyright 2018, 2019, 2020 for your use and modification.

Other current Goal-directed Template AFAST®, TFAST®, Vet BLUE® and Global FAST® versions may be found at FASTVet.com under the Premium Membership, then Resource Library, and then Free Resources.

AFAST® Add-on Information
AFAST® can answer many clinical questions within its 5-view framework. Add-on skills include the following:

- The AFAST® Cysto-Colic View urinary bladder volume estimation formula (LxHxWx0.625) to estimate urine volume and thus over time non-invasively estimate urine output (Lisciandro and Fosgate 2017)
- Pneumoperitoneum (Enhanced Peritoneal Stripe Sign)
- Gastrointestinal peristalsis
- Renal perfusion
- Volume status via characterization of the caudal vena cava and hepatic veins – *See Global FAST® Proceedings*
- Intrathoracic abnormalities especially pleural and pericardial effusion and lung abnormalities along the pulmonary-diaphragmatic interface - *See TFAST® Proceedings*

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AFAST®and Its Abdominal Fluid Scoring System for Bleeding Patients – Everyday Extension of Your Physical Exam

Emerald Coast Veterinary Conference, Sandestin, FL, USA
Gregory R. Lisciandro, DVM, Dipl. ABVP, Dipl. ACVECC
Hill Country Veterinary Specialists & FASTVet.com, Spicewood, Texas USA
Email LearnGlobalFAST@gmail.com
Cell 210.260.5576
Website FASTVet.com
Textbook Point-of-care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2021

The AFAST®

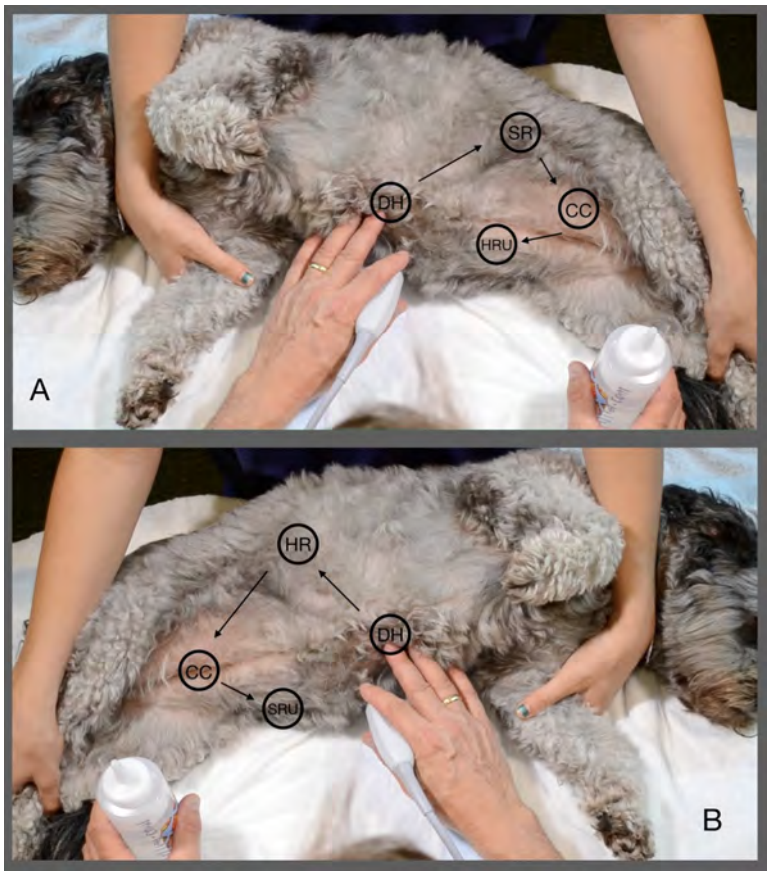
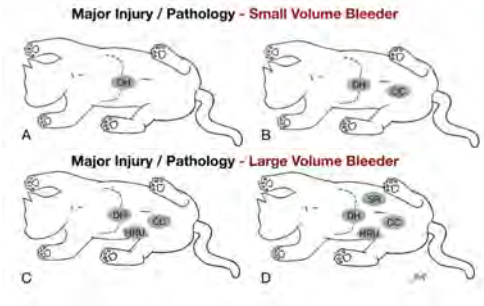


Figure. The AFAST® views used for abdominal fluid scoring are shown on a dog and analogous for cats (and non-human primates and exotic companion mammals). Note *not shown* is the Hepato-Renal 5th Bonus view when in right lateral and the Spleno-Renal 5th Bonus view when in left lateral recumbency. *This material is reproduced with permission of John Wiley & Sons, Inc, Focused Ultrasound Techniques for the Small Animal Practitioner, Wiley ©2014 and Gregory Lisciandro, DVM, FASTVet.com ©2020.*

Patient Positioning, Preparation, Probe Type, Preset, Probe Maneuver
Positioning. *See Proceedings entitled AFAST® Introduction and Its Target Organ Approach – Everyday Extension of Your Physical Exam*

AFAST® Order and Views. *See Proceedings entitled AFAST® Introduction and Its Target Organ Approach – Everyday Extension of Your Physical Exam*

AFAST®-Applied Fluid Scoring System



The AFAST®-applied fluid scoring system is defined as follows (4-point scale): abdominal fluid score (AFS) of 0 (AFS 0) means negative at all 4 views to a maximum score of AFS 4 means positive at all 4 views.

*Low-scoring AFS1 and 2 (<3) are considered major injury/pathology, small volume bleeders.
*High-scoring AFS 3 and 4 (≥3) are considered major injury/pathology, large volume bleeders.
Modified from Lisciandro, et al. JVECC 2009; 19(5): 426-437, JVECC 2011;20(2); 104-122. *Gregory Lisciandro, FASTVet.com, Wiley ©2020.*

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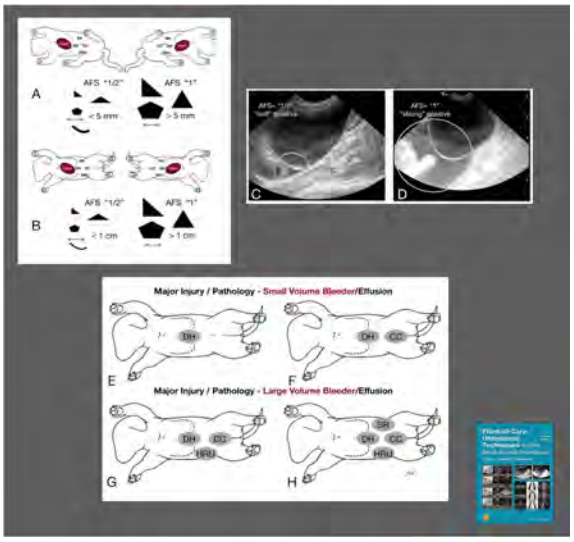
The AFAST®-applied is hugely impactful and should be assigned and recorded in every patient. It's a simple 0-4 scoring system and has significant advantages over subjective terms of trivial, mild, moderate and severe as well as designating each respective positive and negative AFAST® view. Recording positive and negative views may help with origin of bleeding or effusion (peritonitis) in lower-scoring patients. For example, in a bleeding trauma patient that has an AFS of 1 and positive at the DH view, that over time becomes a large volume bleeder with an AFS ≥ 3, logic would dictate the source of bleeding is likely the liver and/or its associated vasculature.

Small versus Large Volume Bleeder/Effusion. The abdominal fluid score (AFS) helps rapidly categorize the patient as a small volume (AFS 1 and 2, or < 3) versus large volume bleeder (AFS 3 and 4, or ≥ 3). AFS 1 and 2 (< 3) do not have enough blood intra-abdominal to directly result in anemia. Thus, if an AFS 1 or 2 is anemic, then there are the following 4 major rule outs in the acute setting: 1) preexisting anemia, 2) bleeding somewhere else - always do Global FAST® and a good physical exam, 3) hemodilution (less common with graduated fluid therapy strategies), or 4) lab error. The AFS allows tracking of worsening (increasing AFS), resolving (decreasing AFS), or static (no change in AFS). Patients also become volume depleted from non-hemorrhagic effusions and thus small versus large volume effusion principle works for anticipating hypovolemia from fluid loss (without the need for hemoglobin).

Modification of the Abdominal Fluid Scoring System - 0 or 1/2 or 1

Scoring as 0, ½, or 1. The author for several years has been categorizing positives as “weak” if the maximum pocket is <1 cm (<5 mm in cats) scoring as a "1/2" versus a “strong” positive if >1 cm (>5 mm in cats) making the score a full "1." The small vs. large volume bleeding concept remains as AFS 1 and 2 small volume (< 3), and AFS 3 and 4 (≥ 3), large volume bleeders. Clinical judgment always should be considered; however, this “weak” versus “strong” positive modification provides an option to better assess and semi-quantitate volume in bleeding patients and those with other forms of ascites and peritonitis. The modification of our original scoring system is based on a recently accepted study and an ongoing project (Lisciandro et al. 2020).



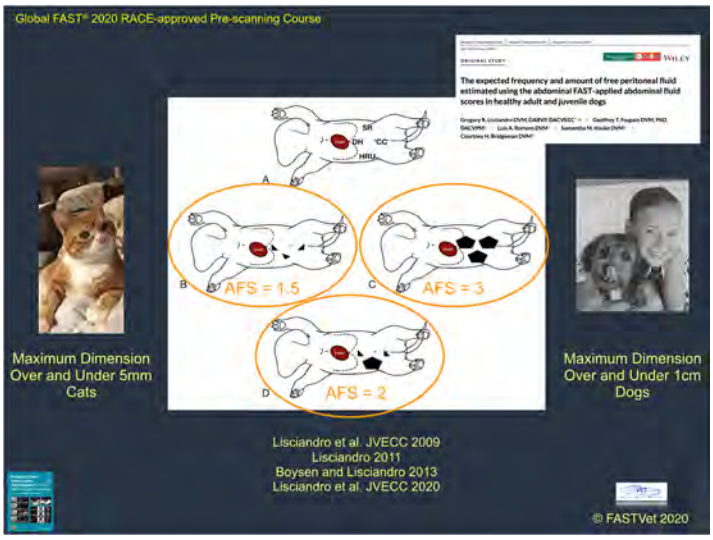


Figure on Left. A cartoon of a cat and dog in lateral recumbency showing the modification of the abdominal fluid score (AFS) to better differentiate between small volume versus large volume bleeding/effusion by assigning a score of "1/2" or "1" for “weak” versus “strong” positive views, respectively.

Figure on Right. For example, a dog may have small pockets at the DH, CC, and HRU views of <1 cm, that would now be considered more accurately a small volume bleeder/effusion with a score of ½ + ½ + ½ = "1 1/2" in B) rather than a 1 + 1+ 1 = "3" in C). Note in D) the score is ½ + ½ + 1 for a total of “2.” This approach may also be translated to non-human primates and exotic companion mammals. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

Use of Serial AFAST® and Determining the AFS

The use of serial AFAST® and serial application of its abdominal fluid scoring system is imperative to maximize information. The serial exam not only improves the sensitivity of the exam (true negatives) but also searches a second time for the developing presence of fluid, tracking the abdominal fluid score (0-4), and evaluating the presence or absence of the urinary bladder, and measuring the urinary bladder dimensions for estimating its volume. Always perform one more 4-hour post-admission serial AFAST® (better Global FAST®) in all stable patients and sooner if the patient is unstable or of questionable status. Then continue serial AFAST® (better Global FAST®) as needed and as part of patient rounds and recheck exams.

Hemoabdomen

***Trauma**. Use small versus large volume bleeder principle for anticipating degree of anemia from the intra-abdominal hemorrhage. Most dogs and cats are treated medically through the use of titrated fluid therapy, blood transfusions, and correction of coagulopathy when present with surgery uncommonly needed.

***Non-trauma.** Use small volume versus large volume bleeder principle. Transfusion needs may be anticipated for a component of supportive care (and correcting coagulopathy when present) in coordination with surgical treatment and other possible interventions for stopping bleeding masses. Canine anaphylactic hemoabdomen is a species-unique, medically treated (not surgical) complication of dogs; and its coagulopathy treated when present. See Canine Anaphylactic Webinar off our website FASTVet.com and our most updated Canine Anaphylactic Proceedings.

***Post-interventional.** Use small volume versus large volume bleeder principle. As a general rule, large volume bleeders should be surgically treated when, if present, coagulopathy is first corrected and coagulopathy not the direct cause for the bleeding. Blood may be harvested from clean cavities in both dogs and cats and re-administered to the patient without anticoagulant but with a mandatory blood filter to catch clots from entering the patient's circulation.

AFAST®-applied Abdominal Fluid Scoring for Non-hemorrhagic Ascites

The small volume versus large volume effusion principle serves a similar role for predicting degree of hypovolemia keeping in mind that for example patients can become markedly volume contracted from a septic peritonitis. The difference in non-hemorrhagic effusions is that volume needs to be restored but without the need for hemoglobin unless the patient is anemic for another reason.

Expectations for Resolution of Hemoabdomen and Lavage Fluid

FASTVet 48-hour Rule. Expect cavitary bleeding to be resolved or nearly resolved within 48-hours with near negative abdominal fluid scores of AFS ≤ 1 (resorption of blood by the patient) once bleeding has stopped and coagulopathy, when present, is corrected. When positive fluid scores persist, especially large volume bleeders, then the cause must be investigated further because a major problem, i.e. active bleeding or coagulopathy, is present until proven otherwise. Of note, post-interventional cases should have their abdominal cavity free of fluid at surgical closure when possible, so that positive post-interventional fluid scores may be better interpreted. From author experience and most interestingly, lavage fluid lasts much longer than 48-hours in contrast to blood, and also inhibits neutrophil function in fighting peritonitis. *Thus, lavage fluid should always be as completely removed as possible before surgical closure because it persists for several days unlike blood.*

Radiographic Serosal Detail is Unreliable

Radiographic serosal detail been shown to be unreliable for not only the detection of ascites but also its amount in both human and veterinary imaging. In our original study, we found that 24% of dogs with normal radiographic serosal detail were in fact positive for free intra-abdominal fluid (abdominal fluid score, AFS, 1-4), and 33% with decreased serosal detail were in fact negative for free intra-abdominal fluid (AFS 0). The use of the AFAST®- abdominal fluid scoring system is evidence-based by "seeing" and scoring its volume comparable to the gold standard test of computed tomography.

Clinical Indications for AFAST®

The use of AFAST® should be simply stated as an "extension of the physical exam" in other words everyday applications for nearly every patient. *Global FAST® should be your first line "free fluid and soft tissue screening test" because it exceeds the yield radiographically in the great majority of our patients; and be as part of a work-up as blood and urine testing.* Think about long list of effusive and soft tissue conditions missed or only suspected by radiography that are detected and evidence-based using the AFAST® target-organ approach. *See Proceedings entitled AFAST® Introduction and Its Target Organ Approach – Everyday Extension of Your Physical Exam*

In summary, AFAST® is an" extension of the physical exam" and used for triaged trauma, non-trauma and post-interventional cases, your pre-anesthetic test, your semi-annual and annual checkup, your geriatric screening test, part of patient rounds and recheck exams, and for surveying patients with shock and part of basic and advanced life support in cardiopulmonary resuscitation.

GOAL-DIRECTED TEMPLATE FOR AFAST®

Patient positioning: right or left lateral recumbency or standing or sternal
Gallbladder: present or absent, contour, wall, content, unremarkable or abnormal
Urinary bladder: present or absent, contour, wall, content, unremarkable or abnormal

Positive of negative at the 4-views (0 negative, 1 positive)

Diaphragmatico-Hepatic (DH) site:	0 or 1/2 or 1
Spleno-Renal (SR) site:	0 or 1/2 or 1
Cysto-Colic (CC) site:	0 or 1/2 or 1
Hepato-Renal Umbilical (HRU) site:	0 or 1/2 or 1

Total Abdominal Fluid Score (0-4): _____

HR5th Bonus View: 0 or 1/2 or 1 **or Indeterminate or Not Assessed (NA)**
Focused Spleen (*add-on after completing the* AFAST® HR Umbilical View): **_____**

DH View:
Pleural effusion: absent, present (mild, moderate, severe) or indeterminate or NA
Pericardial effusion: absent, present (mild, moderate, severe) or indeterminate or NA
[$]**Hepatic venous distension:** unremarkable or present (Tree Trunk Sign) or indeterminate or NA
[&]**Caudal vena cava characterization:** bounce (unremarkable) or FAT or flat or indeterminate or NA
[#]**Vet BLUE®:** B-lines: 0, 1, 2, 3, >3, or ∞ and if Shred__cm, Tissue__cm, Nodule__cm, Wedge__cm

Comments:_____
Note: The AFAST® is a rapid ultrasound examination used to detect the presence of free abdominal fluid and obvious soft tissue abnormalities as a screening test in order to better direct resuscitation efforts and diagnostics, detect complications, and manage patients. AFAST® is not intended to replace a complete detailed abdominal ultrasound exam.

[$]**The hepatic veins** should *not* be apparent in both dogs and cats placed in lateral recumbency. When imaged the branching has been referred to by the author as the "Tree Trunk Sign."
[&]**The caudal vena cava** can be alternatively referred to as a bounce = fluid responsive cava (~35-50% diameter change); FAT = fluid intolerant cava (distended with maximum height > 1 cm in dogs < 9kg and > 1.5 cm in dogs > 9kg with little height change [< 10%]); flat = hypovolemic cava (small with maximum height < 3 mm in dogs < 9 kg, < 5 mm in dogs > 9 kg with little height change [< 10%]).
[#]**Vet BLUE®** screens for lung abnormalities along the Pulmonary-Diaphragmatic Interface.

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TFAST® for the Accurate Diagnosis of Pleural Pericardial Effusion – Everyday Extension of Your Physical Exam

Emerald Coast Veterinary Conference, Sandestin, FL, USA
Gregory R. Lisciandro, DVM, Dipl. ABVP, Dipl. ACVECC
Hill Country Veterinary Specialists & FASTVet.com, Spicewood, Texas USA
Email LearnGlobalFAST@gmail.com
Cell 210.260.5576
Website FASTVet.com
Textbook Point-of-care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley

Introduction

The clinical utility of TFAST®, its use for the rapid detection of pneumothorax, pleural and pericardial effusion, and its target-organ approach for the heart, will be reviewed. The previously published T^3 designation was meant to include Trauma, Triage (non-trauma), and Tracking (monitoring) to avoid the confusing acronyms in human and now veterinary medicine, in which similar FAST ultrasound examinations are given different acronyms and names for different subsets of patients. However, TFAST®, as with Global FAST®, is best now considered as "an extension of the physical examination" and the T^3 designation is unnecessary as more and more veterinarians understand its widespread applications. Moreover, TFAST® is standardized and has *exact* clarity to its 5-acoustic windows or views. The TFAST® carries greater potential to positively guide clinical course and improve patient outcome by detecting conditions and complications otherwise missed or delayed based on traditional first line evaluations of physical examination, laboratory testing, and radiographic finding. TFAST® findings are made more clinically relevant for the clinician, client, and referring veterinarian by using its standardized format, and by recording TFAST® findings on standardized goal-directed templates for medical records (see below). The mindset for those using TFAST® is one of a *ruling in* and *ruling out* test (highly specific and highly sensitive) for pleural or pericardial effusion, and for pneumothorax. TFAST® also serves as a *ruling in* test for soft tissue abnormalities of its target-organ, the heart (specific but variably sensitive [user dependent]). Meaning, if you see an abnormality, it's likely real, however, if you don't see an abnormality, then it may have been missed since we are not trained like a cardiologist or radiologist. Importantly, the TFAST® does not replace complete detailed echocardiography.

The standardization and clarity of Global FAST®, the term used for combining AFAST®, TFAST® and Vet BLUE®, is the author's recommended approach for using point-of-care ultrasound (POCUS) because it avoids "selective imaging" and "satisfaction of search error." "Selective imaging" leads to "confirmation bias error", searching for evidence to fulfill the clinician's preconceived bias for the diagnosis. "Satisfaction of search error" is a common error in radiology and occurs when the evaluator stops the exam at the first abnormality carrying the potential to miss other important findings. Advantageously, the Global FAST® Approach provides exact clarity to an unbiased set of 15 data imaging points of the abdomen and thorax, including heart and lung; and should preempt all other POCUS examinations. *The bottom line, POCUS examinations should be considered as an add-on to Global FAST®, or the 2 approaches should be used together to avoid such imaging errors.* The Global FAST® Approach is our 3rd standardized veterinary ultrasound examination, unique in that it screens both cavities, in addition to complete detailed abdominal ultrasound and complete echocardiography. Global FAST® including TFAST® should be used as a first line "extension of the physical exam" in most if not all patients.

Distinguishing Global FAST® from Flashing and POCUS

Flash exams. The "Flash Approach" is a term applied to a desultory sweep (no organized direction, no defined acoustic windows, no clarity) of the abdomen, thorax, and now lung answering a simple binary question of fluid positive or fluid negative within the abdomen and thorax; and the presence or absence of B-lines (also called lung rockets). The "Flash mentality" should be likened to performing an incomplete physical examination and for most veterinarians we know the risk of missing important clinical information by doing so. *AFAST®, TFAST®, Vet BLUE®, and Global FAST® should never be used interchangeably with the "Flash approach." These terms have been and continue to be erroneously and misleadingly used by some of our colleagues.*

Point-of-care Ultrasound (POCUS). Point-of-care ultrasound (POCUS), which includes FAST (focused assessment with sonography for trauma, triage and tracking) examinations, is defined by the author as a goal-directed ultrasound examination(s) performed by a healthcare provider point-of-care (cageside) to answer a specific diagnostic question(s) or guide performance of an invasive procedure(s). *The Global FAST® Approach should be used as a baseline set of unbiased data imaging points surveying both cavities and then POCUS or Focused Exams as add-on evaluations to prevent "selective imaging" and "confirmation bias error", "satisfaction of search error", and for increasing the probability of an accurate assessment through integration of clinical findings.*

Patient Positioning, Preparation, Probe Type, Preset, Probe Maneuver

Positioning. Standing (sternal) and lateral recumbency are used. Standing and sternal are generally safer for respiratory compromised or distressed patients. However, patients that are stable and confortable in lateral recumbency may be evaluated in that position as well. Right lateral recumbency is preferred over left lateral because of it is advantageous for echocardiography, electrocardiography, and imaging the caudal vena cava, however, the AFAST® abdominal fluid scoring system is validated in either lateral positioning. Generally, if a patient is standing, TFAST® and Vet BLUE® are first performed from the left side followed by AFAST® and a Focused Spleen after which a right Vet BLUE®, TFAST® echo views and the HR5th Bonus view of AFAST® are performed on the patient's right side. The order is referred to as the Global FAST® blend and is low impact for the patient requiring minimal restraint and patient risk if hemodynamically fragile. Regarding AFAST®, if the patient is AFAST®-negative in standing (or sternal), then lateral recumbency is unnecessary. If the patient is AFAST®-positive, then follow the "AFAST® 3-minute fluid scoring rule" of moving to lateral recumbency when safe, and waiting 3-minutes to allow free fluid to redistribute for an accurate abdominal fluid score. Right lateral recumbency is generally only added to a standing AFAST®- Global FAST® when TFAST® echo views and characterization of the caudal vena cava and its associated hepatic veins are unsatisfactory, or changes in positioning are warranted to better interrogate target-organs. *Dorsal recumbency is never used because it is too risky for hemodynamically fragile or unstable patients especially with intrathoracic problems including cardiac and pulmonary conditions and pleural space disease.*

Preparation. Fur is not shaved but rather parted with minimal amounts of isopropyl alcohol followed by alcohol-based hand sanitizer because it couples as well as commercially available gel with the advantage of evaporating off the patient. *Make every attempt to part the fur and place the probe as directly as possible on skin to maximize the image quality and minimize "air-trapping" between the probe head and the skin.*

Probe Type. The microconvex (curvilinear) probe is used for the entire Global FAST®. A phased-array (sector) cardiac probe and linear probe may be used but each are unnecessary, only adding more time to the examination, and are generally reserved for more complete detailed examinations.

Preset. The TFAST® and entire Global FAST® are performed with the abdominal preset. Changing the preset is unnecessary and only adds time, reserved for more complete detailed examinations.

Probe Maneuver. The probe maneuvering is standardized for lung and cardiac views. Lung begins with the Gator Sign orientation and maintaining longitudinal planes. The left Pericardial Site View has the "TFAST® slide" cranial and caudal to the heart into pouches named the cardiac-diaphragmatic and cardiac-cervical pouch, respectively.

The TFAST®

Strengths and Weaknesses of the TFAST® Views

There are 5 acoustic windows for TFAST®. These are the bilaterally applied Chest Tube Site and Pericardial Site views, and the singly applied Diaphragmatico-Hepatic view. As an aside, the CTS site view followed by Vet BLUE® are performed first, followed by the Pericardial Site views on both the left and right side.

Pneumothorax - Chest Tube Site (CTS) views. The bilaterally applied CTS view is best used to rule out pneumothorax (PTX) and survey for lung pathology (see Vet BLUE Proceedings) on both the left and right sides. The CTS view is along the highest accessible locations on the thoracic wall where the free air within the pleural cavity would rise to in the presence of PTX. Thus, if lung is observed in direct opposition to the thoracic wall at the CTS view, most commonly by "lung sliding" or B-lines, then PTX is ruled out. When PTX is suspected, then search for the "Lung Point" to determine the degree of PTX (see below).

Pleural and Pericardial Effusion, Echo Views - Pericardial Site (PCS) views. The bilaterally-applied PeriCardial Site (PCS) views on both the left and right sides are used to screen for the presence of pleural and pericardial effusion; and the right side for TFAST® echocardiography views including for volume status and contractility assessment via the left ventricular short-axis "mushroom" view (LVSA), for the "quick peek" short-axis left atrial to aortic ratio (LA:Ao) to screen for left-sided cardiac problems (increased left atrial filling pressure); and for the long-axis 4-chamber view to screen for right-sided conditions (RV:LV) (increased right ventricular filling pressures); and the long-axis 4-chamber view with the left ventricular outflow tract (LVOT) for abnormalities within the LVOT and aorta. The use of the PA:Ao (PA, pulmonary artery, Ao, aorta) ratio may also be learned as an add-on skill because the PA:Ao is accessible at the short-axis LA:Ao view and the long-axis 4-chamber view.

The TFAST® Echo Views and The Global FAST® Fallback View Strategies

"Global FAST® Non-echo Fallback Views" are hugely impactful for 2 major reasons. **First,** when performing the TFAST® echo views, suspect problems may be double-checked with its fallback views. For example, the sonographer thinks that the LA:Ao is increased, then uses Vet BLUE® and finds that the lung is dry (absent B-lines),and therefore no evidence of any clinically-relevant left-sided congestive heart failure. The patient may have left-sided heart disease, but has no evidence of left-sided *congestive* heart failure, important clinical information. Conversely, if Vet BLUE® shows wet lung, and its regional, pattern-based approach supports left-sided *congestive* heart failure (versus pneumonia), then there is an urgency to continue the work-up and treat the patient. The same logic holds for an increased RV:LV, however, the caudal vena cava and its associated hepatic veins are used for assessment, because right-sided congestive heart failure results in hepatic venous congestion and thus at the DH view, a distend caudal vena cava and its associated hepatic veins (Tree Trunk Sign). As for poor volume, the caudal vena cava is also assessed. A flat (small maximum height) cava supports more severe depletion than a caudal vena cava with a bounce. And, Vet BLUE® and the DH View should always be used in tandem to assess for left-and right-sided congestive heart failure to better assess and treat and monitor the patient. As for poor contractility, Vet BLUE® and the DH view are used to screen for concurrent left- and right-sided failure. **Second,** the "Global FAST® Non-echo Fallback Views" are used when it's too risky for TFAST® echo views because of patient status, or because the patient is difficult to image. Dry lung, or absent B-lines on Vet BLUE®, rules out left-sided *congestive* heart failure; and a "bounce" to the caudal vena cava along with an absence of

hepatic venous distension (no Tree Trunk Sign), rules out right-sided *congestive* heart failure. Typically, these "Global FAST® Non-echo Fallback Views" are easier to acquire in critical patients than the TFAST® echo views; and TFAST® echo views or complete echocardiography can wait until the patient is more stable. *See Global FAST® Proceedings for greater detail.*

The TFAST® Diagnosis of Pericardial versus Pleural Effusion

When performing the TFAST® left and right PeriCardial Site (PCS) Views make it a habit to have enough depth to see the heart globally or in other words in its entirety. Your landmark is the hyperechoic (bright white) pericardium in the far-field. The sonographer should be aware that too shallow of depth easily leads to the possibility of mistaking heart chambers for pleural and/or pericardial effusion especially in distressed patients that provide only quick glimpses of the heart (short-lived acoustic windows) due to air interference from lung. The concept is illustrated in the images below.

New: The TFAST® Slide for Pleural Effusion. The probe is slid caudal first and then cranial to the heart into the gravity dependent regions called the “cardiac diaphragmatic pouch” and the “cardiac cervical pouch” with “pouch” inferring the most gravity dependent region away from the heart where fluid would pocket. The detection of pleural effusion is confirmed by the observation of the “curtain sign” of pleural effusion.

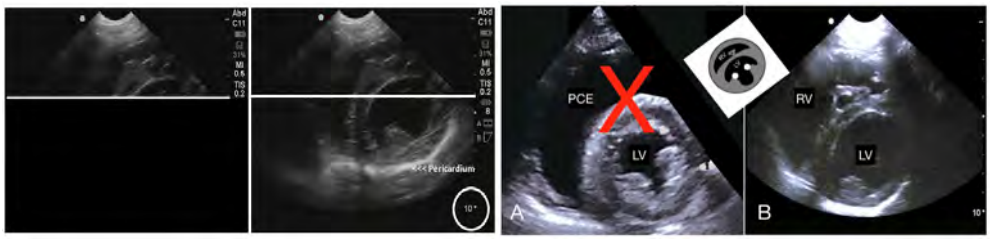

Figure on the Left. Make Sure You Image the Heart in Its Entirety. Shows how having *too shallow depth* can lead to serious mistakes. To the left, the image shows how it is difficult to accurately distinguish pleural or pericardial effusion from the crescent-shaped right ventricle, that, in haste, can be easily mistaken for pleural or pericardial effusion and its papillary muscles for pathology (see the next figure). Insist as best practice to always image the heart *in its entirety* using the hyperechoic (bright white) line of the pericardium in the far-field as your habitual landmark. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com ©2014, 2020.*

Figure on the Right. The Right Ventricle Mimicks Pericardial Effusion - The Danger of this View. The figure shows how the short-axis “mushroom” and its other short-axis views are dangerous as a single view for the non-cardiologist sonographer for the following reason: the image to the left shows pericardial effusion labeled as "PCE" compared to the image to the right that shows how the normal cardiac anatomy of the crescent-shaped right ventricle (RV) can mimic pericaridal (or pleural effusion). This mistake, common enough, leads to the most potentially catastrophic of interventions of performing centesis on a heart chamber (Lisciandro JVECC 2016). Best practice is *not* use the right Pericardial (parasternal) left ventricular short-axis views for pleural and pericardial effusion *unless combined with* other views because the mistake is easy to make without this TFAST® mindset. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com ©2014, 2020.*

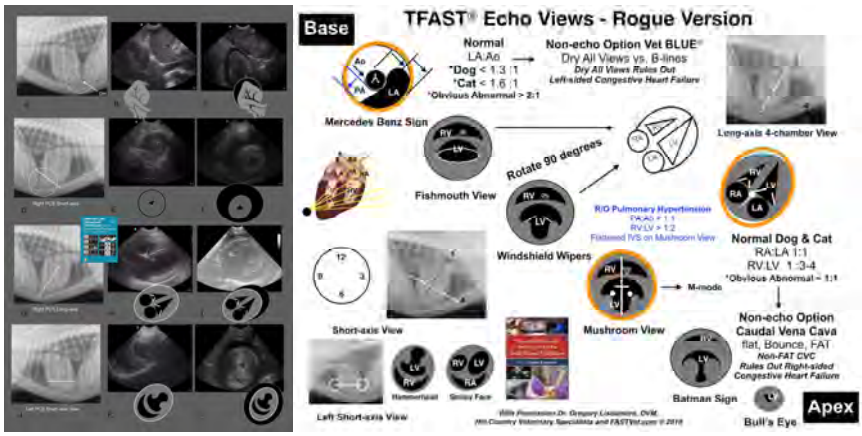

Figure. Pericardial Effusion Composite and TFAST® Echo Chart. In the first column are didactic radiographic images showing the scanning planes for each respective row of images of which there is normalcy to the right of each radiograph followed by pericardial effusion for that respective view. Note that the "Hammerhead View" from the left TFAST® PCS view is also an acceptable view (see TFAST® Echo Chart), because there are only 2 heart chambers located there, the left and right ventricles, and both ventricles may be clearly identified with fluid outside of them. Thus, it is difficult on both the long-axis 4-chamber view (G,H,I) and "Hammerhead View" (J,K,L) to mistake a heart chamber for pericardial (or pleural) effusion. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com ©2014, 2020.*

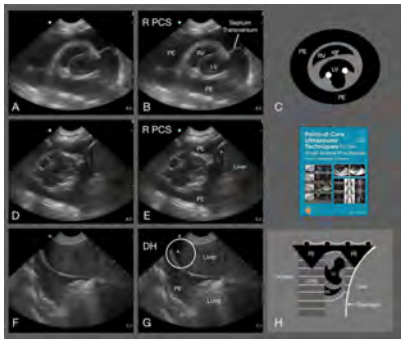

Figure. Pleural Effusion Composite. Shown are examples for pleural effusion from the TFAST® PCS views and the TFAST®-AFAST® DH view. Note how triangulations similar to ascites exist for pleural effusion, unlike pericardial effusion, that is rounded being contained within the pericardial sac. Another manner in which to diagnose pleural effusion is by default, in other words, it is *not* pericardial effusion, which is much easier to identify, and thus is pleural effusion. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com ©2014, 2020.*

TFAST® Diagnosis of Pericardial Effusion The Gold Standard for the Diagnosis of Pericardial Effusion is Ultrasound - Radiography is Unreliable Pericardial Effusion is Contained in the Pericardial Sac that Attaches at One Atrium and Rounds the Muscular Apex of the Heart to Attach to the Other Atrium		
Imaging Strategy	**TFAST® DH View**	**TFAST® PCS View**
*Image toward the muscular apex of the heart where no heart chambers can be mistaken for free fluid	*TFAST® DH View – Racetrack Sign	*TFAST® Right PCS View – Bull's Eye Sign
*Long-axis 4-chamber view where all 4 chambers are identified		*TFAST® Right PCS View
*Image the heart globally in its entirety using the bright white pericardium in the far-field as a landmark	Make Habitual Best Practice for Echo Views	Make Habitual Best Practice for Echo Views
TFAST® Diagnosis of Pleural Effusion The Gold Standard for the Diagnosis of Pleural Effusion is Debatably Computerized Tomography Radiography is Generally Good Pleural Effusion is Uncontained and Unrestrained Unless Compartmentalized		
Imaging Strategy	**TFAST® DH View**	**TFAST® PCS View**
*Image the heart globally in its entirety using the bright white pericardium in the far-field as a landmark		*TFAST® Right and Left PCS – Anechoic (Black) Triangulations
*Image toward the muscular apex of the heart where no heart chambers can be mistaken for free fluid	*TFAST DH View – Anechoic (Black) Triangulations	
*"TFAST® Slide" caudally and then cranially into the "cardiac-diaphragmatic pouch" and the "cardiac-cervical pouch"		"Curtain Sign" for pleural effusion
Gregory Lisciandro, DVM, DABVP, DACVECC, FASTVet.com and Hill Country Veterinary Specialists *© 2016, 2018, 2019*		

Clinical Indications/Applications for TFAST® - Global FAST® is Our New Quick Assessment Test

The use of *standardized* TFAST® and Vet BLUE® should serve as routine as an "extension of the physical exam" for all dogs and cats that are abnormal or respiratory suspects (*better* the Global FAST® Approach). Questionable findings within the thorax using the FAST DH View should be confirmed via TFAST® PCS View(s) or Vet BLUE® or both and by serial exams, repeating TFAST® and Vet BLUE® at least once 4-hours later.

Goal-Directed Templates for TFAST®

*****Right and left sides are listed in templates for the CTS and PCS views

***Chest Tube Site (CTS) - Glide Sign?** **Present** (normal) -- no Pneumothorax **or** **Absent** – Pneumothorax **or Indeterminate** or **Not Assessed**

***Location of Lung Point?** **Upper 1/3** or **Middle 1/3** or **Lower 1/3** or **Indeterminate** or **Not Assessed**

***CTS - Lung Rockets (also called B-lines)**? **Present** (no PTX) – interstitial lung fluid (edema, hemorrhage) or **Absent** – no interstitial lung fluid or **Indeterminate** or **Not Assessed**

***CTS - Step Sign?** **Present** – concurrent thoracic wall trauma (rib fractures, hematoma, intercostal muscle tear) or pleural space disease is suspected or **Absent** - no concurrent thoracic wall trauma or pleural space disease is suspected or **Indeterminate** or **Not Assessed**

***PCS view - Pleural or Pericardial Eff.?** **Absent**- no pleural or pericardial fluid **or Present** - pleural or pericardial fluid **or** both (mild, moderate, or severe) or **Indeterminate** or **Not Assessed**

TFAST Echo® Views:

Left Ventricular Short-axis Mushroom View (LVSA): Filling: **Adequate** suggesting normovolemia or **Inadequate** suggesting hypovolemia or **Indeterminate** or **Not Assessed**
Contractility: **Unremarkable** or **Decreased** or **Indeterminate** or **Not Assessed**

Left Atrial to Aortic Ratio (LA:Ao) on Short-axis: **Unremarkable** or **Increased** or **Indeterminate** or **Not Assessed**

Right Ventricular to Left Ventricular Ratio (RV:LV) on Long-axis: **Unremarkable** or **Increased** or **Indeterminate** or **Not Assessed**

DH View:
Pleural effusion: **Absent** or **Present (mild, moderate, severe)** or **Indeterminate** or **Not Assessed**
Pericardial effusion: **Absent** or **Present (mild, moderate, severe)** or **Indeterminate** or **Not Assessed**
[$]**Hepatic Venous Distension:** **Present** or **Absent** or **Indeterminate** or **Not Assessed**
[&]**Caudal Vena Cava Characterization:** **FAT** or **flat** or **bounce** or **Indeterminate** or **Not Assessed**

Cardiac Tamponade: **Present or Absent or Indeterminate** or **Not Assessed**

Comments: _____

KEY: CTS = chest tube site; **PCS** = pericardial sac; **LV** = left ventricle, **PTX** = pneumothorax, **NA** = Not Assessed.

Note: The TFAST® is a rapid ultrasound procedure used to help detect major chest wall, lung, and pleural and pericardial space problems as a screening test in order to better direct resuscitation efforts, help better direct diagnostics, and manage hospitalized critically ill patients. TFAST® exam is not intended to replace thoracic radiographs, or complete echocardiography.

[$]The hepatic veins should *not* be apparent in both dogs and cats placed in lateral recumbency or standing or sternal. When imaged, the branching has been referred to as the "Tree Trunk Sign."

[&]The caudal vena cava can be alternatively referred to as a bounce = fluid responsive cava (~35-50% diameter change); FAT = fluid intolerant cava (distended with increased maximum height < 1cm in dogs < 9kg, and > 1.5cm in dogs > 9kg with little maximum height change [< 10%]); flat = hypovolemic cava (small with decreased maximum height of < 0.3cm in dogs < 9kg and < 0.5cm in dogs > 9kg with little maximum height change [< 10%]). *See Global FAST® Monitoring Proceedings.*

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See also Vet BLUE® Proceedings for additional References.

Vet BLUE® Introduction to Its Regional, Pattern-based Approach,
Its B-line Scoring System, and Its Visual Lung Language for the Respiratory Patient

Emerald Coast Veterinary Conference, Sandestin, FL, USA
Gregory R. Lisciandro, DVM, Dipl. ABVP, Dipl. ACVECC
Hill Country Veterinary Specialists & FASTVet.com, Spicewood, Texas USA
Email LearnGlobalFAST@gmail.com
Cell 210.260.5576
Website FASTVet.com
Textbook Point-of-care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2021

Use of Lung Ultrasound in Small Animals - The Vet BLUE®

The historical reluctance to proactively apply lung ultrasound (LUS) to small animals with respiratory distress is irrational in many respects. The overriding belief that air-filled lung creates insurmountable obstacles, and the continued belief in small animal medicine that imaging lung is difficult to perform leading to mistakes, perpetuate its delayed use in small animal veterinary medicine.

TFAST® (2008), referring to our thoracic FAST protocol, was the first standardized abbreviated veterinary ultrasound exam of the thorax. TFAST® included the Chest Tube Site (CTS) for the detection of pneumothorax (PTX) and lung contusions; the Pericardial Sites for the detection of pleural and pericardial effusion plus fundamental echocardiography views; and the Diaphragmatico-Hepatic View for pleural and pericardial effusion, cardiac imaging, and lung imaging along the pulmonary-diaphragmatic interface. Moreover, the TFAST® DH View may also be used for assessing volume status via characterization of the caudal vena cava and hepatic veins.

With the finding of lung pathology during TFAST®, the author extended the lung examination from the TFAST® CTS View with an additional 6 regional lung views plus the Diaphragmatico-Hepatic View. This novel regional pattern-based proactive LUS exam was named Vet BLUE® - "Vet" for "veterinary" and "BLUE" for "brief lung ultrasound exam" and "BLUE" also implying cyanosis and all respiratory small animals. The Vet BLUE® protocol was developed in 2010, being the first published proactive LUS protocol. Vet BLUE®is also the most studied in our veterinary literature with over 8 peer-reviewed publications.

The Fundamentals of Vet BLUE®

Patient Preparation. Vet BLUE® sites are not shaved! All images shown by the author are from unshaved sites. To optimize the image quality, the fur is wetted with minimal amounts of 70% isopropyl alcohol, the fur parted to expose the skin, followed by the application of gel and the probe head then directly opposed to skin. A common mistake is placing the probe head on a wetted mat of hair, which leads to the phenomenon of air trapping within the wetted mat. Air trapping causes the deflection of the echoes from the probe because ultrasound cannot transit

through air; and minimizing the numbers of echoes making it to the region of interest compromises image quality.

Patient Positioning. Vet BLUE® is preferably performed in standing (or sternal) which is safer for dogs and cats respiratory compromised, in respiratory distress, or those that are hemodynamically fragile or unstable. A roll of towels (or paper towels) under the forelegs of a cat is an easy tolerated maneuver to gain access to the ventral Vet BLUE® views, and the TFAST PeriCardial Site views. Vet BLUE® may also be performed in dogs and cats in lateral recumbency when they are laterally recumbent. The concept that air rises to least gravity-dependent regions and fluid conversely falls to most gravity-dependent regions should be kept in mind relative to patient positioning. This concept is especially important when drawing conclusions regarding pneumothorax (PTX) because free air in the pleural cavity (rises); and pleural effusion because free fluid in the pleural cavity (falls) into gravity dependent areas. Pericardial effusion also falls into gravity dependent regions but is contained within the pericardial sac.

Probe Type. The curvilinear (convex) probe is the preferred probe in human medicine by the non-radiologist. In veterinary medicine, the curvilinear (microconvex) probe is also most preferred because it is flexible enough to be used to image all aspects of the Global FAST® Approach, which includes AFAST®, TFAST®, and Vet BLUE®, our lung ultrasound protocol. The linear probe may also be used, and it does in fact give exceptional detail of the lung surface, however, it is unnecessary. The linear probe has the disadvantage of not being able to extend beyond Vet BLUE® to AFAST® and TFAST®. The phased-array cardiac probe should not be used because it cannot accurately identify the gator sign orientation or count numbers of B-lines.

Probe Frequency. Generally, frequencies of 5-10MHz adequately image lung. Other considerations that affect the image include the focus position, which should be across or just below the “lung line”, the time gain compensation and overall gain, generally turned down for more contrast, however, with enough gain to image through the far field, and the preset. The author uses the abdominal preset for the entire Global FAST® Approach, including lung and heart. This saves time and works well once the sonographer becomes experienced with their machine. Other factors discussed below include proper orientation, depth, and manipulating the echoes to your advantage. The one-eyed gator sign (rib in the center of the screen) and manipulating the angle of insonation, when imaging the “lung line”, optimize the image.

Lung Imaging Orientation. All LUS orientation is founded on the visualization of the “gator sign” for its importance in properly identifying the intercostal space. By identifying the intercostal space, the “lung line” may be identified, which has also been referred to as the pulmonary-pleural interface, where visceral and parietal pleura are directly opposed.

We prefer to use “lung line” because when pathology exists within the pleural space, the lung is displaced away from the parietal pleura. The scanning plane is oriented perpendicular to the long axis of the ribs with the probe marker and screen orientation marker toward the head and to the left of the screen, respectively.

By doing so, the head will be to the left of the screen (cranial) and the tail to the right of the screen (caudal). Depth is generally set between 4-8 cm with a good way to remember is the l-u-n-g has 4 letters, so start with 4cm for small dogs and cats and increase to 8cm (sometimes

greater) for large dogs. If the "gator sign" is not identified then other bright white or hyperechoic lines can easily be mistaken for the "lung line" including the spine of the scapula, an air-filled stomach, A-lines, and even fascial planes within thorax-associated muscle.

The "GATOR SIGN" – Fundamental Lung Ultrasound Orientation

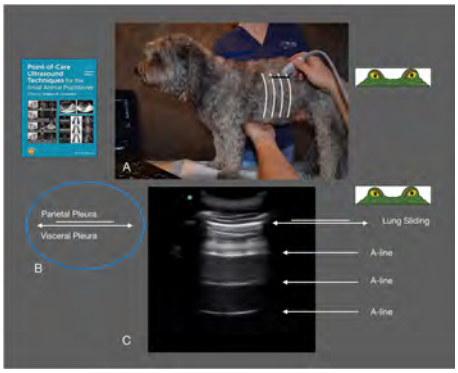
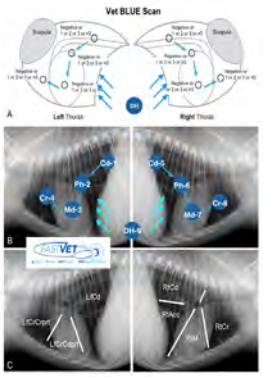


Figure. Gator Sign Orientation. The rounded ribs are likened to the eyes, and the bright white hyperechoic "lung line" or "pulmonary-pleural interface" to the bridge of its nose as a partially submerged alligator (gator) peers at the sonographer. The proximal bright white, hyperechoic line, is the focus of ALL lung ultrasound to ensure one accurately identifies where lung is expected to be in normalcy, referred to as the "Lung Line." The "Lung Sliding" may be described as sliding pleural and visceral pleura (micro level) versus the author's preference of lung sliding along the thoracic wall (macro level). *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and FASTVet.com © 2014, 2020 and the "Gator Sign" in the veterinary literature (Lisciandro et al. Vet Radiol Ultrasound 2014).*

The Vet BLUE® - Its 9 Acoustic Windows



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How to Perform. There is no shaving of hair. Small amounts of 70% isopropyl alcohol are used to wet and part the hair for direct visualization of the skin, followed by alcohol-based hand sanitizer or commercially available acoustic coupling gel. The Vet BLUE® begins at the CTS view of TFAST® and establishing the "gator sign" orientation. The probe is then moved through

regional locations that are bilaterally applied as follows: caudodorsal lung region, perihilar lung region, middle lung region, and lastly the cranial lung region. The methodology has changed from our original protocol by better defining each regional view by locating the caudodorsal transition zone (CdTZ). The CdTZ is located by starting directly above the xiphoid in the upper third of the thorax and locating the "curtain sign" effect that distinguishes between pleural and abdominal cavities. If it is not immediately located, the probe is generally slid caudally searching for obvious abdominal structures and then sliding cranially finding the "curtain sign." The principle is very important because abdominal structures are easily mistaken for lung pathology without this training.

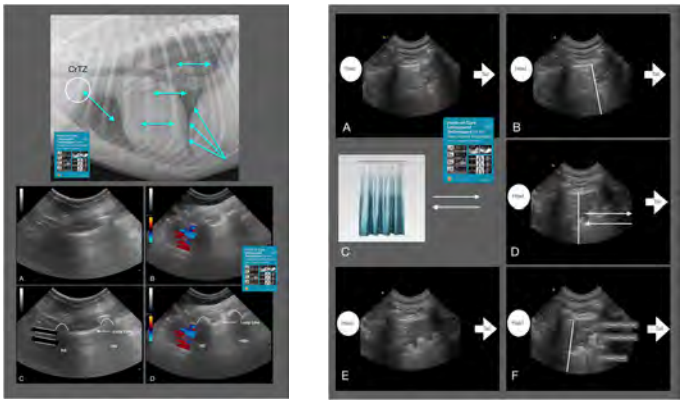

Figure. Caudal and Cranial Vet BLUE® Transition Zones. The composite to the left is the cranial transition zone found by sliding along the "Lung Line" unit it ends in the soft tissue of the thoracic inlet with its jugular and carotid vessels. The caudal transition zone is identified by the "Curtain Sign" and the linear border of air that identifies the pleural cavity to the left and the abdominal cavity to the right. *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and FASTVet.com © 2014, 2020.*

Once the CdTZ is located, the probe is slid 2-3 intercostal spaces cranially and away from the CdTZ. While sliding away from the CdTZ lung can be evaluated for pathology since the sonographer knows they are in fact over the pleural cavity. The location 3 intercostal spaces away from the CdTZ is considered the starting point. Its intercostal space is surveyed and then the sonographers slides one space caudally over its intercostal space, returns to the primary ICS and then moves one more ICS cranially so that a minimum of 3 ICSs are interrogated at the caudodorsal lung region. A line, referred to as the "Vet BLUE Line", is then drawn from the caudodorsal starting point to the patient's elbow and approximately halfway to the elbow is the perihilar region. The methodology is repeated as done previously. Interrogating the primary ICS, sliding caudally and interrogating its ICS, before returning to the primary and sliding one ICS cranially, again imaging a minimum of 3 ICSs. The middle lung region is generally at the level of the elbow in a standing dog or cat. The heart is a good landmark and when found ventrally, the probe is moved dorsally until the "gator sign" is located immediately dorsal to the heart. The methodology performed at the two previous views is repeated sliding caudally first, then back to the primary and another ICS cranially for a minimum of 3 ICSs. The cranial lung region is imaged a little differently by finding the cranial cervical transition zone (CrTZ) by following the "lung line"

until it drops off into soft tissue of the thoracic inlet. The path for the Vet BLUE® is like a check mark, meaning as the sonographer slides cranially into the thoracic inlet, they must also slide dorsally. Assurance of being in the thoracic inlet is the presence of pulsating arteries with a rib shadow immediately caudal followed by the "gator sign" and a "lung line." The 3 ICSs at the cranial lung region are performed by sliding from the thoracic inlet and then the first rib, and counting first rib, first ICS, second rib, second ICS, third rib, third space. This completes the Vet BLUE® and we have found this protocol to be very repeatable compared to our original methodology. Another key (Vet BLUE® rule) is always slide caudally first anytime when imaging the thorax to always ask the question, where is the abdominal cavity? Another Vet BLUE® rule is that if you do not have a "gator sign" orientation you cannot confidently assess lung.

Always perform Vet BLUE® in this same order as findings are better remembered, starting left and finishing on the right if the patient allows. We believe the most efficient Global FAST® protocol in a standing patient is beginning with the left Vet BLUE® and then moving to the left TFAST® Pericardial Site followed by a standing AFAST® and Focused Spleen before moving to the right Vet BLUE®, right Pericardial Site and its TFAST® echocardiography views, and ending on the AFAST® Hepato-Renal 5th Bonus View. A video may be found in the Free Resources of the FASTVet.com website.

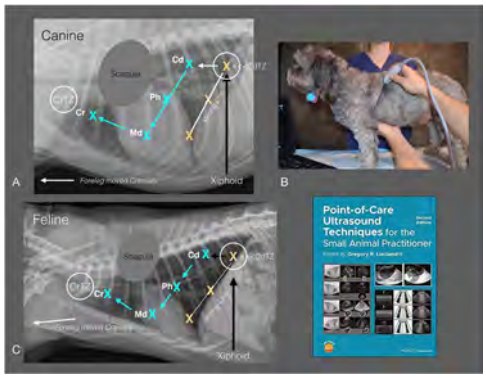

Figure. Selecting the Vet BLUE® Line. Find the Caudodorsal Transition Zone (CdTZ) by finding the "Curtain Sign" – see text for greater detail. The probe is slid 3 intercostal spaces cranial to the CdTZ for your starting Vet BLUE® view, the caudodorsal lung region view. From there survey 3 intercostal spaces, one caudal and one cranial from your starting point. Drawing a line to the elbow is performed next, this is your Vet BLUE Line. Approximately halfway is the perihilar lung region, and at the approximately of the elbow is the middle lung region. Then, the probe is slid cranio-dorsal into the thoracic inlet to find the Cranial Transition Zone (CrTZ). From the CrTZ the probe is lid caudally over the first rib, first intercostal space, the second rib, second intercostal space, third rib, third intercostal space. This Vet BLUE® methodology is newer and more reproducible than previously published (Lisciandro et al. ongoing research 2020). *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

Vet BLUE® - Wet versus Dry Lung Concept

Dry Lung. Dry aerated lung at its surface is defined as a bright white, hyperechoic "lung line" accompanied with "lung sliding" and A-lines repeating through the far field. Remember A-lines as air reverberation artifact because "Air" begins with the letter "A." "Lung sliding" is the to and fro motion of the lung sliding along the intercostal space much like an Etch-a-Sketch cursor moving to and fro. The micro description for "lung sliding" is the sliding or parietal and visceral pleura. On a macro level, "lung sliding" the lung surface sliding along the intercostal space. The distinguishing feature between normal aerated lung on its surface and pneumothorax is presence and absence of "lung sliding", respectively. Each of these conditions is highlighted by a strong air interface at the intercostal space. From our Vet BLUE® research, expect absent B-lines at all views in adult dogs and cats and puppies and kittens over 6-weeks of age. A single B-line at a single regional view is uncommon but can be also support a "dry Vet BLUE® profile." The bottom line is to place any and all B-lines during Vet BLUE® in clinical context and record your findings for future comparison.

Wet Lung. Alveolar-interstitial edema creates a unique artifact referred to as B-lines or ultrasound lung rockets. These terms may be used interchangeably meaning the same thing. The use of "B" in B-lines is only because "B" follows "A" in the alphabet. The vertical laser-like bright white, hyperechoic streaks originate off the "lung line" while extending through the far field *without* fading while swinging like a pendulum in synchrony with phases of respiration. Their presence is referred to as alveolar-interstitial syndrome because a pattern-based approach is needed for developing a working diagnosis as to their cause. For the vast majority of our small animal patients, these unique artifacts in lung are created by the strong difference in acoustic impedance between fluid and air and the cuffing of air (alveoli) around fluid (Soldati personal communication). However, pleural surface fibrosis also can create the artifact and so may pulmonary nodules and ingesta within the stomach that can mimic lung as well, referred to as pseudo B-lines because these conditions do not represent forms of alveolar-interstitial edema.

Quick Facts - B-lines or ultrasound lung rockets do the following:

- Immediately rule out pneumothorax at that location on the thoracic wall
- Are lung contusion in trauma until proven otherwise
- Guide diuretic use in left-sided congestive heart failure patients
- Their absence with dry lung all Vet BLUE views rules out all common wet lung conditions including left-sided congestive heart failure, non-cardiogenic pulmonary edema, pneumonia, pulmonary hemorrhage, and lung contusions.

Vet BLUE® - Wet versus Dry Lung Approach in Respiratory Patients

From our Vet BLUE® research, expect absent B-lines at all views in adult dogs and cats and puppies and kittens over 6-weeks of age. A single B-line at a single regional view is uncommon but can be also support a "dry Vet BLUE® profile." The bottom line is to place any and all B-lines during Vet BLUE® in clinical context and record your findings for future comparison.

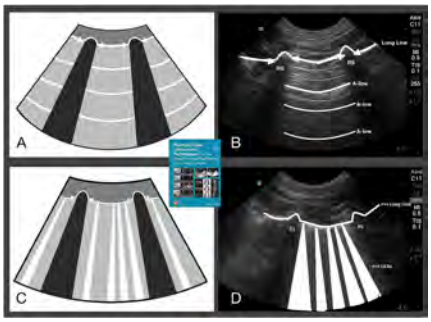

Figure. Wet versus Dry Lung. Dry Lung is defined as A-lines with "Lung Sliding." Wet Lung is defined as B-lines with hyperechoic laser like vertical streaks that obliterate A-lines and swing like a pendulum in respirophasic synchrony. If they don't follow this rule, then they are not B-lines (also called Lung Rockets). *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

It is important to work through the following cases and what would be expected in each barring complications: left-sided congestive heart failure in dogs other than Doberman Pinschers, left-sided congestive heart failure in cats; non-cardiogenic pulmonary edema (electrocution, strangulation, neurogenic); tracheal collapse, laryngeal paralysis, infectious tracheobronchitis (now referred to as CIRD, canine infectious respiratory disease), aspiration pneumonia, bacterial bronchopneumonia, pericardial effusion, pyrexia/fever/heat stroke, feline asthma, canine bronchial disease, canine anaphylaxis, feline anaphylaxis as common respiratory examples.

The most rapid and sensitive manner in which to rule out left-sided *congestive* heart failure is the use of Vet BLUE® and the finding of absent B-line, dry lung all Vet BLUE® views (plus all other common wet ling conditions).

Rule Outs for Dry Lung All Vet BLUE® Views
RESPIRATORY
Pulmonary Thromboembolism (PTE)
Pneumothorax
Dynamic Upper Airway Conditions (e.g., Collapsing Trachea, Laryngeal Paralysis)
Upper Airway Obstruction (e.g., Mass, Oropharyngeal Swelling)
Chronic Obstructive Pulmonary Disease (COPD), Bronchitis
Feline Asthma
Tracheobronchitis (e.g., Infectious, Inflammatory, Irritant)
Centrally located lung pathology away from the lung line (missed by Vet BLUE)
CARDIAC
Pericardial Effusion / Cardiac Tamponade
Cardiac Arrhythmia
Dilated Cardiomyopathy (DCM)
Right-sided Congestive Heart Failure (CHF)
*Pulmonary Hypertension
UNDIFFERENTIATED HYPOTENSION
Canine Anaphylaxis

Hemoabdomen
Hemothorax
Hemoretroperitoneum
Cavitary Effusion
Gastric Dilatation Volvulus/Bloat
Sepsis
OTHER NON-RESPIRATORY
Pyrexia / Heat Stroke
High Fever
Severe Metabolic Acidosis
Severe Anemia
Note: Dry Lung All Vet BLUE® Views Rules Out Clinically-Relevant Wet Lung Conditions
***Pulmonary Hypertension can be Wet Lung as well*
Gregory Lisciandro, DVM, FASTVet.com, Copyright 2015

Pseudo B-lines

Ingesta in the stomach and nodules also cause B-lines similar to the differences in acoustic impedance and cuffing of alveoli around fluid with forms of alveolar-interstitial edema. We call these "pseudo B-lines" to differentiate them from alveolar interstitial edema and categorize them as a different subset.

Vet BLUE® Scoring System

In 2006, Volpicelli and colleagues showed that numbers of B-lines on LUS correlated with degree of alveolar- interstitial edema on computed tomography (CT) in human lung. This is truly a remarkable finding because LUS may be performed point-of-care, is rapid and radiation sparing, and time sensitive whereas CT is expensive, limited availability, risky and is comparable to 100 chest x-rays. We developed a Vet BLUE® B-line Scoring System taking the maximum number of B-lines over a single intercostal space at each respective Vet BLUE® region as 1, 2, 3, >3, and infinity.

Vet BLUE® Scoring System Use for Guiding Diuretic Usage

By using the Vet BLUE® B-line Scoring System in cases with known left-sided *congestive* heart failure, our scoring carries the potential to guide loop diuretic therapy by using the strong positive model of >3 and infinity being strong positive and 1, 2, and 3 as being weak positive. Loop diuretic therapy in the author's opinion is as abused as any other drug like glucocorticoids by its administration empirically in respiratory patients without evidence-based information; and lack of sensitive ways to determine the degree of alveolar-interstitial edema based on lung auscultation and thoracic radiography. Now, Vet BLUE® is your new more sensitive tool for guiding loop diuretic therapy and preventing its many side effects of metabolic alkalosis, hemoconcentration and renal injury and failure.

Vet BLUE® Scoring System Use for Lung Contusion Severity

The use of maximum numbers of B-lines over numbers of positive regional Vet BLUE® views provides a lung contusion scoring system. By recording results, lung contusions may be monitored for worsening, their resolution or without change (static).

Figure. The Vet BLUE® B-line Scoring System. The Vet BLUE® B-line Scoring System for Use in Wet versus Dry Lung, Guiding Diuretic Use in Left-sided Congestive Herat Failure, and Assessing and Severity and Monitoring Lung Contusions (and Pulmonary Hemorrhage). *This material is reproduced with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists, FASTVet.com © 2014, 2020.*

Vet BLUE® Visual Lung Language for Signs of Consolidation

The lecture will briefly touch on signs of consolidation of Shred Sign (air bronchogram), Tissue Sign (hepatization of lung), Nodule Sign, and Wedge Sign (pulmonary thromboembolism).

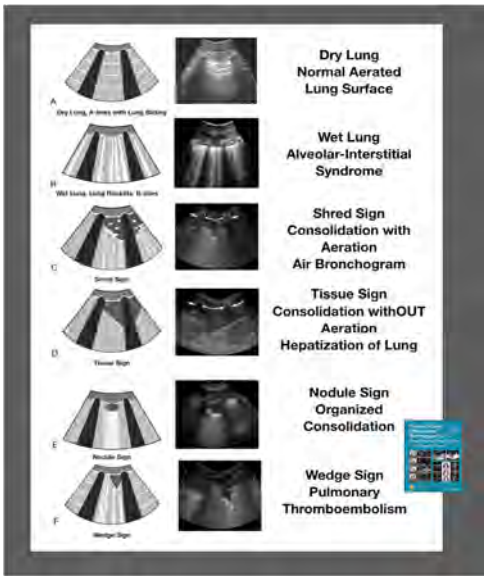

Figure. Vet BLUE® Visual Lung Language and Its 6 Signs. Vet BLUE® Visual Lung Language from most to least normal, from less severe to more severely affected lung as Dry Lung to Wet Lung (alveolar-interstitial edema) to Shred Sign (air bronchogram) to Tissue Sign (hepatization of lung) to Nodule Sign to Wedge Sign (pulmonary thromboembolism). *This material is reproduced and modified with permission of John Wiley & Sons, Inc., Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, Wiley ©2014, ©2020 and Greg Lisciandro, Hill Country Veterinary Specialists and FASTVet.com © 2020.*

Comparison of Vet BLUE® to Thoracic Radiography and Computed Tomography

Vet BLUE® is proving itself as a more sensitive test than thoracic radiography for wet lung conditions (types of alveolar-interstitial edema) and pneumothorax comparing much more closely to computed tomography (CT). We have several clinical studies published, in press, and in the process of being submission for peer review that support this statement. As for types of consolidation including nodules, more studies are needed to make a statement on the performance of Vet BLUE® to thoracic radiography and CT. One recent study of which the author was involved showed that Vet BLUE® was similar but not superior to thoracic radiography in sensitivity and specificity for pulmonary nodules.

Always Strive for The GLOBAL FAST® APPROACH

"Selective imaging" leads to "confirmation bias error" and "satisfaction of search error" and is a major concern with the POCUS movement. For example, a Vet BLUE® profile on a large breed dog is dry all Vet BLUE® views and along with an unremarkable thoracic radiograph the conclusion is drawn that the dog has upper airway or bronchial disease. However, the Global FAST® Approach provides an unbiased set of 15 data imaging points of the abdomen and thorax including heart and lung surface. By using this standardized global approach important findings are integrated into the final assessment. In fact, this dog has obvious poor contractility on TFAST® echocardiography views (likely dilated cardiomyopathy), and on AFAST® has a splenic mass with a negative AFAST® abdominal fluid score of 0. In this case you can see how integrating the Global FAST® Approach provided a much better patient assessment over thoracic radiography (and physical exam and blood and urine testing).

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Global FAST® Integration for Patient Monitoring and Rapidly Ruling Out the Hs and Ts of Treatable Shock

Emerald Coast Veterinary Conference, Sandestin, FL, USA
Gregory R. Lisciandro, DVM, Dipl. ABVP, Dipl. ACVECC
Hill Country Veterinary Specialists & FASTVet.com, Spicewood, Texas USA
Email LearnGlobalFAST@gmail.com
Cell 210.260.5576
Website FASTVet.com
Textbook Point-of-care Ultrasound Techniques for the Small Animal Practitioner, 2nd Edition, Wiley ©2021

Global FAST® For Patient Monitoring

Global FAST® is the combined use of AFAST®, TFAST® and Vet BLUE® as a single ultrasound exam. Global FAST® is unique in screening both body cavities and is a standardized format that includes 15 acoustic windows or views.

Global FAST® can do the following with no additional views from its standardized 15 view format:

- AFAST® and its target-organ approach for obvious soft tissue abnormalities
- AFAST® and its abdominal fluid scoring system and categorizing small volume versus large volume hemorrhagic and non-hemorrhagic effusions
- AFAST® Cysto-Colic View Formula for urinary bladder volume estimation and urine output
- AFAST® Spleno-Renal View in right lateral for pneumoperitoneum
- AFAST® Hepato-Renal Umbilical View for gastrointestinal motility
- TFAST® for pleural and pericardial effusion
- TFAST® for pneumothorax (PTX)
- TFAST® for semi-quantitating and tracking PTX through the use of the Lung Point
- TFAST® echocardiography views for problems with volume, contractility, and left- and right-sided heart conditions
- TFAST® for obvious soft tissue abnormalities of the heart and thorax
- TFAST®-AFAST® for characterization of the caudal vena cava and hepatic veins for volume status
- Vet BLUE® regional, pattern-based approach for respiratory assessment
- Vet BLUE® B-line Scoring System
- Vet BLUE® Visual Lung Language for types of lung consolidation

Performing Global FAST® Most Efficiently

There are 2 major ways to efficiently perform Global FAST® and we refer to these as Global FAST blends of its 3 components.

In a standing patient, the left Vet BLUE® views are performed first, followed by a depth change for the left TFAST® PeriCardial Site view followed by the Diaphragmatico-Hepatic (DH) View and then continuing with a standing AFAST® with a Focused Spleen. The sonographer then moves to the right side of the patient and performs the right Vet BLUE® followed by the TFAST®

echocardiography views at right PeriCardial Site followed by the final AFAST® HR5thBonus View. The external placement of the probe is the same for AFAST® whether the patient is standing or in right lateral recumbency. If AFAST® is negative for free fluid in standing, then lateral recumbency is not necessary. If AFAST® is positive for free fluid then when safe to do so, place the patient either right or left lateral recumbency and wait 3 minutes for fluid to re-distribute, followed by AFAST® fluid scoring. See the Global FAST® video at FASTVet.com under Free Resources.

If the patient arrives and is best left in lateral recumbency the following order is described. When in right lateral recumbency, the AFAST® and Focused Spleen are performed first, then the left Vet BLUE®, left TFAST® PeriCardial Site view followed by the right TFAST® PeriCardial Site view for TFAST® echocardiography. The patient then must be moved to access the right Vet BLUE® to complete the Global FAST® and AFAST® HR5th Bonus View. The analogous approach would be used when the patient is in left lateral recumbency.

AFAST®

The Abdominal Fluid Scoring System

Small animals are preferably in right lateral because it facilitates right TFAST® Pericardial Site views including the 4 TFAST® Echocardiography Views (short-axis left ventricular view, short-axis LA:Ao ratio, and long-axis 4-chamber view, long-axis left ventricular outflow track). Either lateral recumbency, however, is validated for AFAST® abdominal fluid scoring was originally as follows: an abdominal fluid score (AFS) of "1" is assigned to any positive AFAST view thus making the scoring system range from 0-4.

Thus, an abdominal fluid score (AFS) of "1" is a positive at any *single* AFAST® view; and an AFS of "2" positive at any two views; and an AFS of "3" positive at any 3 views; and an AFS of “4” positive at all 4 AFAST® views. The AFS gives more objective semi-quantitative assessment for effusions over terms like “trivial”, "mild", "moderate" and "severe" allowing for better tracking of effusions as static (no change in score), resolving (lower score), and worsening (higher score). Very importantly, the actual AFAST® views are recorded as positive or negative, thus potentially providing support for the origin of the effusion in lower scoring patients. As an example, if a bleeding patient has an initial AFS of 1, scoring positive at the DH View, and then progresses to an AFS of 4 and requires exploratory surgery to stop the bleeding, logic would dictate that the region of the DH View is likely the origin of hemorrhage. If only "AFS 1" is recorded without location, this information is lost.

Modification of the AFAST®-Abdominal Fluid Scoring System

More recently the author modified the AFS to account for smaller pockets of free fluid as a score of "1/2" rather than a full "1." In cats and dogs, an AFS of "1/2" is assigned when the fluid pocket's maximum dimension is ≤5mm and ≤ 1cm, respectively. This modification better classifies patients that have combinations of small and larger fluid pockets. The use of this modification is based on more recent clinical studies (Lisciandro et al. 2020). *See AFAST® Proceedings for more detail.*

Making Sense of the AFS in The Bleeding Patient

In cases of hemorrhage, the AFS help categorize intra-abdominal bleeding as small volume bleeding, AFS <3, versus large volume bleeding, AFS ≥3. AFS <3 (1/2 to 2 1/2) dogs and cats do not have enough intra-abdominal hemorrhage for anemia. Thus, if a dog or cat has an AFS <3 and is anemic, there are the following 4 major scenarios in the acute setting: 1) pre-existing anemia, 2) bleeding somewhere else thus do Global FAST® and look internally - pleural cavity, retroperitoneal space, pericardial sac and lung; and consider gastrointestinal, urinary, and reproductive tracts, as well as fracture sites, 3) hemodilution (less common with graduated fluid administration strategies), or 4) lab error. Conversely, when AFS is ≥ 3 then the patient is considered as having potentially life-threatening hemorrhage having enough intra-abdominal hemorrhage to predictably become anemic.

The AFAST® Cysto-Colic Urinary Bladder Volume Formula

At the AFAST Cysto-Colic View the urinary bladder is imaged in longitudinal (sagittal) and the best largest oval is acquired in this plane and measured followed by transverse orientation and acquiring the largest oval which is measured. Measurements in (cm) will give you an estimation of urinary bladder volume in (ml) by using Length x Width x Height (cm) x 0.625 (Lisciandro and Fosgate 2017). With measurements over time, urine output may be non-invasively estimated.

The AFAST® Spleno-Renal View for Pneumoperitoneum

Air rises and fluid falls into gravity dependent regions we refer to as “pouches.” Thus, in right lateral recumbency, the Spleno-Renal View, least gravity view where air would rise, is used to screen for the enhanced peritoneal stripe sign of pneumoperitoneum. The concept is easy to understand once explained as free air is continuous with the hyperechoic peritoneal lining or body wall. If an anechoic gap exists, then the air is from the gastrointestinal tract and not free air. Ultrasound is extremely sensitive for free air and post-operative cases are an excellent way to learn the detection of pneumoperitoneum since most have free air from their laparotomy. When free air is suspected, radiography is an excellent confirmatory imaging modality.

The AFAST® Hepato-Renal Umbilical View for Gastrointestinal Motility

The stomach/proximal duodenum and the jejunum may be observed for peristalsis, expecting 4-5 minute^{-1} and 1-3 minute^{-1}, respectively, if food is present in the canine gastrointestinal tract, helping detect ileus. With food absence, including intentional fasting, ileus occurs in normalcy and must be placed into clinical context.

TFAST® Echo Views

Left Ventricular Short-axis View For Volume and Contractility

The left ventricular short-axis view (LVSA) is acquired just below the mitral valves at the level where the chordae tendinae come off the left papillary muscles referred to as the short-axis "mushroom" view. The filling and size of the "mushroom" is a reflection of patient volume status. Contractility is also assessed subjectively. Poor filling indicating poor volume can be supported or refuted by assessing the caudal vena cava; and contractility by triggering complete echocardiography. A patient thought to have poor contractility, i.e., dilated cardiomyopathy, may be treated and better stabilized during the delay of acquiring complete echocardiography. Your

"Global FAST® Non-echo Fallback View" for volume status is characterization of the caudal vena cava and its associated hepatic veins at the AFAST®-TFAST® DH View.

Long-axis 4-chamber View and Its Right Ventricular (RV) to Left Ventricular (LV) Ratio (RV:LV)
The normal RV:LV ratio is 1:3-4 or the RV being a small triangle when compared to the LV being a much larger triangle. When the RV is nearly the same size of the LV, then right heart problems should be suspected, and complete echocardiography is indicated as right sided heart disease is present until proven otherwise. By recognizing the abnormality of right heart enlargement, therapy may be adjusted to better avert complications. In acute respiratory distress, the finding of an enlarged RV suggests massive PTE and Vet BLUE is used to search for the "Wedge Sign" in upper lung regions. Your "Global FAST® Non-echo Fallback View" for right-sided heart problems is characterization of the caudal vena cava and its associated hepatic veins at the AFAST®-TFAST® DH View.

Left Ventricular Short-axis for The Left Atrial (LA) to Aortic (Ao) Ratio (LA:Ao)
The normal LA:Ao Ratio is <1.3 (dogs) and <1.6 (cats) and the most challenging TFAST® echocardiography view. Your "Global FAST® Non-echo Fallback" strategy is performing the easier, less stressful, Vet BLUE®. Dry lung all Vet BLUE® views rapidly rules out any clinically relevant left-sided *congestive* heart failure (Lisciandro et al. 2016).

The Non-Echo Fallback Views for Left- and Right-sided Cardiac Problems
Remembering each is fairly straight forward - left-sided *congestive* heart failure must have lung edema (Vet BLUE®); and right-sided *congestive* heart failure must have hepatic venous congestion and thus an enlarged caudal vena caval and branching of hepatic congestion at the AFAST®-TFAST® DH View.

Characterizing the Caudal Vena Cava and Hepatic Veins
The caudal vena cava (CVC) where it traverses the diaphragm reflects volume status, an approximation for central venous pressure (CVP). Eyeball and characterize the CVC as being 1) "FAT" or distended with a maximum height > 1.0 cm in smaller dogs <9kg and >1.5cm in dogs 9kg along with a <10% change in height, high CVP, and called a fluid intolerant CVC, or 2) "flat" or collapsed with a maximum height <0.3 cm in smaller dogs < 9kg and <0.5cm in dogs > 9kg along with a <10% change in height, low CVP, and called a hypovolemic CVC, or 3) having a "bounce" (~35-50% change in diameter, in the ballpark of normal CVP) called a fluid responsive CVC. *See the CVC Chart with maximum height measurements at FASTVet.com under Free Resources.*

Measuring the CVC
Using M-mode can be challenging and difficult with a lot of patient movement and prone to error by not having the optimal sonographic plane. B-mode is another option and used by freezing and rolling the cine ball for minimal and maximal diameter measurements over several seconds that include the cardiac and respiratory cycle. Scrolling for the maximum and minimum heights can be used to calculate its distensibility index (change in CVC max and CVC min/maximum diameter of CVC x 100%). However, absolute height measurements have been created for dogs and are

generally much easier (and faster) than calculations. *See the CVC Chart with maximum height measurements at FASTVet.com under Free Resources.*

Use of The Lung Point for Diagnosing and Monitoring Pneumothorax (PTX)

The use of the Lung Point is a means to increase the sensitivity for the diagnosis of PTX and to track worsening or resolving PTX and help with decision-making regarding need for thoracocentesis. For example, post lung lobe aspirate, chest tube placement/removal, during anesthesia and mechanical ventilation, or other invasive thoracic procedures, the Lung Point semi-quantifies the degree of PTX. The use of the "TFAST® PTX 1/3s Rule" helps track and semi-quantitate the degree of pneumothorax, has been developed and is used by the author.

Table Using the Author's "TFAST® PTX 1/3s Rule" for the Location of the "Lung Point" to Categorize the Degree of Pneumothorax (PTX) and for Monitoring

Location of the "Lung Point" with the Patient in Standing or Sternal and Categorizing the Degree of Pneumothorax (PTX) - General Guidelines	
Upper 1/3 of thorax	***Trivial PTX*** - Expect Mild If Any Clinical Signs
Middle 1/3 of thorax	***Moderate PTX*** - Expect Increased Respiratory Effort
Lower 1/3 of thorax	***Severe PTX*** - Expect Overt Respiratory Distress
Not Found along thorax	***Indeterminate Study*** or Most Severe form of PTX - Look at Your Patient's Respiratory Effort!
Dr. Gregory Lisciandro, Hill Country Veterinary Specialists, FASTVet.com, Spicewood, Texas, Copyright 2019.	

Use of Vet BLUE® - Dry Lung, Wet Lung, Shred Sign, Tissue Sign, Nodule Sign and Wedge Sign

By using the Vet BLUE® regional, pattern-based approach, aspiration pneumonia, left-sided congestive heart failure/volume overload, pulmonary thromboembolism, and non-cardiogenic forms of lung edema may be rapidly sorted out point-of-care. *See the Vet BLUE® Proceedings.*

Global FAST® for Rapidly Detecting Treatable Forms of Shock - The Hs AND Ts of CPR

Knowing Your American Heart Association Hs and Ts for Rapidly Detecting Treatable Conditions during CPR or Imminent Cardiopulmonary Arrest (CPA). The veterinary profession should be well commended for standardizing CPR Guidelines through RECOVER. However, the reason why your patient is going to experience CPA, or why you are doing CPR in the first place has been overlooked. Global FAST® can rapidly detect treatable causes for imminent CPA and help rapidly detect treatable causes for CPR when minutes count and decisions need to be made. Global FAST® rapidly detects treatable conditions point-of-care easily missed or only suspected based

on traditional means of physical exam, laboratory testing, and radiography. The RUSH (Rapid Ultrasound in Shock) exam in human medicine was developed for these same reasons.

Global FAST® Rapidly Evaluates the Veterinary Hs & Ts of the American Heart Association (AHA) Guidelines for Treatable Causes of CPR.

The author has modified the AHA Hs and Ts. The Ts are ruled out as follows: **T**ension PTX by presence of A-lines without a lung sliding and the search for the Lung Point; **T**rauma Hemorrhage through the detection of free fluid in the intra-abdominal cavity, the retroperitoneal space, the pleural cavity and the pericardial sac, and the presence of B-lines during Vet BLUE® in trauma patients; **PTE** is diagnosed by the severe dilation of the RV during TFAST and the RV:LV Ratio, and/or the presence of the Wedge Sign in dorsal views during Vet BLUE®; and **T**amponade at the FAST DH View with or without additional PCS views; and **T**oxin-Anaphylaxis by the observation of the gallbladder halo sign, intramural edema causing sonographic striation of the gallbladder wall. However, gallbladder wall edema is not pathognomic for canine anaphylaxis. *A chart with the causes for gallbladder wall edema can be found in the Free Resources page of FASTVet.com*

Table: Use of Global FAST® for Rapidly Ruling Out your Veterinary Hs and Ts in patients nearing CPA and during CPR modified by the Gregory Lisciandro, DVM from AHA CPR Guidelines.

Knowing Your Veterinary Hs and Ts During Shock, Cardio-Pulmonary Arrest and Advanced Life Support and Using Global FAST® for Rapid Point-of-Care Detection	
The Hs Evaluated for Using Venous Blood Gas, Physical Exam, Vital Signs, and Global FAST®	**The Ts** Evaluated for Using Global FAST®
Hypothermia	**Tension PTX** (TFAST)
Hypotension (AFAST®, TFAST®, Vet BLUE®)	**Trauma, Hemorrhage** (AFAST®, TFAST®, Vet BLUE®)
Hyperkalemia, Hypokalemia	**Thromboembolism (PTE)** (TFAST® echo views, Vet BLUE®)
Hypoglycemia	**Tamponade, Pericardial Effusion** (TFAST®, AFAST®-TFAST® DH View)
Hydrogen Ion (Acidosis)	**Toxin, Anaphylaxis** (AFAST®-TFAST® DH View)
Hypertension, Pulmonary (TFAST® echo views, AFAST®-TFAST® DH View)	
Hypocontractility, DCM (TFAST® echo views, AFAST®-TFAST® DH View)	
Hypoventilation, Pleural Space Disease (TFAST®)	
Greg Lisciandro, DVM Dipl. ABVP, Dipl. ACVECC and FASTVet.com © 2015, 2016, 2017, 2018, 2019	

What the Global FAST® Approach has Over the RUSH Exam

- AFAST® Abdominal Fluid Scoring System
- AFAST® Target-Organ Approach
- AFAST® CC Urinary Bladder Volume Estimation Formula and Urine Output Overtime
- AFAST® for Pneumoperitoneum
- AFAST® for Gastrointestinal Motility
- TFAST® PTX 1/3s Rule for Semi-quantifying Degree of Pneumothorax (PTX) and for Monitoring PTX
- Vet BLUE® as Regional, Pattern-based Approach for Respiratory Conditions
- Vet BLUE® B-line Scoring System

Baseline Admission Global FAST® & Serial Exams are Key

The use of repeat Global FAST® exams cannot be overemphasized. Minimally a 4-hour post-admission exam should be performed (sooner in questionable or unstable patients), and the author incorporates Global FAST® as part of daily rounds immediately after a complete physical exam.

Summary of Global FAST® for Patient Volume Status & CPR & ALS

The use of the Global FAST® is an effective, point-of-care evaluation that is non-invasive and low risk for critical patients providing invaluable information for patient volume status during resuscitation and during advanced life support (ALS) post-CPR. Furthermore, Global FAST® should be used as standard of care for rapidly surveying for treatable and reversible causes of uncharacterized hypotension/shock and CPR as well as comoplications after return to spontaneous circulation (ROSC). By incorporating Global FAST®, many conditions missed by traditional training without ultrasound are detected cageside with low patient impact, and clinical course is modified and adjusted earlier in their course. As a result lives are saved, complications better avoided, and next best tests are better determined by "seeing" the problem list with evidence-based information.

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Working with Distraction

Laura Smallwood, DVM DACVIM (SAIM) RYT-200

From patient care to inter-personal relationships, the ability to be fully present for what is unfolding in our present moment awareness is key to responding in ways that lead to best outcomes. However, distraction—including the distraction of our own busy minds--frequently stands in the way of us attending to what is most important in any given moment and there is growing evidence that distraction is also a significant factor in stress and burnout.

In this workshop, participants will have any opportunity to explore practical strategies for working with different types of distraction in order to be more fully present for things that really matter. Topics to be explored include:

- The negative impact of distraction on patient care, interpersonal relationships, mental health, physical health, and the overall wellbeing of the workplace community.
- The neuroscience of distraction.
- The neuroscience of attention and how mindfulness practices can be utilized to strengthen the capacity to maintain focused attention in busy work environments.
- Ideas for identifying and reducing unnecessary distraction in the work environment.

Participant Notes

Mindfulness Defined

As defined by the founder of Mindfulness Based Stress Reduction (MBSR), the definition of mindfulness is "paying attention in a particular way: on purpose, in the present moment, and nonjudgmentally."

Paying attention: Scientists believe that the ability for humans to think and KNOW that they are thinking is an ability that separates humans from other species. The term for this is metacognition and metacognition is what allows us to recognize when we are lost in thought. The ability to be attentive is the foundational skill for awareness and focus and is what connects us to our present moment experience.

On purpose: Mindfulness is an intentional practice. We have the capacity to **choose** to be fully present and attentive to the present moment. If we don't make this choice, the brain will default to mind wandering.

In the present moment: The present moment is the only moment that matters. It is the only moment in which we can live our lives. It is the only moment in which we will find joy, connection, love, opportunity. Mindfulness expands our capacity to notice when the mind is wandering, to return our attention to the present moment, and to keep it there longer.

Without judgment: Mindfulness teaches us to be open to whatever experience the present moment brings. Rather than succumbing to the need to label everything as good/bad, pleasure/pain, we cultivate the capacity to bring some curiosity to what we are experiencing.

Mindfulness is a state of awareness that is cultivated through practices such as meditation. Mindfulness is the ability to intentionally bring our awareness out of distraction and into our present moment experience, doing so without judgment of ourselves, others, or the circumstances of the

situation. In cultivating this ability to be fully present, we gain greater awareness of when the mind is distracted and the ways in which distraction prevents clarity, connection, and compassion.

The Power of the Present Moment

The brain is hard-wired to think and, left to its own devices, the prefrontal cortex will automatically begin to drift away from the experience of the present moment and into thought. As a result, we spend much or our lives in a state of distraction—thinking about the past or the future—only to miss out on the present moment, the ONLY moment we have to live and the only moment we can affect. Mindfulness allows us to recognize when we are lost in thought (that recognition being a capacity referred to as metacognition) and intentionally bring our attention back to the present moment.

In 2010 researchers at Harvard University published a study that examined mind wandering. Their data showed that test subjects were not paying attention to what they were doing 46.9% of the time. Researchers also found a correlation between mind wandering and feelings of unhappiness, regardless of what activity the test subjects were engaged in. In other words, even when doing something that would be pleasurable, thinking about something else caused test subjects to experience feelings of unhappiness. This research suggests that for almost half of our waking hours, we are not fully present for what is happening in our lives. Imagine how many opportunities for joy, connection, and creativity we can miss in that amount of time! And not only that, these researchers concluded that "a wandering mind is an unhappy mind".

How does Mindfulness work?

Very simply mindfulness trains the mind to focus and refocus over and over again, freeing us from the random thinking patterns of our busy minds. Each time we bring the mind back to the present moment, we create new neural networks that are connected with **sustained attention.** This reorganization of neuronal networks and alteration of neuronal function within our nervous system over time is referred to as **neuroplasticity**. Like building muscle at the gym--when we use our minds in a particular way we physically change and rewire the brain over time. Each time the mind wanders and we bring it back to the present moment, we build the neural networks of attention and focus.

Through mindfulness practice we learn about where the mind wants to go and we can learn the pattern of our preoccupations. Every time we notice that we're thinking we have a moment of **metacognitive awareness**. Specifically, mindfulness as an expression of metacognition is:

The skill of **seeing** that the mind is not where you want it to be
The skill of **detaching** the mind from where you don't want it to be.
The skill of **placing** the mind where you want it to be.
The skill of **keeping** the mind where you want it to be.

How do we know Mindfulness works?

Since 1979 when Jon Kabat-Zinn recruited patients suffering from chronic pain to participate in the first 8-week stress reduction course that served as the prototype for what we now call Mindfulness Based Stress Reduction (MBSR), thousands of behavioral, medical, and neuroscientific studies have examined the effects of meditation on the brain. Demonstrated behavioral benefits include reduced psychological stress, increased empathy, and improved working memory capacity and attention. Studies have

demonstrated that meditation can decrease negative emotions; shift brain activity into the left prefrontal cortex—an area of the brain associated with happiness and optimism; and improve physical health.

The introduction of the functional MRI (fMRI) in 1990 has allowed neuroscience research to study the effects of meditation on the brain. fMRI studies have demonstrated that the activity and the size of the amygdala (the primitive part of the brain associated with fight or fight response) is decreased in meditators compared to non-meditators. A 2003 workplace study demonstrated increased activity in the left prefrontal cortex and the experience of feeling more energized, alert and joyful in workers who practiced meditation. There was also an improved response to flu vaccination in this group compared to non-meditators. Additional studies examining the impact of meditation on the immune system have demonstrated reduced cortisol levels, less decline in CD4+ T cells in HIV+ patients; and faster resolution of lesions is patients suffering from psoriasis.

A 2009 study published in the Journal of the American Medical Association demonstrated that training in mindfulness meditation reduced psychological distress and burnout in physicians and improved their well-being while also expanding their capacity to relate to patients which, in turn, resulted in enhanced care.

Distraction Due to Technology

Research abounds on the perilous impact of technology in society. Our attention spans are getting shorter, our long-term memory is degraded, and as a result, we are feeling less connected to one another. Emails, texts, social media--how often do these things divert our attention from attending to what really matters in the present moment? We now live in a state of what can be described as "continual partial attention"—a state where we are only slightly aware of what is happening around us and very much lost in thought. It has become increasingly difficult for many of us to maintain sustained attention.

Technology, in particular portable technology, is by design, both distracting and addictive. When we engage with these devices, we are typically swiping and scrolling—jumping from one thing to another rather than investing sustained attention on any one thing. This very act of jumping from one thing to another, trains the brain to be more easily distracted. To make this worse, the anticipation of that next thing—whether it is triggered by seeing an alert that may signal a Facebook like, something new on your Twitter feed, or even just seeing your device and wondering if there is anything new—results in the brain releasing dopamine. Dopamine is a reward, a really powerful reward, that reinforces whatever behavior preceded it because it makes us feel good. But it doesn't last very long so, as it wears off, we are compelled to seek out another hit. This dopamine hit reinforces the checking, swiping, scrolling behaviors associated with personal technology. It also reinforces the need to keep personal technology close at hand and feeds into the anxiety we often feel when separated from our devices.

Multi-tasking

In the workplace, we embrace the idea that multi-tasking is a valuable and effective work strategy. The fast pace of clinical practice in the technological age requires the juggling of multiple tasks (a multiple technology platforms) but we are mistaken in thinking that we can focus on multiple sources of

cognitive-rich input simultaneously. The truth is that we alternate between tasks, "juggling" them sequentially. Studies have shown that each time we switch tasks, the brain needs time to recover and, during the recovery period, we work less effectively and are more prone to mistakes.

Multi-tasking affect the brain somewhat like driving a standard transmission in stop and go traffic on the interstate affects the transmission. We have to downshift, stop, and go back through gears to get up to speed again. When we do this over and over in a car, it is hard on the transmission and horrible for gas mileage. When we do this with the brain, we diminish working memory and make mistakes. Like with car, the more often we stop and start whatever we are doing, the greater the cost. However, the perception that we are successfully doing two things at once causes the brain to release dopamine. So, this rapid serial tasking feels good so we keep doing it and, in so doing, train the brain for distraction rather than sustained attention.

Addicted to Distraction

Addiction is defined as continued use despite adverse consequences and, in his book *The Craving Mind*, Judson Brewer suggests that distraction can be classified as an addiction. Neuroscientific investigation has revealed a relationship between the dopaminergic response in the brain and a range of distractions from daydreaming to smartphone use—the same response that occurs with substance addiction. This dopamine response sets the stage for cravings that trigger behaviors related to distraction. We experience this when we see someone check their smartphone and then feel an overwhelming urge to check our own, or when we are triggered by a notification to check an email or text at the expense of being distracted from a conversation we are having.

Training the Mind

Mindfulness practices provide an opportunity to work with the mind in a way that strengthens the dorsolateral prefrontal cortex, an area of the brain associated with cognitive control while reducing the activity of the medial prefrontal cortex and posterior cingulate cortex, areas of the brain involved in the self-referential, impulsive reacting. When we practice being mindful—noticing when the mind is wandering and choosing to bring attention back our present moment experience—we are harnessing neuroplasticity to create and strengthen connections in the brain that lead to a greater capacity to focus and sustain attention skillfully.

It's important to note that the brain is always being trained and that we also unconsciously train the brain to be more distracted and less focused. Our smartphones and other devices are potent trainers as is the act of multi-tasking. Unconsciously, we spend much of our day engaged with activities that adversely affect focus and the capacity to maintain sustained attention. There are, however, strategies available to us that can reduce harmful training of the brain and foster positive training of the brain.

One of the most effective ways to reduce the harmful training is to pay attention to how we use technology starting with setting some limits on use. We might "use" at predetermined times or commit to "using" it less. We can also just bring some mindful attention to our use—observing our patterns, noticing how we really feel after a social media binge, for instance.

We can also pay attention to how we work. Rather than working under the delusion that multi-tasking is possible, a good start might be to call what we are doing "serial tasking". There can still be times when we need to switch back and forth between two things but we can pay attention to that—recognizing that like the person driving the standard transmission car in stop and go traffic—there is a cognitive cost to this and that it pays to keep this to a minimum.

Finally, we can commit to practices that strengthen the neural networks of focus and attention like meditation. Meditation provides an antidote to the inevitable brain training that results from all the distractions embedded in our workplace and home environments. I think of the impact of distraction on my brain somewhat like the impact that lying on the couch watching Netflix and eating chocolate chip cookie dough would have on my body and meditation like the work-out I might do the next day. It is still important to have some self-restraint but cookie dough and distraction happen so work-outs and meditation are a good idea.

Mindful Practices

There are two ways to practice mindfulness. The first is formal practice. Formal practices commonly taught in the context of mindfulness include meditation (which can be practiced seated, standing, or walking), body scan, and mindful yoga. All three of these practices are considered meditation practices and all three serve to cultivate awareness of the body, awareness of thinking and thoughts, awareness of the emotions and feeling tone, and an increased capacity to sustain focus and attention.

Informal practices refer to utilizing daily activities (i.e., brushing the teeth, preparing food, driving, taking a walk) as opportunities to bring our full attention to what we are doing, noticing when the mind is wandering, and coming back to the task at hand. Informal practice are really helpful because they offer many opportunities to practice being mindful in the context of normal day to day activities.

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Triage, Life Saving Priorities

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette, Indiana

In general, most veterinary practices have emergencies that walk through the door that they were not expecting. Everyone has heard the word "Triage", but do you really know what it means? Triage comes from the French word "trier" or "to sort".[1,2] The word was generated around the 1790's during the French revolutionary war/Napoleonic war time when men had to care for the wounded on the field. Obviously, battlefield triage is very different than hospital triage. You can only address those that can be helped.

The basis of triage is to identify the cases that need immediate care to maximize the survival of the patients that are presented to the hospital. You will have to identify the patients that are stable enough to wait and the patients that are critically ill. It is crucial to also identify those patients that are stable, but shouldn't wait in the lobby. For example, a patient that has a small laceration but is dripping blood all over the lobby.

In order to triage effectively, walk around the clinic to ensure there is an area where you can take critically ill patients and tend to them. Be prepared.[1]Where do you triage? The lobby? Is there an exam room that you can take them to? An area of the lobby? What about cats and exotics? Do you even see exotics? Do you know where the nearest exotic clinic is? Find the best place that will work for both the clinic and the patient.

It is always beneficial for the patient and the staff if there is an area of the treatment floor that is designated for a critical/emergency patient. Have catheters, fluids, oxygen administration, a crash cart/tray, blood pressure supplies, warming devices, monitoring devices (ECG), and quick diagnostic tools (microhematocrit tubes, glucometer, lactometer, I-stat™, etc.) ready to go. Having this equipment at hand can help a clinician quickly diagnose a critical patient and create a better emergency experience for both the staff and the patient. A crash cart/box/tray is the most essential tool to have at the practice. It should include catheter supplies, emergency medications (lidocaine, epinephrine, atropine, vasopressin, etc), syringes for drawing blood and emergency medications, endotracheal tubes, fluids, fluid lines, etc. Make sure the crash cart is stocked every day. Supplies are borrowed, taken, or used for other situations which can then in turn make a critical situation more complicated. Nobody wants to be running to the surgery suite for endotracheal tubes when a patient has to be intubated. Mark the crash cart to signify that it is stocked. The last thing that anyone can do to be prepared for an emergency is to make sure that everyone practices. Practice, practice, practice. In an emergency situation, the staff will make a more cohesive team if they all know where the supplies are kept, where the crash cart is, and how to stay calm.

Triage can be done in two ways. Triage can be done in person when multiple emergency patients come in at the same time or triage can happen over the phone. Receptionists are the first to see the patient when they walk into the door. It is strongly suggested to train the receptionists what is an emergency and what isn't an emergency. They are the ones to determine when to call a technician for help. When a client calls the clinic for an emergency, it is better to have medical personnel on the phone to help ask the correct questions and make sure that the emergency is a true emergency. For example, a client calls and says her cat is in the litter box all the time and thinks he is constipated. A veterinary professional may ask, "Has your pet been urinating in the box as well?" When the client says, "Well, now that you mention it, I don't remember seeing urine in the box either." If this cat is blocked, it would be considered a true emergency whereas a non-veterinary professional may have told the client to come in as an appointment the next day for constipation. DO NOT DIAGNOSE OVER THE PHONE! In the phone call, it may have been easier to say, "I know your cat is blocked, please bring it in." But unfortunately, this is illegal for technicians and

receptionists to do. State what you are concerned about to the client, “I am concerned that your cat hasn’t urinated in a long time and I would suggest that you bring him in immediately.” If the client is asking multiple questions over the phone or is getting emotional, express concern for the patient. Let them know you will answer all of their questions when they get in. Keep control of every conversation. Be aware that if the client thinks they have an emergency, then you should treat it as such.[3]

Know your clinic’s limits when it comes to emergency. Can the hospital care for a patient for 24 hours? Are you open on the weekend? Is there an overnight technician that can care for patients? Can you handle wildlife/exotic emergencies? Can the hospital perform surgery at any point in time? These are all questions to ask the clinic and the staff to help ensure preparedness for an emergency you cannot handle. It is acceptable to send a client to a different clinic if the hospital does not have the capability to handle different types of emergencies.

Always assess the most critical patient first. Remember your ABC’s (airway, breathing, circulation) and infectious/dramatic cases can be brought into the treatment room and placed in a kennel while the technician is getting a more accurate history. If the patient is very critical and needs to be brought back right away, the next step is to have a triage estimate ready.[1] This will ensure not only that the patient will be taken care of quickly, but that the owner is prepared for what the cost may be. This estimate usually is a range that can include radiographs, blood tests, intravenous catheter placement, and intravenous fluid administration. This does not include any other tests or medications that may be done after the diagnosis is made. The form also includes a CPR code. It is better to be ready for CPR than to ask the owner as CPR needs to be performed. The receptionist can go over this form if necessary. Everything should be written in a clear format so everyone understands what is included in the estimate. Communicate to the client that this is a way for their pet to get the quickest, best care possible.

Client communication is key in an emergency situation. Explain to the client why and where you are taking their pet. This can be distressing to them because they cannot be with their “family member”. Keep control of a resistant client. Focus on the patient and assure them that the staff is doing what is best for their pet. Keep updating the client frequently. Any medical or financial decisions should be made with the client. The receptionists should also always remind the other clients waiting the in the lobby that it is better to not be first in the ER.

A history should be taken quickly once the emergency patient walks in. This includes the presenting complaint, when the patient was normal last, what has been done/given already, have there been any previous medical issues, and is the patient receiving any medications or is allergic to any medications. A more thorough history can be taken after the patient is stabilized. This should only take less than five minutes!

Triaging should be prioritized in order of, respiratory compromise, cardiovascular compromise, neurologic compromise, and then other emergencies. Assess each patient’s “ABC’s”. A=Airway, B=Breathing, C=Circulation, D=disability/neuro, E= external assessment.[2,3] After assessing each, a temperature, pulse, and respiratory rate must be done to complete the primary assessment.[2] DO NOT FORGET ABOUT PAIN MANAGEMENT! Assess the patient’s airway first. Keep the patient calm, cool, and supplement oxygen if needed. Supplementing oxygen is never the wrong thing to do. Intubate the patient if it is warranted. A tracheostomy tube may be needed if an endotracheal tube is impossible. A tracheostomy tube can be made out of an endotracheal tube if the hospital does not have tracheostomy tubes available. Next, assess the patients breathing. Ascult the patient’s lungs. Are there any crackles, wheezes, harsh lung sounds, no sounds? A pulse oximeter can tell you the oxygen status of a patient. It is never good if a patient is cyanotic. Place on oxygen and minimize stress. Pink gums do not necessarily mean that the patient is stable. A patient can still have low oxygen saturation with pink gums. There are many ways oxygen can be supplemented to a patient.

A few ways are: oxygen cage, incubator, e-collar with plastic wrap on it, a cat carrier with a plastic bag over it, a mask, nasal cannulas, etc. Next, assess the patient's circulation. Start by assessing the patient's mucous membrane color, capillary refill time, pulse quality, level of consciousness, heart rate, and extremity temperature. If the blood pressure is normal, this does not mean that the patient is stable, but if the patient's blood pressure is low, this is an indicator of shock. Shock is a physical exam diagnosis. If the patient is externally hemorrhaging, stop the bleeding. Place an intravenous catheter to give fluids to replace the volume lost. If the patient is in cardiogenic shock, fluids may be contraindicated. ECG, blood pressure, bloodwork, stat chemistry values, "Big 4" (PCV/TP, BG, Lactate), should be assessed. If imaging is deemed necessary, the patient must be stable. No patient should die on the radiology table! Use sedation if needed. Remember a patient should stay calm.

If a neurology emergency comes into the clinic, assess the patient's level of consciousness. This can determine if you bring the patient to the treatment room, or place the patient in the room to be looked at by the clinician next. Ask yourself, is the patient able to walk? Did they have some sort of trauma? Are they seizing?

Lastly, perform an external assessment.[2] Look over the entire patient and check every side. Attend to any wounds, lacerations, punctures, or abrasions. Assess for any crepitus, fractures, or pain in the abdomen. Are there any skin issue? These are all things to look for when assessing the whole patient.

Infectious diseases are always something to keep in mind while triaging patients. Where would you triage an urgent infectious patient? Is the patient going to transfer that infectious disease to you? To other patients?

During this time, owners should always be on the mind of every staff member that is working with their pet. The staff should be triaging the owner as much as they are triaging the patient. Keep the owner calm, cool and informed. The more informed the owner is, the more they will feel comfortable that the staff and veterinarian are in control of the pet's health.

A secondary assessment should be done after the patient is stable.[1,2] A full physical exam, bloodwork results, imaging interpretation, and repeated ABC assessments are performed. Repeating and reassessing the ABC's are crucial because these may change quickly in any patient. Shock may reoccur, pain may surface, or other symptoms may come up. Keep the patient clean, dry, and comfortable. Change bandages and splints as needed. Always keep an eye on the patient's neuro status, pain, anxiety level, urine output, and hydration status.

In conclusion, triaging is important in any hospital setting. If prepared, emergency situations can run smoother with the hospital staff. Remember to assess the ABC's and always communicate effectively with the client. This can save your patients.

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Environmental Emergencies in Veterinary Medicine

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette, Indiana

Environmental Emergencies happen every day in the veterinary practice setting. Depending on the area in which you live, there are different environmental emergencies that may occur. Some examples of these emergencies are heat stroke, near drowning, spider bites, insect bites, snake bites, toxins, hypothermia, or smoke inhalation, just to name a few.
In this lecture, we will go through what each emergency is defined as, the pathophysiology, and how to treat the emergency.

Heat Stroke

Heat stroke is defined as a condition where the patient has either exercised too much and is overheating or the patient was in a hot environment for a prolonged period of time and could not regulate their core body temperature.

The pathophysiology of heat stroke can get a little complicated. As veterinary professionals, we must not confuse heat stroke with a true fever. Heat stroke happens when a patient cannot dissipate its own body heat anymore due to overexertion or exposure to intense heat for an extended period of time. Overweight patients, brachiocephalic breeds, patients with upper airway problems, patients with prolonged seizures, and certain breeds like Labradors, can be more likely to get heatstroke. The hypothalamus is responsible for temperature regulation of the body. Once a patient's core body temperature gets above 105 degrees Fahrenheit, the patient's central nervous system/hypothalamus can start to function irregularly. This can affect how the patient can regulate its own body temperature.
70% of a patient's heat loss occurs by radiation and convection via skin. When a patient gets this hot, cardiac output increases and peripheral vasodilation occurs. The body is trying to move heat to the surface of the skin to attempt to dissipate it. Panting will also occur because the body is attempting to evaporate the heat through the respiratory tract. As a result, the gastrointestinal tract is the first organ to experience decreased blood supply. This will then cause diarrhea. Dehydration also occurs as the patient is losing body water through panting and excessive salivation. The inflammatory cascade then activates, along with hemostatic processes. This can cause Systemic Inflammatory Response Syndrome (SIRS), which then progresses to Multiple organ dysfunction syndrome (MODS). The combination of dehydration and possible sepsis can lead to dissemination intravascular coagulopathy (DIC). Patients that present with severe heatstroke have a grave prognosis if their body temperature exceeds 106 degrees over an extended period of time.

When a patient arrives with heatstroke, it is important to get their core body temperature down in a slow, controlled manner. If the body cools down too quickly, this can result in cerebral edema. Cerebral edema can cause the patient to not raise internal body temperate back to normal. Intravenous fluids, at room temperature, should be started immediately. Room temperature water with a fan blowing on the patient is an effective way to cool the patient and bring internal temperature down. Putting alcohol on the patient's paw pads, or even their whole body, is not an effective way to cool down the patient. Alcohol and ice baths can cause vasoconstriction and move all the body heat back to the core, which in turn,

will not cool the patient down and can cause further complications. Cool water enemas, cool gastric lavage, and cool peritoneal lavage can also be somewhat effective in cooling a patient down from extreme heatstroke. Gastric lavage does run the risk of the patient developing aspiration pneumonia. Shaving a very long-haired patient could also be of benefit. Active cooling should stop when a patient's rectal temperature becomes 103 degrees.

Hypothermia

Hypothermia is defined when a patient's body temperature drops below 99 degrees. This can be primary hypothermia, also called accidental, or secondary hypothermia, caused by a systemic disease process.

The Hypothalamus is the thermoregulator organ in the body. There are also thermoreceptors that are present in the skin, spinal cord, abdominal viscera, and veins. Hypothermia can cause multiple effects in the body. Cooler body temperatures can thicken blood viscosity, decrease cardiac output, cause bradycardia, cause coagulopathies, decrease respiratory rate, impair wound healing, or decrease metabolism. Patients that are thin, exposed to extreme cold, or under anesthesia for an extended period of time, are more likely to become hypothermic. When the cold receptors are activated in cold environments, vasoconstriction and piloerection occurs in order to help minimize heat loss. If this doesn't work to keep the body warm, the body tries to warm itself by shivering. Shivering is increased skeletal muscle activity, which in turn, increases body temperature.

Treatment of hypothermia depends on how severe the hypothermia is and what caused the hypothermia in the first place. There are three types of rewarming: active rewarming, external passive rewarming, and active external rewarming. Active rewarming includes warming the main core of the body. This can include warmed intravenous fluids, warm water enemas, warm peritoneal lavage with isotonic crystalloid fluids, warm urinary bladder lavage, or warm humidified inhaled air. External Passive rewarming is the use of blankets and the patient's own body warming techniques to help increase core body temperature. Active external rewarming is the use of warm water blankets, warm water bottles, and warm forced air. Intravenous fluids should be initiated once a patient is admitted with hypothermia. Dehydration can occur during a hypothermic event.

Drowning

Drowning is defined when a liquid is forced into the lungs, causing respiratory impairment.

Drowning occurs most often when a patient is submerged in water and is unable to get to the surface for air. Patients will then aspirate the water and then experience hypoxemia. According to Silverstein and Hopper, patients that survive have aspirated less than 22mls/kg of water. Depending on the type of water or fluid aspirated, electrolyte imbalances can occur. Saltwater aspiration is usually more severe. The more hypertonic fluid pulls water from the circulation into the alveoli, which can reduce lung compliance. Drowning can result in cerebral hypoxia and eventually death.

Treatment for drowning victims can be a multimodal approach. A catheter and intravenous fluids should be initiated immediately. Intravenous fluids can help with the perfusion of the organs as well as keeping the cardiovascular system stable. Oxygen should also

be administered as quickly as possible. Acid/base status should be assessed and an arterial blood gas should be performed. Blood pressure should also be performed. Cerebral edema can occur in drowning patients. It is crucial to evaluate blood pressure and heart rate for Cushing's reflex. Mannitol can be given to reduce cerebral edema. Mechanical ventilation may be appropriate for these patients if their blood gas values indicate it. Prognosis is based upon how long the patient was under the water and how much liquid it has aspirated.

Spider Bites

There are multiple different venomous spiders found in the United States. Two of the most common are the Brown Recluse and the Black Widow. Unfortunately, spider bites are most often diagnosed based on signs alone, unless the spider was actively seen biting the patient. Black Widow Spider bites present clinical signs including restlessness, tachypnea, heightened anxiety, painful muscle rigidity, and possible shock. Brown Recluse spider bites present clinical signs of a bullseye-type lesion on the body, pain at the site, or a necrotic area where the spider bite occurred. Systemic signs may occur that would include thrombocytopenia, hemolysis, and disseminated intravascular coagulopathy. If systemic signs do occur, the patient may present with fever, vomiting, hemoglobinuria, renal failure, and septic shock.

Black widow spiders carry a neurotoxin called alpha-latrotoxin. Alpha-latrotoxin releases neurotransmitters from the terminals. This process depletes the amount of synaptic vesicle contents which then blocks neurons from firing appropriately. Dopamine, Acetylcholine, noradrenaline, and glutamate systems are all sensitive to the toxin and will not function appropriately.

Brown Recluse spider venom has enzymes, like Sphingomyelinase D, that are known to cause hemolysis. Other enzymes are present in venom that degrade fibrinogen, collagen, fibroectin, gelatin, elastin, and basement membranes of the cell. These enzymes can cause local tissue necrosis and eventually systemic signs.

Treatment of the Black Widow spider bite can be complicated. Antivenin is made commercially in the lyophilized form. This means it is inexpensive and has a long shelf life. Antivenin should be given as soon as possible when a patient presents with a Black Widow spider bite. Anaphylaxis can occur so antivenin should be administered slowly. Diphenydramine can be a pre-treatment before antivenin administration. Theories exist whether calcium gluconate can help with the treatment of muscle spasms and decrease the pain of spider envenomation.

Treatment of a Brown Recluse spider bite should include an ice pack to the area of the bite. Some studies have shown that Sphingomyelinase D is temperature sensitive and ice will help limit tissue necrosis. Keep the area clean and dry to prevent infection. Steroids can help decrease hemolysis. Pain medication should be used as needed. Once systemic signs occur, treatment of those individual signs should be initiated.

Snake Bites

There are two families of venomous snakes in North America: the Elapidae (coral snakes), and the Crotalida (pit vipers or rattlesnakes, copperheads, or cottonmouth moccasins).

Coral snake envenomation signs include neurotoxic effects. The enzymes in snake venom are responsible for immobilization of their prey. It blocks the synaptic transmission in the neuron at the acetylcholine receptor site which can cause skeletal muscle paralysis and respiratory paralysis. Cardiac arrhythmias have also been seen but are not common.

Crotalid snakes have the ability to control the amount of venom they choose to deliver. 25% of Crotalid bites are “dry” bites, meaning that no venom was delivered. Crotalid bites are the more severe of the two types of snake bites. Puncture wounds at the site of the bite are usually oozing. If swelling is present an hour after the bite, there is a good chance that the patient experienced a venomous bite instead of a “dry” bite. Tissue damage does occur at the site of the bite. The protein venom metalloproteinases, or VMPs, are what causes the tissue damage and inflammation. VMPs then activate pro-tumor necrosis factor, TNF alpha, which results in further tissue breakdown and damage. Some rattlesnakes also have a neurotoxin associated with their venom. This neurotoxin blocks the calcium channels which then prevents the release of acetylcholine, thus preventing the activation of acetylcholine receptors. This prevents muscle contraction. Some other clinical signs that can be seen with snake envenomation are vomiting, diarrhea, incontinence of the urinary bladder, and lethargy. Rattlesnake venom also contains kininogenases. These kininogenases act on plasma globulins and form bradykinins. This process vasodilates vessels and causes hypotension. Swelling and edema can form at the location of the bite. Lymphadenopathy can also occur because the venom can travel via the lymphatic system.

Treatment for Coral snake envenomation is mainly supportive care. This care involves Intravenous fluids, pain medication if needed, care for a possible paralyzed patient, and measures to prevent aspiration pneumonia.

Treatment for Crotalid snake bites is dependent whether the veterinary clinic has antivenom. Antivenom comes in two types: Antivenin Crotalidae Polyvalent (ACP) and Crotalidae Polyvalent Immune Fab (CroFab). Anaphylaxis can occur while giving antivenom however, it is rare. These patients should be hospitalized until symptoms resolve. NSAIDs and Steroids are contraindicated in these patients.

Insect Bites

Not every one of our patients are hypersensitive to insect bites. Some insects that may pose more of a problem for patients are bees, wasps, hornets, and some types of ants. These bites can cause an anaphylactic reaction.

There are two types of anaphylactic reactions: the classic pathway and the alternative pathway. In the classic pathway, patients are usually exposed to the sensitivity agent for the second time. IgE is then produced and then binds to mast cells and basophils. Cross-linking of IgE occurs and the cell releases histamine, heparin, tryptase, and a few other mediators. In the alternative pathway, platelet-activating factor (PAF) is responsible for the degranulation of the cell, not histamine. Degranulation of the cell occurs and interactions between mediators and organs can cause the clinical signs that include erythema, pruritus, and urticaria. Some reactions can include vomiting, diarrhea, tachycardia, respiratory distress, hypotension, shock, and death.

Treatment of anaphylaxis is based on clinical signs and how severe the anaphylactic reaction is. In less severe reactions, antihistamines alone could treat the anaphylactic reaction.

In more severe reactions, epinephrine, glucocorticoids, bronchodilators, vasopressors, and possibly anticholinergics may be used. Epinephrine is considered the number one treatment for anaphylaxis. It can cause α-adrenergic effects, β_1-adrenergic effects, and β_2-adrenergic effects. Fluid therapy should be used in patients with hypotension caused by vessels that are vasodilated and "leaky" due to the cytokine and histamine release. In very severe reactions, vasopressors and anticholinergics should be used if epinephrine and fluids are not enough to improve the hypotension. Prognosis of these patients depends on how severe the allergic reaction is.

Smoke Inhalation

Smoke inhalation injury usually occurs when a patient is in a house fire or some type of fire situation.

Inhaling smoke can be very detrimental to mucous membranes. It can irritate and burn those membranes. This also depends on what is burning in the house. These patients can have internal chemical burns due to the toxic gasses that are released. The two gasses that are common in house fires are carbon monoxide and hydrogen cyanide. Carbon monoxide can go undetected because it is odorless and colorless. Carbon monoxide binds to hemoglobin but takes oxygen's place on the hemoglobin. When carbon monoxide is present on the hemoglobin, oxygen cannot be perfused to the other organs of the body, resulting in tissue hypoxia. If a pulse oximeter reading on these patients is taken, it may result in a 100% reading. This is because 100% of the hemoglobin receptors are taken. It doesn't matter if it is oxygen or carbon monoxide. Hydrogen cyanide disturbs the oxidative phosphorylation process, leading to decreased ATP production and increased lactic acid production. Patients that had increased exposure to hydrogen cyanide can present with vomiting, tachycardia, arrhythmias and neurologic dysfunction. These patients may also present with ocular problems such as ulcers and thermal burns.

Treatment of these patients should immediately include an intravenous catheter and fluid therapy combined with oxygen therapy. Bronchodilators can also be used to decrease bronchospasm that may be due to irritation. The patient should also be treated for any thermal injuries the fire may have caused. It is imperative that the entire patient, not excluding the bottoms of paw pads, eyes, ears, and inside the mouth, be checked. Smoke inhilation patient's prognosis is all dependent on how long the patient was in the fire and how much smoke they did inhale.

Common Toxins

There are so many toxins that can affect our patients. In this lecture, I am only going to go through a few. These will include chocolate, grapes/raisins,

Food:

Chocolate: Clinical signs are caused by theobromide and caffeine and include vomiting and diarrhea, tachycardia, muscle tremors, seizures, coma and death. Decontamination and administrating activated charcoal are first steps, and IV fluid administration if clinical signs warrant. Medications like propranolol to decrease heart rate may be necessary.

- Milk Chocolate: wt (#) x 0.3 = oz needed for reaction
- Dark Chocolate: wt (#) x 0.12 = oz needed for reaction

- Baking Chocolate: wt (#) x 0.04 = oz needed for reaction

Grapes/Raisins: Unfortunately, the mechanism of action and toxic agent is unknown. These can cause gastrointestinal irritation as well as renal toxicity. The majority of dogs show clinical signs of vomiting within 24 hours of ingestion. Renal bloodwork changes occur within 24 hours of ingestion, and declines around 48-72 hours. Decreased urine output and lethargy can occur after 5 days. When a patient first presents with grape toxicity, vomiting can be induced. Activated charcoal can be given, but there is limited evidence on if it actually is effective. IV fluids help preserve the kidneys. Chemistry values should be monitored. If AKI persists, dialysis could be considered.

Xylitol: Xylitol is a sugar substitute showing up in multiple products and people are using it more in baking and cooking over sugar. Xylitol can cause an insulin release in dogs leading to hypoglycemia. Patients can come in Higher doses can lead to hepatic failure. The effect of Xylitol on cats is unknown. When patients come in that have ingested xylitol, vomiting may or may not be effective depending on when they ingested the toxin. Xylitol is absorbed into the body very quickly. Intravenous Dextrose, even in asymptomatic patients, can ensure that patients will not become hypoglycemic. Liver and GI protectants should also be used.

Medications:

Acetaminophen: Cats have an increased sensitivity to acetaminophen. The metabolism of the drug in both dogs and cats can lead to hepatic failure. GI signs occur first followed by facial edema and cyanosis. Acetaminophen also causes methemoglobinemia (hemoglobin is damaged and hangs on to oxygen instead of releasing it to tissues) which is characterized by brown mucus membranes and chocolate brown blood. Hypoxia occurs in these patients. Heinz bodies can form on red blood cells causing them to be destroyed, and anemia can also occur. Overdose is treated first with decontamination and charcoal. The treatment for acetaminophen is N-Acetylcysteine (140mg/kg loading dose, 70mg/kg q6h for 7 treatments) and can be administered either IV (filter recommended) or PO. N-Acetlycysteine should be diluted to a 5% solution before patient administration. Treatment is rounded out with vitamin C (thought to reduce methemoglobinemia), GI protectants, oxygen and supportive care.

- Cats: 5-10mg/kg toxic dose
- Dogs: >50mg/kg toxic dose
-

NSAIDS: Clinical signs often include, vomiting, and diarrhea and can lead to GI ulceration. At higher doses, renal toxicity can occur. In both cases, decontamination, activated charcoal and Gi protectants are recommended. Treatment also includes IV fluids and possibly Misoprostol to protect against GI ulceration. Chronic use of NSAIDS in patients can result in liver damage.

Rodenticides:

There are multiple different types of rodenticides so it is essential to know what type of rodenticide that the pet has ingested. Always encourage the client to bring in the packaging that the rodenticide came in. Color and consistency of the rodenticide cannot always used because most rodenticides come in a green color and have the same consistency. The three major types of rodenticides are anticoagulant rodenticides, cholecalciferol rodenticides, and bromethalin rodenticides.

- Anticoagulant rodenticides:

  Anticoagulant rodenticides inhibit the activity of vitamin K. Vitamin K is required to fully activate coagulation factors II, VII, IX, and X. When a patient cannot activate coagulation factors, clinical signs of bleeding are observed. Patients come in with a history of possibly coughing up blood, having nasal bleeding, or lacerations that will not stop bleeding. Other clinical signs can include lethargy, anorexia, weakness, and shock. PT/PTT should be tested when anticoagulant rodenticide is expected. PT/PTT will be elevated. Vitamin K is the typical treatment for anticoagulant rodenticide. If bleeding is already occurring, blood products should be used. Vitamin K is a fat-soluble vitamin, so it is best absorbed orally with food. If the patient is not eating, it can be given subcutaneously.
- Cholecalciferol rodenticides:

  Cholecalciferol is Vitamin D3 and ingestion results in hypercalcemia and hyperphosphatemia. This will lead to PU/PD, GI signs, muscle weakness and renal failure. If the patient is seen quickly, emesis and repeated doses of activated charcoal should be used. IV Sodium Chloride should be given for a few days. This reduces calcium reabsorption in the renal tubules and enhances urinary calcium excretion.
- Bromethalin rodenticides:

  Bromethalin is metabolized by the liver to a more toxic product desmethyl bromethalin. Both products are lipid soluble so the bromethalin accumulates in the brain and fat. Brain edema and lipid peroxidation occur and can cause cellular damage and necrosis. Patients may present with ataxia, hind limb paresis, paralysis, seizures, tremors, hyperexcitability hyperthermia, circling, CNS depression and death. Stimulating Emesis and activated charcoal are best for decontamination. If the patient is already neurologic, emesis would not be effective. Once the patient develops clinical signs, prognosis is guarded to grave. Lipid emulsion therapy early in the course of toxicity has been proposed as a possible “antidote” for this toxin.

Pesticides/Antiparasitics:

- Permethrins

  These are generally used as topical medications for flea repellants. When a patient comes in with pyrethrin toxicity, the first treatment should be to bathe them with a dish soap to cut through the medication to remove it. Muscle relaxants can assist with decreasing the muscle tremors that a patient usually presents with. IV lipid emulsion has been used and seems to help with the muscle tremors of patients. There is limited studies on the use of lipids with pyrethrins. The most common pyrethroid that the ASPCA poison control center received questions about from Florida was Etofenprox.
- Ivermectin

  Ivermectin is another antiparasitic that is used for endo and ecto parasites in dogs and cats. It is available over-the counter in oral and injectable form. Patients that experience ivermectin toxicity usually present with ataxia, paralysis, bradycardia, blindness, coma, or are dead upon arrival. Ivermectin, in high doses, crosses the blood brain barrier and enters the CNS and prevents neuron depolarization. Treatment consists of decontamination, multiple doses of activated charcoal, IV lipids can also be used since

ivermectin is lipid soluble. Do not use benzodiazepines to treat seizures as this may not help the seizures.

Plants:

- Sago Palm are found usually in tropical areas. All parts of this plant are toxic, and we mostly see feline patients for this toxicity. Cycasin is the most toxic element of the plant. Cycasin can cause hepatotoxicity and is also a GI irritant. It can also cause cerebellar necrosis which can cause ataxia. These patients will come in with vomiting, diarrhea, melena, icterus, ataxia, and possibly seizures and death. There is no antidote, so emesis, activated charcoal, IV fluids, and GI protectants will be needed. Sometimes, even with treatment, the patient may need long life hepatic support.
- Lilies are found in many gardens and especially in many bouquets around Easter time. All parts of the lily are toxic. If a patient even just nibbles on a leaf of a lily, it can cause toxic effects. These patients will present with vomiting, depression, and eventually renal failure. If patients are exposed for an extended period of time, they can present with CNS signs can occur which include ataxia, head pressing, and seizures. Treatment includes emesis, if possible, IV fluids, activated charcoal, and hemodialysis. Kidney values should be monitored daily for 48 to 72 hours.
- Marijuana is starting to become legal in more states and comes in many forms. Pets are exposed via ingestion and second hand smoke. The active ingredient in marijuana is tetrahydrocannabinol (THC). THC is a depressant that interacts with many neurotransmitters. Patients will present to the hospital with mental depression, hyperesthesia, ataxia, tremors, mydriasis or miosis, hypothermia, bradycardia, and respiratory depression. Patients will also be known to “dribble urine” and act like they are falling asleep standing up. With more marijuana being laced with more potent medications, you can often see patients coming in with more profound clinical signs leading to coma and possibly death. If a patient comes in quickly after ingestions, emesis can be initiated. Do not induce vomiting if neurologic signs are already present. Activated charcoal can be given with repeated doses. Treatment after decontamination consists of IV fluids and supportive care. THC is a lipid soluble toxin, so IV lipids can be used to possibly speed up recovery.

Critical Thinking Skills

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette, IN

Every good veterinary technician needs to have some critical thinking skills in order to properly care for their patients. Critical thinking is defined as the ability to make a decision on the basis of thorough consideration of data discovered through investigation, analysis, and evaluation. Critical thinking is not something that everyone is born with. It is a skill that must be learned, practiced, and implemented in every day practice. When working with patients on the floor they never "follow the book".

Most veterinary technicians love the technical aspect of the job. Placing IV catheters, drawing blood, placing central lines, and taking radiographs are "fun" but few technicians want to put the work in to master physiology, pharmacology, understanding diseases, and watching for subtle changes in any patient. These skills set good veterinary technicians from amazing veterinary technicians.

Nursing requires critical thinking all the time. Every veterinary technician must understand the equipment, working parts of catheters, central lines, and how to administer medications and what they react with. Nursing also requires interpersonal skills and their development as one interacts not only with fellow nurses and technicians but also veterinarians, assistants, client services, and especially pet owners.

There are multiple parts to critical thinking. These parts consist of: gathering of information or assessing a situation, focus, remembering, organizing, analyzing, generating, integrating, and evaluating.

- Assessing a situation is simply data collection. Data can be gained from many sources throughout the hospital. A veterinary technician must perform a physical exam on each patient at the beginning of their shift to gain more information about that patient.
- Focus on the task at hand and remember all the facts about that patient. Try not to get two patients confused.
- Organize the information that you have just received and analyze that data. Ask yourself questions about the treatment of that patient.
- Generate and integrate some thoughts about the treatment of that patient. What nursing techniques can you do to benefit that patient's care?
- Evaluate what nursing techniques worked, and what did not. Is that patient improving? The nursing team is critical to all aspects of patient care and the nursing process must reflect this.

The more you go through this process, the easier it gets. Each case is an opportunity to learn. Encourage your team to go through physiology and pharmacology while rounding, then everyone can be on the same page when it comes to that patient.

In order to encourage the team to think critically, one must understand the problem and how to solve it. Questions must be encouraged. NO QUESTION IS A DUMB QUESTION! Encourage a learning environment with your team, and do not single out or embarrass a single team member. As employees grow accustomed to these question and answer sessions, they will soon see them not as punishment, but look forward to the opportunity to learn and grow in their job.

A good way to help staff critically think, is with rounds. Ask questions during rounds and if rounds are not occurring, then make hypothetical cases during down time. Some examples could be: What is happening in that patient that requires this medication? Why are they doing this treatment now? How does passive-range-of-motion help this patient? Should we keep blood sampling on this patient? What reacts with this medication? Most staff will be comfortable with what needs to be done for this patient, but few will understand why.

No patient is going to follow the book. I encourage all veterinary technicians to ask “Why”. Medicine is complex and using critical thinking skills can help veterinary technicians understand what to do with that critical case while working with the clinician.

Anticipating what the clinician and patient may need is crucial to make a great veterinary technician. Anticipate results of vital signs every time treatments are performed on a patient. Troubleshooting why a result is different than 2 hours before is crucial to the well-being of that patient. If their blood pressure is elevated, find out why. Is it a different size blood pressure cuff? Do they need to go outside to urinate? Are they painful? Are you taking a blood pressure on a different leg? Utilizing your critical thinking skills is important so every patient has the best chance of getting out of the ICU. Writing down numbers on a treatment sheet is not helping anyone, especially the patient.

As a technician, you may not have the authority to change orders, add medications, or make a diagnosis, but those limits do not mean that you should not educate yourself in all of those areas. When medical orders are made, ask yourself why? Why are we using this antibiotic over that one? Why is this patient having an arrhythmia now? Why is the blood pressure dropping in this situation and can I do anything about it? Why are we giving a fluid bolus now? As you learn more you will be better about anticipating these changes in the next patient that you treat and you will be prepared. When the doctor orders that fluid bolus you will be ready. You will know that the blood pressure is dropping and be ready with the treatment.

The most important monitoring tool in the hospital is a veterinary technician. You can have the most fancy, expensive equipment, but having a veterinary technician to OBSERVE the patient is the most important. You, the technician, can anticipate what is coming next. A monitor can only tell you what is happening right now. As you progress in your career, remember what has happened in the past. Collect anesthesia records and case reports of interesting diseases and experiences to help you remember them. Rely on your observations. Are the gums less pink than they were an hour ago? Do those pulses feel weaker than when the dog came in? Is that

breathing pattern different? These are clues that no monitoring equipment will be able to detect. A skilled technician can never be replaced if they are using their critical thinking skills.

Encourage everyone in the clinic to practice their critical thinking skills. Teaching critical thinking is vital to the job satisfaction of the staff. It teaches technicians to become proactive and not reactive. Technicians should be empowered to think critically with every patient that walks into the door.
This process is something that must be built in to a clinic's culture. Information withholding, bullying, and horizontal violence are all too common in veterinary practices and spell death to critical thinking. When staff are afraid to speak up, afraid to ask questions and afraid to make mistakes, progress cannot be made. When only one person knows how to take dental radiographs or place difficult catheters the entire practice suffers. Critical thinking must be taught and practiced. While difficult, senior staff members must step back and allow employees to think for themselves. If a new technician or nurse is struggling with a question or technical skill, the instinct is to step in and do it for them for the sake of time. This is taking away a potential learning opportunity. If they cannot answer a question, reword it or ask another question with a similar theme to try to get them to the answer. This process takes energy and patience, but the best leaders and teachers are willing to help others grow to be their best. Fight bullying by structuring learning expectations so that everyone is involved with teaching. Reward not only the learning and progression of staff but also reward those who are teaching. Again, a culture change needs to occur and all teams need to be on board with the process and the goals, but the outcome is beneficial to the team as well as the financial gain of the hospital. Engaged employees stay longer and contribute more than just their hours in the clinic.

Encourage every technician on the staff to learn about their favorite disease. The more that person learns, the more everyone on the staff learns. Ask questions, participate in case rounds, attend all of the continuing education that you can. Don't just do it "because you have too." Use your brain. If veterinary technicians don't learn something new everyday, they will eventually leave the practice because they get bored. Cultivate and grow your critical thinking skills and you will be the best resource in your practice.

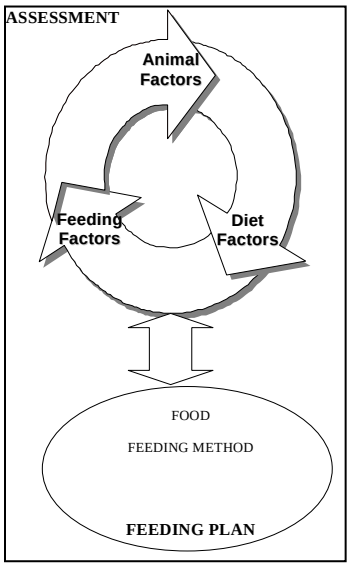
CASE STUDIES!

Sunday, July 11, 2021

MAKING NUTRITIONAL ASSESSMENT PART OF PATIENT CARE

Joe Bartges, DVM, PhD, DACVIM, DACVN
Board Certified Veterinary Nutritionist®
Professor of Medicine, Nutrition, and Interventional Radiology
The University of Georgia

The American Animal Hospital Association released the Guidelines for Nutritional Assessment (July/August JAAHA 2010). Utilizing the two-step iterative process, a screening assessment is made and if concerns are found, then a more detailed assessment is made. Following assessment, data are analyzed, a plan formulated and initiated, and repeated evaluation and modification of the plan is made. The American College of Veterinary Nutrition recommends a two-step iterative process in making nutritional recommendations where the first step is ASSESSMENT. During this step, assess the ANIMAL, the DIET, and the FEEDING factors.



ANIMAL FACTORS assessed include gathering historical information, performing physical examination, body condition and muscle condition scoring, and evaluating laboratory and imaging results if indicated. Gather information on any health or disease-related conditions, medications (including over-the-counter and nutraceuticals/supplements), reason for visit, and other household members. A thorough physical examination is performed and a body condition score assigned. There are 5- and 9- point body condition scoring systems; either can be used. In either scale, the middle number of the scale represents ideal body condition and a body fat content of 15-25%; numbers lower than this correspond to lower body condition and less body fat (0-15%) while numbers higher than this correspond to higher body condition and greater body fat (≥ 35%). Assigning a body condition score provides more information than body weight alone and can be used with a muscle condition scoring system where the scoring is normal, mildly decreased, moderately decreased, and markedly decreased (sarcopenia). Body condition scoring provides a measure of fat mass while muscle condition scoring provides a measure of lean body mass.

Descriptor	Description	5 point	9 point
CACHECTIC	Ribs are easily palpated with no fat cover; bony structures are prominent and easy to identify; muscle tone and mass often decreased; little to no subcutaneous fat; hair coat often poor; pronounced abdominal tuck	1	1
UNDERWEIGHT	Ribs are easily palpated with little fat cover; abdominal tuck present; bony structures are palpable but not prominent; hair coat may be poor; muscle tone and mass may be good or slightly decreased	2	3
IDEAL	Ribs are easily palpated, but fat cover is present; hourglass shape present and abdominal tuck is present, but not pronounced; bony prominences are palpable but not visible some subcutaneous fat, but no large accumulations; muscle tone and mass good; hair coat quality is good	3	5
OVERWEIGHT	Ribs are difficult to palpate due to overlying fat accumulation; hourglass shape is not prominent and abdominal tuck is absent; subcutaneous fat obvious with some areas of accumulation; muscle tone and mass good; hair coat quality may be decreased; cannot identify bony prominences	4	7
OBESE	Ribs are impossible to palpate due to overlying fat; hourglass shape is absent and animal may have a round appearance; subcutaneous fat is obvious and accumulations are present in the neck, tail-base, and abdominal regions; muscle tone and mass may be decreased; hair coat quality may be decreased	5	9

DIETARY FACTORS include gathering information on dietary intake and inspection of the food, if needed. Take the dietary history from the person that actually feeds the pet(s) asking for type of food, amount fed, frequency of feeding, table food or treats, access to other food (garbage, outside, etc.), supplements, and medications (including over-the-counter). If necessary, inspect a sample of the food or send a sample for analysis. You or your staff or your client can contact the company for more detailed information. When contacting a company, several questions should be asked based on the World Small Animal Veterinary Association; however, there are issues with these guidelines and so they are modified here:

1. Do you have a Veterinary Nutritionist or some equivalent on staff in your company and are they full-time or a consultant for your company? Are they involved with formulation of diets and validation of adequacy? Are they available for consultation or questions?
2. Who actually formulates your diets and what are their credentials?
3. Which of your diets are AAFCO Feed Trial tested? Have all been through feeding trials or are you using a lead product system?
4. Which of your diets have undergone a complete nutrient analysis? Are these analyses available for evaluation? Do you have a third party validate your results?
5. What specific quality control measures do you use to assure the consistency and quality of your product line? Can and will you produce these documents?
6. Where are your diets produced and manufactured? Can this plant be visited?

The guaranteed analysis provides information regarding the 4 major components of a pet food as percentages of the diet as fed including minimum amount of crude protein, minimum amount of crude fat, maximum amount of crude fiber, and maximum amount of moisture, and the caloric content. "Crude" refers to the analytical procedure and does not refer to the quality of the ingredient. The nutritional adequacy statement must be included and is designed to ensure that the product, when fed as the sole source of nutrition, is complete and balanced for one or more life stages, including how this adequacy was verified. The four recognized life stages by AAFCO are pregnancy, lactation, growth (non-large and giant breed dogs and large and giant breed dogs), and adult maintenance, and nutritional adequacy can be determined by feeding trials or by calculation. Note that there is no AAFCO nutrient profile for "senior" or "geriatric" dogs or cats. The calculation method involves determining the amount of nutrients in the diet and comparing to AAFCO nutrient profiles for that/those life stage(s). Feeding trials are performed by feeding the diet to the animals in that/those life stage(s) following AAFCO protocol. Feeding trials, while not perfect, provide indirectly information on bioavailability of nutrients and is preferred method for validation of nutritional adequacy. Keep in mind that companies may label foods as having been through feeding trials when in actuality they have not. They are allowed to use "lead product" testing. That is, the lead product in a line undergoes feeding trial and other products in that line/family may carry a feeding trial claim even when they have not been put through a feeding trial so long as the products in the line do not differ substantially from the lead product. Therapeutic diets, supplements, and treats often do not carry a nutritional adequacy statement but may. Therapeutic diets are formulated for specific non-healthy conditions, which are not recognized by AAFCO and for which no nutrient profiles exist (e.g. renal failure, liver failure, etc.); they usually carry a statement such as "intended for intermittent use" or "use only under the supervision or direction of a veterinarian". Snacks and treats are not formulated or intended to be the sole source of nutrition; therefore, they are not required to carry a nutritional adequacy statement. The label often contains other information, much of that may or may not have official definitions.

FEEDING FACTORS to be assessed include how the nutrition is provided and must take into account owner and animal factors. Simply filling a bowl within reach of the animal is not enough; the appropriate diet must be provided in the appropriate amount. The amount of energy required by the pet can be determined using one of two formulae: Linear: [(30 x BWkg) + 70] or Exponential: 70 x (BWkg0.75). This provides the RESTING ENERGY REQUIREMENT and this result is multiplied by a life stage or activity factor depending on the individual.

Life Stage	Canine Factor	Feline Factor
Gestation	1.0 – 3.0	1.6 – 2.0
Dogs – first 1/2 - 2/3	1.0 – 2.0	
Dogs – last 1/3	2.0 - 3.0	
Lactation	2.0 – 8.0	1.0 – 2.0
Growth	2.0 – 3.0	2.0 – 5.0
Adult intact	1.6	1.4
Adult neutered	1.4	1.2
Senior	1.0-1.4	1.0-1.1
Work – light	2.0	
Work – moderate	3.0	
Work – heavy	4.0 – 8.0	
Obese prone	1.2	1.0
Weight loss	1.0	1.0
Weight gain	1.2-1.4 ideal	0.8-1.0 ideal
Critical care (usually)	1.0	1.0

The second step is FORMULATION AND INITIATION OF A FEEDING PLAN. The nutritional plan is formulated based on the assessment phase and initiated. It is important that this plan is re-evaluated periodically (iterative process) and adjustments made based on what is found during assessment. Recommendations for the feeding plan are made based on life stage and physiological or pathological condition of the pet as well as the life style of the owner. Working within the constraints placed by the owner helps to ensure compliance; otherwise, recommendations will not be followed. There is no "one best" diet available for healthy pets or for pets that suffer from a disease. Oftentimes, many options exist including homemade diets. Today's health care providers, veterinarians and technicians, need to be able to assess a pet, evaluate diets, and make recommendations on diets and feeding. Knowledge of assessment and formulation of a nutritional plan should be part of a patient's health care. Use body condition scoring in addition to weight to assess nutritional status.

FEEDING ADULT DOGS AND CATS

Often we compare nutrient requirements for different life stages and disease states with those for adults. Adult dogs and cats have a wide margin of safety nutritionally while other life stages and disease states have a much narrower margin of safety. Typical diets for healthy adult dogs and cats are presented below.

Nutrient	Dogs	Cats
Crude Protein (dry matter basis)	20-30%, can be more	40-60%, can be more
Crude Fat (dry matter basis)	12-20%, can be more	12-20%, can be more
Crude Fiber (dry matter basis)	2-5%, can be more	2-5%, can be more
Calcium:Phosphorus ratio	1.1:1.0 – 2.0:1.0	1.1:1.0 – 2.0:1.0
Carbohydrate	None	none

Remember that all diets that carry an AAFCO nutritional adequacy statement must meet nutrient requirements defined by AAFCO; therefore, these diets are meant for the average adult dog or cat. Also, note that there are no published carbohydrate requirements for dogs and cats. Keeping pets healthy during early to middle adulthood may improve quality and quantity of life. One means to accomplish this is identifying patients at risk for diseases that may have a food responsive component. There are known breed predispositions for common diseases and if asked to identify three common conditions for a certain breed, this is easily accomplished. Identifying and intervening before these conditions occur is preventative medicine rather than waiting for the condition to occur and then modifying diet and using pharmacologic therapy that is reactive medicine.

FEEDING THE GERIATRIC DOG AND CAT

The idea that older dogs and cats have nutrient requirements that differ from those of young or middle-aged animals was introduced when a commercially available diet formulated for older dogs was introduced in 1977. Remember, though, that there are no AAFCO nutrient profiles for "senior" or "geriatric" only "adult" and so these diets must meet or exceed AAFCO nutrient requirements for an adult dog or cat. Underlying the formulation of so-called senior diets is the theory that proper nutrition can increase the length and quality of life and that some optimal nutrient formulation exists for accomplishing this goal. Recommendations have been made for restriction or supplementation of particular nutrients. Unfortunately, there is little data to prove a specific nutrient formulation will definitely increase longevity or delay the onset of disease. It should be realized that some nutritional effects accrue over a long period before becoming clinically evident. Thus, the nutrient intake of the first three-fourths of an animal's life is likely to impact the nutritional consequences manifested in the last fourth of life.

Chronological ages have been proposed to identify the point at which a dog or cat has reached old age.

	Dogs			
Cats	Small	Medium	Large	Giant
7	7	7	7	5
12 (8-9?)	11.5	10	9	7.5

A better criterion for judging seniority would be physiologic age; however, chronologic age has proved to be a poor indicator of physiologic age in human beings. Unfortunately, indicators of physiologic age such as glucose tolerance, insulin release, daily conversion of thyroxine to triiodothyronine, strength of connective tissue, various indicators of immunologic competence, nerve conduction velocity, glomerular filtration efficiency, and various cardiopulmonary function tests are often difficult or expensive to measure, and data are often difficult to

interpret for an exact physiologic age. Some authors suggest that an animal should be considered geriatric when visible signs of aging become apparent, such as white hair, general decline in coat condition, decrease in acuity of sight or hearing, decrease in amount of activity, impairment of locomotor function, and lethargy. For the sake of discussion, a distinction should be made between the terms “old” and “senior” when compared with “geriatric”. Not every old dog or cat contracts the same dysfunction or disease, and different geriatric conditions can require different nutrient formulations to ameliorate clinical signs. The purpose of formulating senior diets is to prolong the length and quality of life by delaying the onset of true geriatric states.

Basal metabolic rate (BMR), amount of lean tissue, daily amount of activity, and efficiency of digestive function all interact to determine the total number of calories required daily to maintain body weight. The BMR, which is the largest component of MER, is largely determined by the amount of lean body tissue. Amount of activity constitutes the second-largest proportion of MER. In addition to requiring calories to perform an activity, such activity can also help preserve lean body tissue and, thus, maintain BMR. An animal’s specific BMR and amount of activity combine to account for most of the energy required to maintain body weight. The amount of food required to supply MER is determined, in part, by the nutrient content of the food, but also by the efficiency with which macronutrients that provide energy are digested and made available to the animal. Amount of activity, amount of lean body tissue, and digestive efficiency are all components that may change as animals get older, altering requirements for energy and other nutrients, and, ultimately, the amount and desired composition of the diet to optimally support an older dog or cat.

In several breeds of dogs, the amount of food required to maintain body weight decreases with age. Number of calories required to maintain stable body weight decreases asymptotically as dogs get older, so that approximately 20% fewer calories are needed to maintain an older dog (> 8 years of age) when compared with a young dog (1-2 years of age) of similar weight. Studies have documented that ignoring the age-related decrease in required number of calories will produce overestimates of energy requirements for 3- to 4-year-old dogs. A 20% decrease in required number of calories also has been observed between young adult and older people, with the decrease in number of calories resulting from a decrease in amount activity, decrease in lean body tissue, and the associated decrease in BMR. Similar changes for body composition and activity have been observed in dogs. Loss of lean body tissue is estimated to account for approximately a third of the decreased energy requirements with the remaining half to two-thirds of the decrease accounted for by a decrease in the amount of activity. It also has been documented in people and dogs that individuals maintaining the amount of daily activity near that of young adults will have caloric requirements similar to that for young adults, presumably through maintenance of lean body tissue as well as number of calories used to perform the activity. Cats, on the other hand, do not have a major change in caloric requirements over the course of their life as their activity occurs mainly in bursts rather than sustained exercise and so the additional energy intake requirement for activity is less than dogs and does not change much over time.

The overall efficiency of nutrient digestion is maintained as dogs and cats get older. Although rodent models demonstrate that fat restriction increases life span, decreased life span with increased fat intake is related to atherosclerosis, which occurs rarely in dogs. Furthermore, dietary fat restriction undertaken when a dog is aged probably provides little benefit as long as obesity does not occur.

Alterations in body composition and condition are the anticipated result when there is not a compensation for age-related changes to MER or digestive efficiency via changes in quantity or composition of food. Cross-sectional epidemiologic data for proportion of the dog population that are overweight at a specific age reveals that approximately 20% of 1- to 2-year-old dogs are overweight. The proportion of overweight dogs increases to 40 to 50 percent between 2 and 5 years of age. For dogs, this profound increase in prevalence of overweight animals corresponds to the segment on the calorie-versus-age curve at which calories are decreasing the most for each increase in one year of age. The prevalence of overweight dogs peaks when animals are approximately 8 to 10 years old, and then decreases with increasing age. Concurrent with this decrease in overweight animals is an increase in the proportion of thin animals; the proportion of thin animals increases from a stable value of < 10% of the population in animals that are 1-12 years old to a value of 30-50% of the population in animals that are > 12 years old. Epidemiologic analysis indicates that most dogs that gain excess amounts of weight do so before they reach their senior years. This is also true in cats. Therefore, dietary alterations to control excess weight should be emphasized during early adulthood through middle age; whereas, more emphasis on preventing weight loss in optimal-weight dogs and cats should be given to those that are senior or geriatric.

Protein requirements are often represented as differing for older pets when compared with younger pets. However, it is debatable whether older dogs need more or less protein than younger dogs. The answer depends on the measurements used to define protein requirement and whether the reference point that defines required amounts of protein for young adults is the bare minimum amount of 100% biological-value protein, the minimum amount of protein from typical pet food ingredients, or the protein content of typical commercial maintenance food. Wannemacher and McCoy reported more than 50 years ago that 1-year-old Beagles could achieve and maintain maximum protein repletion with less dietary protein than 12- to 13-year-old Beagles, based on protein-to-DNA ratios measured from muscle and liver. The 1-year-old dogs required 0.4 g of nitrogen/kg of body weight (equivalent to 2.5 g of crude protein/kg; 22.75 g of crude protein/day; 12.5% of calories from crude protein), whereas the 12-to 13-year-old dogs required 0.6 g of nitrogen/kg (equivalent to 3.75 g of crude protein/kg; 34.13 g of crude protein/day; 18/8% of calories from crude protein) to achieve maximum protein-to-DNA ratios. Interestingly, young and old dogs achieved nitrogen balance after 4 to 8 weeks of being fed even the lowest concentrations of protein, indicating that nitrogen balance is a less-sensitive measure for determining protein requirements than protein-to-DNA ratios of tissues. Sheffy, et al fed 4 diets representative of maintenance, senior, high-protein and low protein diets for 1 to 2 years to 16 Beagles that were 10 to 12 years old and 8 Beagles that were 1 to 2 years old. Adverse effects or deficiencies attributable to diet were not observed as determined from results of various hematologic, serologic, radiographic, or organ-function tests, and nutrient –retention studies. Serum urea nitrogen concentration was associated with dietary nitrogen content, independent of age. Older dogs had increased concentrations of cholesterol and serum phosphorus, increased alkaline phosphatase activity, and decreased lymphocyte proliferation responses, independent of the diet fed. It was concluded that nutrient requirements of older dogs were not markedly different from adult maintenance.

Cats are true carnivores and restriction of dietary protein is less desirable than in dogs unless they have a disease where dietary restriction of protein is indicated. Even in the face of these diseases, it is possible particularly with homemade diets or particular commercial diets to feed higher protein than found in more traditional heat-processed foods.

Because it is hard to define physiologic old age in terms of chronologic age or to determine it by other means, it is also difficult to know when to switch the diet to a senior nutrient product. Furthermore, because nutrient requirements and products for older pets are nearly identical to those for maintenance requirements and products for adult pets, it could be questioned whether there really is a need to switch to senior foods. Each animal's body condition and health status, rather than age alone should guide decisions regarding the type and amount of product that should be fed to an older dog or cat. The concept of tailoring feeding methods and amounts to maintain or achieve optimal body condition is not new. Optimal body condition and muscle mass are the ultimate practical clinical criteria for judging whether most diets and feeding methods are appropriate. On the basis of feeding for optimal condition, analysis of the epidemiologic data on body condition of older dogs and cats and documented risks of dogs and cats that are outside the optimal range for body weight and condition, it has been proposed to categorize older pets into four categories.

The first category is the pet that is chronologically old, but physiologically young, and eating a good-quality maintenance diet. This animal is at optimal body weight and condition without a decrease in amount of daily activity and is clinically normal on the basis of results of physical examination, CBC, serum biochemical analyses, and urinalysis. It is difficult to recommend switching the diet to a senior-type food in this situation.

The second category of older pet is chronologically old and metabolically efficient. This animal is still clinically normal on the basis of results of physical examination, CBC, serum biochemical analyses, and urinalysis. However, there has been an evident decrease in amount of activity and a tendency to gain weight. This animal may benefit from switching its diet to a senior-type product with decreased caloric density. The goal should be to adjust the quantity of food to return and maintain and optimum body condition.

The third category of older pet is chronologically old and metabolically inefficient. Again, the animal is still clinical normal; however, this animal tends to lose weight and cannot eat a sufficient quantity of a maintenance diet to maintain optimal body weight. Weight loss should be stopped and body weight returned to optimal amounts by feeding more caloric- and nutrient-dense formulas, such as newer nutrient-dense senior products. Alternatively, diets formulated for growing animals, or animals with increased amounts of activity could also be considered for stabilizing body condition.

The fourth category of older pet is a true geriatric animal. This animal has an identifiable physical or metabolic abnormality in conjunction with old age. The ideal goal for feeding is maintenance of optimal

condition; however, considerations for desired nutrient content in addition to calories will be applicable if the disease can be ameliorated by dietary modulation. Senior-type products may, or may not, be the best choice, depending on the disease and status of the animal.

Older animals are less capable of responding to physiologic challenges. Results from studies of geriatric human beings have documented the utility of maintenance of optimal weight and condition and reducing medical complications and mortality. Investigators assessed 81 nutritional and non-nutritional variables for their usefulness in predicting complications during hospitalization or mortality after discharge in 109 geriatric patients. Percentage of body weight lost during the year prior to hospitalization proved to be the best predictor of mortality during the 1-year period after discharge. Percentage of body weight lost was the third-best predictor of complications during hospitalization (assessment of functional status was best and concentration of serum albumin was second best). Percentage of body weight lost is associated closely with the concept of percentage below optimal weight, which also provided a useful indicator of nutritional status in a group of 501 geriatric patients. Geriatric human beings can quickly become malnourished, and the condition often resolves slowly and makes recovery from illness even more a challenge and less likely to happen. The nutritional status and nutrient support of geriatric patients requires close scrutiny to maximize the chance of a successful resolution or prolonged life of acceptable quality.

The aforementioned rationale is a non-dogmatic means of deciding when dietary changes should be made and which changes to make as dogs get older. This rationale can be used until researchers provide more definitive answers regarding the exact nutrient alterations that are required and the age or set of physiologic variables that determine when those nutrient alterations should be implemented.

Category	1	2	3	4
Chronological	Old	Old	Old	Old
Physiological	Young	Old	Old	Old
Metabolism	Efficient	Efficient	Inefficient	Inefficient
Others	-----	Gains wt.	Loses Wt.	Variable
Clinically	Normal	Normal	Normal	Abnormal
Diet	Maintenance	Senior	Calories	Variable

OTHER CONSIDERATIONS FOR THE OLDER DOG AND CAT

We have already discussed dietary calories and protein, but what are other things to consider. Older dogs and cats may or may not need higher dietary fiber. Certain fibers – primarily soluble or fermentable fiber – may modulate the gut microbiota and that may have some benefit. Increasing fiber intake for presumed decreased gastrointestinal function, though, is not based on scientific data for older dogs and cats. Encouraging drinking and increased water intake may be beneficial for older dogs and cats even if not azotemic. Cats in particular are prone to dehydration and older dogs and cats may either not be as likely to actively seek water or may have some degree of cognitive dysfunction and so may become dehydrated. Adding water to food, changing water frequently or using circulating water dispensers, and adding flavoring agents to water may help.

Older dogs and cats often continue to eat but may eat less. Monitoring weight – especially weight loss – is important for an older dog and cat. Hyporexia may be an indication of an underlying disease or the early stages of a progressive disease such as chronic kidney disease, cancer, or hyperthyroidism. It may also be seen in patients with osteoarthritis or spinal issues where mobility prevents them from getting to the food or from maintaining a position to eat the food. Dental disease may also be an issue in older pets and may inhibit adequate food, and water, intake. In addition to dental procedures with oral cavity disease, adding an oral health product to the drinking water and good at home dental care, if the pet will let the owner do so, is important and helps greatly.

Keep pets especially older pets playing. Cats are hunters and you can use this throughout their life with food puzzles so that the dog or cat remains engaged and inquisitive. Toys also keep pets engaged.

Age is not a disease and not all older pets are physiologically or metabolically old. Nutrition should be tailored to the pet and based on gathering good historical information and performing a thorough physical examination. Use of annual wellness laboratory evaluation may identify diseases early with intervention including dietary modification before the disease progresses.

I CAN DO IT MYSELF: HOMEMADE DIETS

Joe Bartges, DVM, PhD, DACVIM, DACVN
Professor of Internal Medicine, Interventional Radiology, and Nutrition
The University of Georgia
Athens, GA
jbartges@uga.edu

HOMEMADE DIETS

Some owners prefer to prepare homemade foods – feel less guilty and have impression of preparing a "real meal" that is "more natural" and "more traditional". Nearly all dogs and cats in the US consume table foods at some time in their lives. Majority of dogs and cats in US receive >90% of calories from commercial foods. When a client wants to prepare pet foods at home, it is important for veterinarians to understand the client's reasons and motivation. In many cases it is possible to address their concerns and to recommend an appropriate commercial food. If they still wish to cook, then proper guidance can be provided.

Some owners wish to cook homemade diets in order to provide a natural or organic food. Remember, there is no legal definition for the terms "natural" and "organic". Pet owners may also want to prepare vegetarian food for their dog or cat because they are vegetarian or vegan. Because cats are true carnivores, vegetarian cooking should be discouraged. Other owners wish to prepare homemade diets in order to avoid additives, preservatives, and contaminants. Pet food labels may be difficult to read and understand and they do not contain as much information as human food labels; therefore, some choose to home cook because they are more comfortable with being in control. Some pets will only eat table foods because it has become a habit. Lastly, homemade diets may be used for dietary elimination trials.

It is possible to achieve the same nutrient balance with a homemade food as with a commercially prepared food. However, this largely depends on the accuracy and competence of the person formulating the food, and on the compliance and discipline of the owner. Unfortunately, some homemade recipes are flawed even when followed exactly and consistently. IN one survey, 90% of homemade elimination diets prescribed by 116 veterinarians in North America were not nutritionally adequate for adult dog or cat maintenance. Few of the recipes available in books, magazines, and on-line have been tested to document the nutritional adequacy of the diet.

There are common nutrient problems in many homemade foods. Many formulations contain excessive protein, but are deficient in calories, calcium, vitamins, and micro-minerals. Commonly used meat and carbohydrate sources contain more phosphorous than calcium resulting in inverse calcium: phosphorous ratio. Foods designed by clients are commonly deficient in fat and energy density or contain an unpalatable fat source (vegetable oil). Homemade foods are rarely balanced for micro-minerals and vitamins because veterinary vitamin-mineral supplements are not complete nor are the nutrients well balanced within the product.

People are taught that eating a variety of foods is nutritionally sound. Clients often extend this principle to their pet's nutrition. Pet owners perceive that feeding a variety of foods is their best defense against malnutrition. Likewise, many owners feed a homemade diet because they can use a variety of ingredients. Some owners choose meat and carbohydrate sources for their pet's food based on their own preferences, product availability, or affordability. Other pets are fed "leftovers" such as fat trimmings, bones, vegetable skins, crusts, and condiments. Some owners feed their pets according to guidelines for humans not realizing that dogs and cats have different requirements. A common problem with homemade diets is that the vitamin-mineral supplement is left out because of inconvenience, expense, or failure to understand its importance – after all, many humans do not take vitamins. Lastly, some homemade diets use raw ingredients – we will talk more about these in a little bit

Veterinarians encounter a wide variety of pet food recipes from breeders and the popular press. Some owners want an opinion as to whether the recipe is good and others want to alter the recipe. Homemade formulations can be checked for nutritional adequacy and adjusted using the "quick check" guidelines:

1. Do five food groups appear in the recipe?
    a. Carbohydrate/fiber source from a cooked cereal grain
    b. A protein source, preferably of animal origin, or if more than one protein source is used, one source should be of animal origin
    c. Fat source
    d. Source of minerals, particularly calcium
    e. Multivitamin and trace mineral source
2. Is the carbohydrate source a cooked cereal and present in a higher or equal quantity than the meat source?
    a. Carbohydrate to protein ratio should be at least 1:1 to 2:1 for cat foods and 2:1 to 3:1 for dog foods
    b. Sources are cereal such as cooked corn, rice, wheat, potato, or barley

    c. These sources have similar caloric contributions, but some carbohydrates contribute a substantial amount of protein, fiber, and fat
3. What is the type and quantity of the primary protein source?
    a. Overall protein quality of the diet can be improved by substituting an animal-derived protein source for a vegetable protein
    b. Skeletal muscle protein from different species have similar amino acid profiles
    c. Final food should contain 25-30% cooked meat for dogs (1 part meat to 2-3 parts carbohydrate) and 35-50% cooked meat for cats (1 part meat to 1-2 parts carbohydrate)
    d. Providing some liver in the meat portion is recommended once a week or no more than ½ of the meat portion on a regular basis – corrects most potential amino acid deficiencies and contributes fatty acids, cholesterol, energy, vitamins, and microminerals
    e. If owner requests an ovo-lacto-vegetarian food, eggs are best
    f. If vegan food is requested, soybeans are the next best, but incomplete, amino acid profile
4. Is the primary protein source lean or fatty?
    a. Lean protein sources require addition of an animal, vegetable, or fish fat source at 2% of the formula weight for dogs and 5% of the formula weight for cats
    b. If a homemade food lacks sufficient caloric density, addition of cooked beef or chicken fat, poultry skins, vegetable or fish oils can markedly increase caloric density without adding other nutrients
5. Is a source of calcium and other minerals provided?
    a. An absolute calcium deficiency is common
    b. Many owners erroneously assume cottage cheese, cheese or milk added in small quantities provides adequate calcium
    c. Most foods require a specific calcium supplement
        i. When the protein fraction equals or is greater than the carbohydrate fraction, usually only calcium carbonate is added (0.5 g/4.5 kg cat/d and at least 2.0 g/15 kg/dog/d).
        ii. Calcium and phosphorous supplementation may be necessary when the protein fraction is less than the carbohydrate fraction. Steamed bone meal, dicalcium phosphate, and certain proprietary mineral supplements contain @ 27% calcium and 16% phosphorous (about 2:1) and micro-minerals
6. Is a source of vitamins and other nutrients provided?
    a. A human adult over-the-counter vitamin-mineral tablet that contains no more than 20% of the recommended daily allowances for people works well for both dogs and cats at ½ to 1 tablet per day (@ 1 gm/tablet).
    b. One tablet per day of a human adult product will not over-supplement pets with calcium, phosphorous, magnesium, vitamins A, D, and E, iron, copper, zinc, iodine, and selenium according to AAFCO maximum allowances for canine and feline foods.
    c. In general, veterinary supplements provide between 0-300% of vitamin-mineral requirements of dogs and cats

Substitution of ingredients can be done, but should be researched as to the equivalent amounts. One protein source is not the same as another. Other instructions that should be given owners include those for preparation, storage, and feeding. Emphasis should be made to not eliminate an ingredient or indiscriminately substitute ingredients. Owners that wish to use raw eggs and meats should be informed that there is a risk for infectious diseases. Animal ingredients should be cooked for at least 10 minutes at 180F. Vegetable ingredients should be washed or rinsed and cooked if increased digestibility is desired. Since antioxidants are not usually added to homemade diets, storage in airtight containers at refrigeration temperature can be done for 7 day stretches. Large quantities can be frozen. Owners should check appearance and odor daily to make sure rancidity or contamination has not occurred. Starches should be cooked to increase digestibility; however, they should be cooked separately from the protein source. Carbohydrate sources require a longer cooking time; meat and liver should not be overcooked or protein denaturation will occur

Pets should be evaluated routinely whether they are being fed commercial food or homemade food. Stools should be formed although they may contain more water. Body condition and weight should be maintained. If problems are encountered, then either the homemade diet should be re-evaluated and modified or use of a commercially available diet should be encouraged.

RAW FOOD DIETS (BONES AND RAW FOOD OR BIOLOGICALLY ACTIVE RAW FOODS)

Veterinarians deal with pet owners who have access to a large body of information on small animal nutrition. Food is something that everyone relates to because it is one of the necessities of life. Food can have

important effects on psychological well-being. Diet is something that an owner can control. Nutritional therapy is viewed as natural and holistic as opposed to surgical and pharmacological management of disease. For these reasons, there are a growing number of homemade diet recipes available through the internet and published sources that tout health benefits.

An example of a non-traditional pet food is raw food diets. Proponents of raw food diets claim numerous benefits such as improvement in coat and skin; elimination of breath, body, and fecal odor; improvement in amount of energy and behavior; improvement in overall health and immune function; and reduction of the incidence of many medical conditions including allergies, arthritis, pancreatitis, and parasitism.

The rationale for use of raw food is simple. Dogs and cats are carnivores that evolved eating raw foods. In addition, commercial foods are heat processed which alters or destroys nutrients and essential enzymes. Therefore, commercial foods may not be a natural or nutritionally sound diet for dogs and cats.

There are three major categories of raw food diets:

1) Commercially available raw food diets. These diets are intended to be complete and balanced without the need for additional supplements. These diets are typically sold in frozen form.
2) Homemade complete raw food diets. Many recipes for homemade raw food diets are available in books and articles as well as on the internet. The three most popular homemade raw food diets are the bones and raw food (BARF) diet, the Ultimate diet, and the Volhard diet.
3) Combination diets. These consist of commercially available grain-and-supplement mixes. The grain mix is fed in combination with raw meat.

Although there are numerous health claims for these diets, there is no scientifically proven information, only testimonials. There are several serious potential drawbacks to these diets.

Nutritional imbalances. In one small study, raw food diets were found to have one or more of the following: an unbalanced calcium-to-phosphorous ratio, increased vitamin D levels, decreased potassium content, decreased manganese content, decreased or increased zinc content, decreased iron content, and increased vitamin E content.

Intestinal foreign bodies. There are sporadic reports of esophageal foreign body and obstruction due to ingestion of bones.

Infectious agents. Raw foods, especially meat, may contain infectious agents, many of which are zoonotic. *Escherichia coli O147:H7* was cultured from one homemade raw food diet. In one study, approximately 50% of raw food diet contained non-type specific *E* coli while these were not found in commercial dry foods. In another study, *E coli* was identified in 15/25 (64%) diets; however, E. coli O157 was not detected. Salmonella spp. were detected in 5/25 (20%) diets. Clostridium perfringens was identified in 5/25 (20%) samples. A toxigenic strain of C. difficile was isolated from one diet. Staphylococcus aureus was isolated from 1/25 (4%) diets. Campylobacter spp. were not isolated from any of the diets. Raw pork may can contain *Yersinia enterocolitica 4/O:3* and has been isolated from feces of dogs and cats fed raw pork. Listeria monocytogenes has also been isolated from raw pork and has been associated with disease in dogs including reproductive problems. Rendered raw meat has been shown to be contaminated with bacteria, including *Salmonella spp*, (in one study 80% of raw food diets cultured positive), *Proteus spp*, and *Pseudomonas spp*, that may also be carried by flies. *Clostridium difficile* has been isolated from feces from dogs and cats. In addition to bacteria, raw foods may contain *Toxoplasmosis*, *trichinella*, and other parasites including *Echinococcus*. These may pose health hazards to animals as well as to the humans who are preparing the food. One argument given by raw food proponents is that the bacteria do not cause disease in dogs or cats. One concern that is often overlooked is the role of dogs and cats to be carriers of potentially zoonotic infectious agents. For example, dogs have been shown to carry *Escherichia coli* that can cause non-enteric *Escherichia coli* infections in human beings. In addition, indiscriminate use of antimicrobials may result in antimicrobial resistance of enteric organisms, which, in turn, may find its way into human medicine.

There are some publications mainly case reports concerning consumption of raw foods or raw food diets by dogs and cats and reviews expressing opinions concerning raw food consumption by dogs and cats. Cases and case series include: anestrous due to hyperthyroidism associated with consumption of raw meat, hyperthyroidism and associated signs due to consumption of raw meat and glands, hypervitamonisis A in cats resulting in cervical ankylosing spondylosis and hepatic fibrosis, septicemic Salmonellosis in cats fed a raw chicken diet, and diarrhea associated with Salmonella in cats fed raw meat. However, there is a controlled study of growth in kittens fed a homemade raw diet, commercial raw diet, and commercial heat-processed diet that showed decreased stool volume and improved fecal consistency, better overall weight and body composition gain, and no adverse effects including infections in kittens fed the raw chicken homemade diet. There are other known or potential benefits of homemade especially raw food diets: they are typically limited ingredient diets and so may help with food allergies or intolerances, they are more digestible and so decrease on amount of food fed as well as amount of fecal matter

produced, they are not processed and so may be beneficial in certain situations as processed foods have been linked to certain diseases and clinical conditions in humans, and owners have a sense of control over what they feed their pet.

So what kind of recommendation do we make to clients? There are two issues that require resolving when dealing with raw food diets and clients who wish to feed them. First, we must decide whether we believe in their use and feel comfortable in providing advice concerning their use and preparation. Second, we must provide competent advice on their use. These issues extend beyond health issues for dogs and cats to health issues with the human beings that share the same environment and prepare the food. Clients should be made aware of the potential for problems especially infectious diseases associated with raw food diets and hygiene should be emphasized. Raw food diets should be kept on a bottom shelf in the freezer or refrigerator to prevent contamination of other foods and if possible the raw pet food should be kept in a separate refrigerator. Separate food preparation bowls and utensils should be used and they should be washed as soon as possible after using. Homes with young children or immunocompromised adults should be strongly scrutinized concerning risk-benefit to the pet. Most important – good hygiene and common sense.

Standard Pet Formula - adequate for healthy dogs and cats over 6 months of age – from Veterinary Information Network (Susan Wynn, Claudia Kirk, Joe Bartges, Craig Datz)

1 pound fresh boneless skinless chicken breast
2 and 2/3 cup cooked white rice
1 Tablespoon safflower oil
1/4 tsp Morton's lite salt
1/4 tsp iodinated salt
3 grams of calcium carbonate without vitamin D (regular Tums - check size)
1 Centrum adult multivitamin-mineral supplement (no special senior, ocular, women's or other versions)
1/4 tsp taurine powder (or 500 mg tablet) (taurine is optional for dogs - essential for cats)

Sauté chopped chicken breast in oil until thoroughly cooked. Add rice and salt.Grind Tums (calcium carbonate), multi vitamin/mineral tab, and taurine supplement together. Add to cooled mixture. Store in refrigerator. Larger batches may be prepared in advance and stored in the freezer.

Nutritional profile
40% protein (Dry matter basis (DMB))
12% fat DMB
6% calcium DMB
4.3% phosphorus
1.4:1.0 calcium:phosphorus
Calories: 1046 kcal per batch or 1.12 kcal/gram
Batch size: 932 grams
To feed, calculate caloric needs and divide into twice daily feeding. One recipe batch should provide adequate intake for a 40-45 pound dog for 1 day. Adjust intake to maintain ideal body weight

There are many resources available that can be found at the American College of Veterinary Nutrition web site (http://www.acvn.org)

Chronic Enteropathies – Tales from the Crypts

Dr. Joe Bartges
Professor of Medicine and Nutrition
The University of Georgia
Athens, GA, USA 30602

An **adverse reaction to food** is defined as a clinically abnormal response attributed to an ingested food substance, and may be further categorized as immunologic or non-immunologic in nature. Food allergy is an immunologically mediated, reaction to ingested food. This is different than food intolerance, which is a non-immunologically mediated adverse reaction including toxic reactions, pharmacological reactions, metabolic reactions, and idiosyncratic reactions. Certainly, if clinical or subclinical gastrointestinal disease occurs which alters mucosal integrity, absorption of antigenic proteins may occur which may initiate the processes. Inflammatory mediators involved in food allergy may include interleukins, platelet activating factor, histamine and cytokines. Clinical signs of food allergy relate primarily to dermatologic and gastrointestinal. Dermatologic signs often include pruritis, erythema, and secondary pyoderma. Gastrointestinal signs may include vomiting and/or diarrhea, flatulence, perianal fistulae, and anorexia. Other potentially associated disorders include cholangiohepatitis/cholangitis, feline asthma, idiopathic epilepsy, and feline urinary tract disease. Most basic food ingredients have potential to induce an allergic response, although proteins cause the majority of reactions. Dietary components reported to cause food sensitivity in dogs and cats include: cow's milk, beef, mutton, pork, chicken, rabbit, horse meat, fish, eggs, oatmeal, wheat, corn, soy, rice flour, potatoes, kidney beans, canned foods, cod liver oil, dry food, pet treats, and food additives.

Chronic enteropathy

While there are many potential causes of chronic enteropathies, the 4 most common forms are food responsive, antimicrobial responsive, inflammatory bowel disease, and neoplasia (typically small cell lymphoma). All may be associated with inflammation; however, their response to different treatments differ. Often several mechanisms may be occurring.

Treatment of food responsive enteropathy

Elimination diets and dietary challenges. The most useful and reliable aid in diagnosis of dietary sensitivity is the procedure of feeding a restricted or elimination diet followed by dietary challenge with a test meal. Elimination diets must be individualized based on the previous dietary exposure. A detailed study of the animal's diet will allow identification of foods that have not been fed before, and that could be used to formulate a nutritionally balanced elimination diet that will "hypo-allergenic". If it is not possible to formulate a suitable elimination diet, then a restricted diet may be used that contains only one or two potential allergens, preferably ones that the animal has not eaten in the preceding month. Many homemade diets that are used as elimination diets are not complete and balanced (e.g. cottage cheese and rice, or chicken and rice). Supplementation with vitamins and minerals is encouraged, but avoid use of supplements that contain potentially offending foodstuffs (e. g. beef or pork). It is tempting to use commercially prepared "hypoallergenic diets" during the diagnostic period for owner convenience and to ensure feeding of a complete and balanced diet. This may be effective, but

approximately 20% of dogs diagnosed as food hypersensitive when fed a home-cooked lamb and rice diet manifested clinical signs of allergic dermatitis when fed the commercially prepared lamb and rice diet. Gastrointestinal signs may subside in 3-5 days, but if it chronic in nature; it may take 4-6 weeks. Once clinical improvement is noted, it is advised to attempt to identify the offending antigen by introducing foodstuffs to the elimination diet

Protein hydrolysates. Because proteins with molecular weights over 18,000 Daltons are incriminated as being antigenic, modification of proteins to compounds having lower molecular weight may be of benefit. Protein modification is a process that alters the physical characteristics of protein molecules, presumably reducing the antigenicity and rendering them less able to elicit an immune response. By reducing the average weight of the protein molecule, this process can result in a protein that may be truly hypoallergenic. To be effective, it must reduce the molecular weight of the protein below 18,000 Daltons. Recently, several commercially available diets containing protein hydrolysates have been introduced including Exclude, DVM Pharmaceuticals, Hill's Prescription Diet z/d, and Purina's CNM HA-Formula. Anecdotally, these diets appear to be effective as elimination diets, and they have the advantage of being complete and balanced. These diets may be used long-term, but cost more.

Pancreatic enzyme replacement – while we typically use pancreatic enzymes with exocrine pancreatic insufficiency, patients with diffuse gastrointestinal disease may exhibit some degree of malassimilation. Feeding highly digestible diets and pre-digesting food with pancreatic enzymes may improve stool quality by increasing digestion and absorption. Pancreatic enzyme supplementation does not replace feeding a hydrolyzed protein diet; however.

Homemade and raw diets. Feeding a homemade diet including a raw food diet may be beneficial in patients with food intolerances. These diets are composed of whole ingredients and so unprocessed. Processing may induce advanced glycated end-products (AGE's) that are glycosylated proteins and may serve as antigens inducing a reaction. Because some dietary ingredients may become antigenic due to processing, feeding a whole ingredient or raw food diet means there is no processing. Additionally, homemade diets are typically more digestible and smaller quantities may be fed. Homemade diets allow for control of ingredients and is limited only by having a complete and balanced diet formulated and monitoring for dietary drift by the client.

If patients respond to dietary change, often the response is within 2 to 3 weeks. While some may respond after a longer period, it is worth considering adding in therapy. If the dietary change is a complete and balanced diet and there is a response, then the diet can be continued indefinitely.

Antibiotic responsive enteropathy

Recently, enteroinvasive E coli has been found in some boxers with ulcerative colitis. Enteroinvasive bacteria may play a role in other intestinal diseases. This may explain why some patients are "anti-microbial responsive" although other microbes may play a role. Antimicrobials that are used include metronidazole, tylosin, enrofloxacine, and oxytetracycline. An alternative strategy is prebiotic or probiotic therapy. These may change the enteric bacterial population and immune response to the organisms. Chronic large bowel enteropathy tend to response to increased dietary fiber. Fecal transplantation is another therapy to consider.

Probiotics – there are many probiotics available for use. Although some are "veterinary specific", there are not actually species specific probiotics. As a general rule: "more is better" – more bugs of more types in more numbers. For comparison: Visbiome has 450 billion organisms of 8 strains, Culturelle has 10 billion organisms of 1 strain, Proviable has 5 billion organisms of 7 strains, ProstoraMaxx has 100 million organisms of 1 strain, and Fortiflora has 10 million organisms of 1 strain. Probiotics may help with GI disease by altering the gut microflora, competing with pathogenic enteric organisms, and by producing beneficial substances while metabolizing potentially harmful ones.

Inflammatory bowel disease

Glucocorticoids – either systemic (e.g. prednisone: 1-2 mg/kg PO q24h or divided q12h) or topical (e.g. budesonide: 3 mg/m^2 q24-48h (dogs), 1 mg/cat PO q24h (cats)). Glucocorticoids are often very effective if eosinophilic component.

Azathioprine – a purine analog that is immunosuppressive. Used primarily in dogs and has been reported that cats are very sensitive to toxicity. There may be a lag phase between initiation and response; however, newer data suggests that it is not necessarily more than 1 week or so. In dogs, there are a couple of protocols: 2 mg/kg PO q24h x 2-4weeks, then either 1 mg/kg PO q24h or 2 mg/kg PO q48h, then 1 mg/kg PO q48h until decide to stop. It can be used indefinitely if patient tolerates. May induce bone marrow suppression, liver disease, and pancreatitis

Chlorambucil – an alkylating agent also used as a chemotherapeutic drug. Dosages include: Dogs: 6 mg/m2 PO q48h for 2-4 weeks then taper or 0.25-0.33 mg/kg PO q72h for 2-4 weeks then taper; Cats: 2 mg/cat PO q48h for 2-4 weeks then taper or 2 mg/cat PO q72h for 2-4 weeks then taper. Can be myelosuppressive.

Cyclosporine A – inhibits T cell function; therefore, may be more effective with lymphocytic IBD. Dose: 5 mg/kg PO q24h. May be associated with renal and liver toxicity, myelotoxicity, and increased infections.

Mycophenolate – also a purine analog. Dosage: Dogs: 10-20 mg/kg PO q12h; Cats: 10 mg/kg PO q12h. Seems to be well tolerated. Main side effects are GI signs.

Anti-inflammatories – sulfasalazine, which is salicylate bound to a sulfa antibiotic, is a good anti-inflammatory agent for colitis. The bond prevents metabolism prior to entering the large bowel where bacteria cleave the bond releasing the salicylate to act locally. Sulfasalazine: 20-30 mg/kg q 8-12 h, 10-25 mg/kg q 8 h for 6 weeks, then taper; 10-20 mg/kg po q 8-24 h for up to 10 days (cats). Olsalazine: 10-15 mg/kg q 8-12 h, 5-10 mg/kg q 8 h for 6 weeks, then taper. Side-effects may include KCS due to the sulfa drug

Omega-3 fatty acids – exert anti-inflammatory effects by substituting into cell membranes where metabolism results in cytokines of the odd series. The dose is based on the amount of EPA and DHA in the product not the total amount of omega-3 fatty acids: 300 mg EPA + DHA per 10 pounds PO q24h.

Other treatments: Vitamin B12 (cobalamin): patients with small intestinal disease even if not SIBO may have systemic B12 deficiency. With GI disease, oral replacement is not effective; therefore, parenteral therapy is required. Treatment includes: 1000 mcg SQ q 2-3 weeks, dogs/cats; Cats and dogs <5kg 250 mcg SQ q 7 days for 6 weeks, then q 2 weeks for 6 weeks, then q 4 weeks; Dogs 5-15 kg, 500 mcg/injection; Dogs > 15 kg, 1000mcg/injection

Motility modifiers – Motility disorders occur with GI disease and modification of abnormal motility may help with diarrhea. These are typically opioids. Loperamide (Imodium): 0.1 - 0.2

mg/kg q8 - 12h PO (dog), 0.08 – 0.16 mg/kg q24h PO (cat – cautiously); Diphenoxylate (Lomotil): 0.05 – 0.2 mg/kg q8 - 12h PO (dog), 0.05 – 0.1 mg/kg q12h PO
Anti-emetics – vomiting is often a component of diffuse GI disease and anti-emetics may aid with appetite as well as owner compliance to other treatments. H2 receptor blockers have minimal anti-emetic effect and are used more as antacids. Serotonin antagonists are more potent anti-emetics. Ondansetron (Zofran): 0.5-1 mg/kg PO q12-24h. Dolasetron (Anzemet): 0.5 mg/kg SC, PO q24h. Mirtazapine (Remeron): 15 – 30 mg PO q24h (dog), 1.875 – 3.75 mg PO q72h (cat)

Neoplasia – typically chronic enteropathy caused by neoplasia is a diffuse neoplastic process such as small cell lymphoma. Differentiation of small cell lymphoma from lymphocytic enteritis can be difficult. In one study, histologic examination and genetic testing (PARR) was not able to differentiate these two processes consistently in cats. Although not reliable, thickening of the muscularis layer is seen more often with lymphoma while thickening of the mucosa is seen more often with inflammatory bowel disease on ultrasonographic examination. Treatment is similar, however. Usually glucocorticoids are used first to assess response and chlorambucil is added depending on response or relapse of clinical signs.

WHEN KIDNEYS K'ANT: CHRONIC KIDNEY DISEASE

Joe Bartges, DVM, PhD, DACVIM, DACVN
The University of Georgia, Athens, GA, USA

Overview of the Issue

CKD implies irreversible renal failure that remains stable for a period of time, but ultimately progresses. Although many things can cause chronic kidney disease, by the time chronic kidney disease is diagnosed the cause(s) is/are not present and not treatable. Kidneys are involved with whole body homeostasis; therefore, CKD affects general well-being. CKD is ultimately progressive. The cause(s) of progression of CKD is not completely known. It is likely that in typical situation, CKD results from repeated insults over time that result in sequential loss of nephrons. The compensatory response is an increase in single nephron GFR in the surviving nephrons. This results in maintenance of total GFR despite loss of functional renal tissue (renal reserve). There is dilation of the afferent arteriole and an increase in intragomerular pressure resulting in increase in GFR and renal blood flow. There are trade-offs, however. The increase in GFR due to increase in renal blood flow and intraglomerular pressure increases likelihood of increased protein loss. Increased intraglomerular pressure is transmitted distally. There is activation and release of growth factors that promote tubulointerstitial fibrosis and glomerulosclerosis. Eventually, these adaptations result in loss of further nephrons and the cycle continues. Over time, renal reserve is lost as the threshold of nephron mass loss is surpassed resulting in progression of CKD to end stage

Key Clinical Diagnostic Points

Clinical signs involve primarily change in water balance: polyuria / polydipsia (PU / PD), gastrointestinal signs (vomiting, hyporexia / anorexia, halitosis), and signs of chronic disease (weight loss, loss of body condition, unkempt appearance). Laboratory evaluation may reveal: azotemia, inappropriately dilute urine, hyperphosphatemia, metabolic acidosis, hypokalemia, non-regenerative anemia, bacterial UTI, systemic hypertension (occurs in 65-80% of patients), proteinuria.

International Renal Insufficiency Society (IRIS) Staging: The International Renal Insufficiency Society (http://www.IRIS-kidney.com) has developed staging system for animals with CKD and treatment based on staging. The staging system is designed for use with dogs and cats with CKD. A diagnosis of CKD is made first and staging is accomplished by evaluating. CKD is staged by magnitude of renal dysfunction and further modified (sub-staged) by presence or absence of proteinuria and/or hypertension. Proteinuria ONLY refers to renal proteinuria and not pre-renal (e.g. hyperglobulinemia) or post-renal (e.g. urinary tract infection, hematuria, etc), and is based on UPC.

Stage	Plasma creatinine mg/dl		Comments
	Dogs	Cats	
1	<1.4	<1.6	Non-azotemic Some other renal abnormality present e.g. inadequate concentrating ability without identifiable non-renal cause; abnormal renal palpation and/or abnormal renal imaging findings; proteinuria of renal origin; abnormal renal biopsy results
2	1.4 - 2.0	1.6 - 2.8	Mild renal azotemia [lower end of the range lies within the reference range for many labs but the insensitivity of creatinine as a screening test means that animals with creatinine values close to the upper limit of normality often have excretory failure] Clinical signs usually mild or absent
3	2.1 - 5.0	2.9 – 5.0	Moderate renal azotaemia Many systemic clinical signs may be present
4	>5.0	>5.0	Severe renal azotaemia Many extra-renal clinical signs present

UPC value		Substage
Dogs	Cats	
<0.2	<0.2	Non-proteinuric (NP)
0.2 to 0.5	0.2 to 0.4	Borderline proteinuric (BP)
>0.5	>0.4	Proteinuric (P)

Systolic BP mm Hg	Diastolic BP mm Hg	Adaptation when breed-specific reference range is available *	Substage
<150	<95	<10 mm Hg above reference range	AP0: Minimal Risk (N)
150 – 159	95 - 99	10 – 20 mm Hg above reference range	AP1: Low Risk (L)
160 – 179	100 - 119	20 – 40 mm Hg above reference range	AP2: Moderate Risk (M)
= 180	= 120	= 40 mm Hg above reference range	AP3: High Risk (H)
No evidence of end organ damage/complications			No complications (nc)
Evidence of end organ damage/complications			Complications (c)
Blood pressure not measured			Risk not determined (RND)

Tests of Renal Function

Glomerular filtration rate (GFR) can be estimated using both clearance methods and "spot" or single time point tests. Renal or plasma clearance of an injected substance (e.g., iohexol, creatinine) is most accurate estimate of GFR. It is more sensitive means for detecting early CKD than spot methods of GFR estimation. Determining plasma clearance can be a relatively expensive and time-consuming procedure. It is most often performed to establish a decrease in GFR when clinical parameters (e.g., poorly concentrated urine) create suspicion for CKD but cannot confirm its presence, and to determine dosage regimens for therapeutic agents whose excretion is primarily renal in patients with CKD. Measuring reduction of an injected substance in the blood over time) can be used to estimate renal clearance and therefore GFR. Most common exogenous substances used in veterinary medicine for estimation of GFR are iohexol and creatinine. Other substances and techniques can be used, such as inulin, radiolabeled markers, and contrast-enhanced computed tomography (CT). A novel fluorescent tracer has been evaluated as a rapid, non-invasive bedside test in dogs. Ultimately, choice in method used depends on availability of the injected substance and method of measurement as well as the experience. In some cases, estimation of individual kidney GFR (vs. global GFR) is necessary, as is possible with scintigraphy or CT. Iohexol clearance and exogenous creatinine clearance give a measure of total GFR; DTPA (a radiolabelled marker) gives estimate of total as well as individual kidney GFR. One of the main limitations with clearance methods is need for serial, precisely timed blood draws. An accurate clearance calculation requires as many as 8 post-injection blood samples over 6 hours or longer, although reasonable estimates can be obtained with limited sampling (i.e., 2 or 3 post-injection samples). Timing of these limited sample collections varies depending on the substance used. Some studies have found that calculation of plasma clearance based on a single post-injection sample is strongly correlated with 3-sample techniques, as long as an estimated volume of distribution can be determined. This is especially important in cats, where multiple collections can prove difficult. Another limitation with plasma clearance is the large amount of variability in what is considered to be "normal" in dogs and cats. In one study of 118 healthy dogs, iohexol clearance ranged from 0.95-4.25 mL/min/kg. In previously published studies in healthy dogs and cats, the range for various clearance estimates was as wide as 2.45-6.64 mL/min/kg (dogs) and 2.19-3.49 mL/min/kg (cats), although most weighted reference intervals were around 3-4 mL/min/kg (dogs) and 2.5-3.5 mL/min/kg (cats). Therefore, it is difficult to define a normal GFR in a particular animal without a baseline for that patient, and it limits ability of plasma clearance to detect early reductions in GFR. Week-to-week and month-to-month biological variability must also be considered when monitoring plasma clearance in a particular patient. Based on the week-to-week variability of iohexol clearance in a cohort of dogs with mild but stable renal disease, a subsequent measurement must increase or decrease by up to 20% in order to be 95% confident that a true change in clearance has occurred. Interestingly, despite using more measurements, each with its own inherent variability, iohexol clearance variability was similar to that for serum creatinine (sCr) in these dogs. In addition to biological considerations, analytical considerations in plasma clearance calculations are important. When using a limited sampling technique, a correction formula must be applied to correct for the initial distribution phase in order to avoid overestimation of the GFR. Correction formulas for both dogs and cats are available when using iohexol. Normalization to body weight, surface area, or extracellular volume has been recommended, but it is not clear which normalization technique should be used in dogs and cats.

In patients with CKD1 disease, azotemia is not present and a spot test such as urine specific gravity (USG) may not reflect renal function because it is influenced by non-renal factors. Most adult cats have a USG > 1.035 regardless of time of day whereas adult dogs have variable USG throughout the day. Persistently dilute USG may indicate loss of renal function but other non-renal diseases (e.g. hyperadrenocorticism, diabetes, mellitus,

hyperthyroidism, etc) must be ruled out first. Other biomarkers are being evaluated. Symmetric dimethylarginine (SDMA) test is now provided on all biochemical panel testing through IDEXX Laboratories: https://www.idexx.com/corporate/home.html. SDMA is a small molecule that originates from hydrolysis of methylated proteins. This molecule has shown great promise as an endogenous marker of GFR as it appears to be exclusively eliminated by glomerular filtration, and significant extra-renal influences on its production and elimination have not yet been identified. It is stable in whole blood, serum, and plasma at 4oC and room temperature for up to 7 days, and it is not altered with freezing in serum or plasma. In dogs with rapidly progressing CKD, SDMA correlated strongly with GFR estimated using iohexol clearance. Notably, when using reference intervals, SDMA identified a decrease in GFR earlier than sCr, however, when both were trended over time, no major differences in identification of declining GFR were noted. SDMA changed approximately 9 months earlier than sCr. These results support that trending of sCr is necessary for sensitive detection of decreasing GFR and that SDMA might be a useful adjunct to sCr in identification of renal disease, particularly given the tendency to classify a dog as azotemic or not based on a reference interval. SDMA is not influenced by muscle mass, but any non-renal change in GFR will impact it. For example, with dehydration there is a decrease in GFR and therefore SDMA will also be influenced. It might prove especially useful in the initial diagnosis of CKD in those patients for which sCr will not provide a reliable estimate of GFR. In cats with CKD, SDMA correlates with sCr. While data suggest that SDMA might increase beyond its reference interval before sCr in cats and that a higher SDMA:creatinine ratio might indicate a worse prognosis. Similar to dogs, it is not influenced by lean body mass; however, non-renal factors affecting GFR will impact SDMA. SDMA changed approximately 17 months earlier than sCr.

Management of CKD.

The goal of management is to minimize excesses and deficits induced by CKD in order to improve quality and quantity of patient's life

NUTRITION. The goal of nutritional support is to maintain optimal body condition and lean muscle mass. Anorexia and nausea occur commonly with CKD. Treatment involves feeding a highly palatable diet, modifying feeding patterns, and treating uremic gastroenteritis. Treatment of uremic gastroenteritis involves decreasing dietary protein (stimulates gastric acid production), decreasing gastric acidity with H2 blockers, proton pump inhibitors, and/or sucralfate. Mirtazoamine, a noradrenergic and serotonergic antidepressant stimulates appetite and is an anti-emetic. Maropitant is a neurokinin-1 (NK-1) antagonist that is used for motion sickness and is an anti-emetic. Capromorelin is a growth hormone secretagogue that stimulates appetite in dogs. If necessary feeding tubes may be used to facilitate nutritional and fluid support and provides a means to administer medications. One theory of progression of CKD involves intraglomerular hypertension in the remaining nephrons. This is beneficial in that it keeps GFR up; however, the intraglomerular hypertension may ultimately result in loss of surviving nephrons and progression. Feeding diets or administering omega-3 fatty acids has been shown to be beneficial in dogs by reducing intraglomerular hypertension and inflammation. An omega-6 to omega-3 fatty acid ratio of 3:1 to 5:1 appears to be a reasonable intake and is present in many renal failure diets. Other treatments such as a medicinal rhubarb extract (Rubenal) and a proprietary mixture of amino acids and peptides (RenAvast, AminAvast) have not been shown to be beneficial.

ELECTROLYTES. Hypokalemia may occur especially in cats due to anorexia, excessive losses, transcellular shift due to metabolic acidosis, and activation of the renin-angiotensin-aldosterone system. Clinical signs include polymyopathy, worsening of renal failure, and anorexia. Treatment is aimed at maintaining the serum potassium concentration in the mid to upper half of the reference range. Potassium may be added to IV or SQ fluids or supplemented orally using potassium gluconate or potassium citrate. Potassium citrate provides alkalinization as well as potassium. Changes in serum sodium concentration occur rarely; however, sodium retention occurs with CKD and may result in expansion of extracellular fluid volume and hypertension. There is one study of cats with CKD that documented worsening azotemia with increased dietary sodium intake; however, other studies have not shown this and dietary sodium has not been shown to be correlated with hypertension.

PH OF BLOOD (ACID-BASE STATUS). Metabolic acidosis occurs commonly with CKD because of retention of organic acids, decreased renal ability to regenerate and reclaim bicarbonate, decreased ammoniagenesis (ammonia is a buffer and is renally excreted with acid), and generation of acids from catabolism. Treatment involves feeding a diet that is low protein = as dietary protein is a main source of organic acids and alkalinizing (most contain potassium citrate).

PROTEINURIA. Proteinuria is not just a marker of glomerular disease but is also associated with progression of CKD as it stimulates renal fibrosis and activates inflammation. Treatment is indicated with IRIS CKD stage 1 and UPC > 1.0 to 2.0 and IRIS CKD stages 2-4 when UPC > 0.4 in cats and 0.5 in dogs. Treatment involves feeding a renal diet, administering an ACE-I and/or ARB, and omega-3 fatty acids.

HYDRATION. Polyuria due to CKD is offset by compensatory polydipsia but dehydration may occur if this is inadequate. Provide clean and fresh water daily. Supplemental SQ fluids may be administered if needed, which appears to be more common in cats than in dogs. In a hospital situation, IV fluids should be administered.

RETENTION OF WASTES. Elimination of wastes particularly nitrogen-containing compounds is an important function of the kidneys. Reduction of dietary protein seems logical but results of studies are contradictory as to whether dietary protein restriction alters progression of CKD. Dietary protein restriction may be associated with: decreased azotemia, decreased hyperphosphatemia, decreased metabolic acidosis, and decreased gastric acid secreation. Three studies, two in cats and one in dogs, of spontaneously occurring CKD, demonstrated a beneficial effect from feeding a renal failure diet when compared with feeding a maintenance diet. Level of dietary protein found in renal failure diets is adequate for maintenance of adult animals is not likely to be associated with protein malnutrition

Prebiotics: Feeding diets that contain soluble fiber may redistribute a small amount of nitrogen into the gut for elimination thus decreasing the amount required by the kidneys to eliminate (“nitrogen trapping”)

Probiotics: involve administering live bacteria. One formulation, Azodyl, is marketed as “enteric dialysis”. In one study of cats with CKD, there was no benefit and administration of Azodyl was not associated with decreasing the degree of azotemia.

OTHER RENAL INSULTS – AVOID. Dehydration may precipitate an acute renal failure episode making the chronic kidney disease worse. Certain situations and drugs may be directly nephrotoxic or may worsen renal failure including: aminoglycosides, urinary acidifiers, catabolic drugs (e.g. immunosuppressive drugs), and non-steroidal anti-inflammatory drugs, and UTI. NSAIDs may be nephrotoxic if given in high enough dose but at low doses may be beneficial in CKD by decreasing inflammation while maintaining renal vasodilation.

NEUROENDOCRINE FUNCTION. There are 3 neuroendocrine changes occurring with CKD, renal secondary hyperparathyroidism, hypoproliferative anemia, and systemic arterial hypertension.

Mineral and bone disorder in CKD (MBDCKD) MBDCKD occurs, in part, because of phosphorous retention and decreased calcitriol (vitamin D3) metabolism by the failing kidneys. Hyperphosphatemia may result in renal mineralization and loss of nephrons. Hyperphosphatemia is associated with progression of chronic kidney disease and of shortened survival. Treatment is decrease serum phosphorous concentration to normal. Serum phosphorous concentration may be decreased by: feeding a low phosphorous diet, administering phosphate binders with food (e.g. aluminum hydroxide, calcium acetate, sevelamer hydrochloride, lanthanum carbonate, or chitosan + calcium carbonate), and/or administering vitamin D. When administering vitamin D, dietary phosphorous should be restricted and serum phosphorous concentration should be normalized because of risk of hypercalcemia and increasing calcium x phosphorous solubility product. To date, only dogs in stage III or IV IRIS benefit from calcitriol therapy; however, no study has documented benefit of vitamin D in cats with any stage.

Hypoproliferative anemia. Normocytic, normochromic non-regenerative anemia occurs in many animals with chronic kidney disease. It may induce progression of disease due to decreased blood flow, stagnation of blood, oxidative stress, decreased oxygen diffusion, and induction of fibrosis. Causes of anemia include decreased erythropoietin production, nutritional imbalances, and blood loss due to uremic gastroenteritis. Treatment includes maintaining a good nutritional status, minimizing GI blood low, and stimulating red blood cell production. While anabolic steroids have been used, they are associated with heptatoxicity. Recombinant human erythropoietin (rHuEPO) and its synthetic analog darbepoetin have been used successfully in dogs and cats with chronic kidney disease that are severely anemic. Many patients receiving rHuEPO feel better even if their anemia does not improve. Darbepoetin may be associated with fewer incidence of antibody production and is administered weekly and is the hormone replacement of choice. Darbepoietin should be started when even mild anemia is present with the goal of hormone replacement therapy being a PCV of 35-40%. Supplemental iron dextran should be given.

Systemic arterial hypertension (SAH). SAH occurs commonly and is due, in part, to activation of RAAS, activation of sympathetic nervous system, increased ADH due to hypovolemia. End-target organ damage due to SAH include eyes (retinal hemorrhage and detachment, blindness), kidneys (proteinuria, progression), heart (left ventricular hypertrophy), and brain (encephalopathy, seizures, death). The greater the degree of SAH the higher the

likelihood of hypertensive-related complications. The goal of treatment is a systolic blood pressure (sBP) < 150 mmHg. Treatment involves feeding a renal diet and administering anti-hypertensive drugs. Calcium channel blockers are most effective and lower sBP by an average of 50 mmHg. ACE-I are less effective and lower sBP by an average of 10 mmHg but are more effective for treating proteinuria. Benazapril does not slow progression in animals with CKD unless UPC is > 1. Angiotensin receptor blockers (ARB) may be used alone or in conjunction with an ACE-I and/or CCB.

OTHER POTENTIAL TREATMENTS. Renal transplantation has had success in cats and dogs but more so in cats. In one study, 50% survival time was over 500 days. Intermittent hemodialysis may be performed in patients with IRIS CKD stage 4 disease. Mesenchymal stem cells (MSCs) have been proposed as a novel treatment option for the management of CKD although there are no conclusive data.

Serial monitoring.

Chronic kidney disease is progressive and thus a dynamic disease. Serial monitoring of body condition, body weight, thoracic auscultation, blood pressure, CBC and serum biochemical profile, urinalysis, and urine culture are necessary to adjust treatment. Dietary modification can offset many deficiencies and excesses that occur with chronic kidney disease. Dietary modification includes more than just dietary protein restriction as renal failure diets are more calorically dense, may contain omega-3 fatty acids, may contain soluble fiber, low phosphorous, low sodium, potassium replete, alkalinizing, and water soluble vitamin replete

Management of IRIS CKD stage 1 patients

Most publications discuss management of IRIS CKD2-4, where therapy has been shown to improve survival and quality of life in dogs and cats. There is minimal information on prognosis and management of patients diagnosed with IRIS CKD1, and most information applies to patients with proteinuric CKD1 disease.

PROTEINURIC PATIENTS

Overt glomerular proteinuria occurs more commonly in dogs than in cats and is defined as a urine protein creatinine ratio (UPC) > 2.0 that is renal in origin. Approximately 50% of dogs with glomerular proteinuria have an immune-mediated disease either as a primary process or secondary to chronic antigenic stimulation (e.g. infections, inflammatory, or neoplastic disease). Treatment involves achieving a diagnosis of the underlying cause, if possible, and treating it, if possible. Other treatment involves feeding a protein restricted diet (therapeutic renal diet), giving omega-3 fatty acids (300 mg of sum of EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid) per 10 pounds of body weight per day), and pharmacologic agents that inhibit the renin-angiotensin-aldosterone system (RAAS). Conventionally, an angiotensin converting enzyme inhibitor (ACEI) is often used and has the most, albeit, limited data. Enalapril has been evaluated in dogs with glomerular proteinuria and was shown to decrease the degree of proteinuria and increase the serum albumin concentration. Benazepril is often used instead of Enalapril; however, there is one study in dogs comparing Enalapril with benazepril in dogs with CKD and proteinuria and Enalapril gave a more sustained anti-proteinuric effect over 5 months. Angiotensin receptor blockers (ARB) may be used in combination with or instead of ACEI although there is minimal information available. In human beings, use of an ACEI with an ARB provides better proteinuria lowering response than either alone. This has not been evaluated in dogs. While Losartan is the oldest of the ARBs, newer ARBs, such as Telmisartan and Irbesartan, have more selectivity for the angiotensin receptor and may have a greater effect. With any RAAS inhibitor potential adverse events include azotemia, hyperkalemia, and GI disturbances. ACEI are usually administered every 12 hours while ARB are usually administered every 24 hours. Dogs with glomerular proteinuria are often administered drugs that inhibit platelet aggregation (e.g. aspirin or clopidogrel). In dogs with a diagnosis of an immune mediated disease by renal biopsy, in dogs with suspected immune-mediated glomerulonephritis, or in dogs that do not respond well to conventional therapy, immunosuppression should be undertaken. Which imusuppressive drug is best is not known. None have been shown to be effective in controlled studies, although there are sporadic case reports of response. Studies in dogs have shown that in most cases of proteinuria, glucocorticoid administration is not beneficial and is often associated with a worsening of the proteinuria. Glucocorticoids appear to promote glomerulosclerosis and intraglomerular hypertension. Therefore, glucocorticoids are not recommended unless the proteinuria is secondary to glucocorticoid-responsive systemic disease. Often glucocorticoids are given with severe glomerular proteinuria in order to achieve quicker control while other imunsuppressive drugs are initiated. Cyclosporine was not found to be effective in dogs with idiopathic

GN in a controlled, blinded study. Other immunosuppressive drugs that may show benefit, but that have not been evaluated in placebo-controlled, blinded studies are azathioprine (2 mg/kg PO q24h x 2 weeks, then 1 mg/kg PO q24h, then 1 mg/kg PO q48h), cyclophosphamide (50 mg/m2 PO q48h), and chlorambucil (2-6 mg/m2 PO q24-48h). The most promising is mycophenolate (20 mg/kg PO q12h for 3-4 weeks, then 10 mg/kg PO q12h). The decision to use immunosuppressive therapy should be based on the likelihood of an immune-mediated cause of proteinuria, the patient's overall condition, and the ability to monitor the patient. Consider diuretics to decrease sodium retention and edema/ascites. In human beings with nephrotic syndrome, diuretics are often used to decrease ascites/edema. Commonly a combination of a loop diuretic (such as furosemide) and a thiazide diuretic (such as hydrochlorothiazide) are used. Furosemide is often used in veterinary medicine to decrease fluid retention and should be considered in dogs or cats that have nephrotic syndrome. Combination diuretic therapy may be considered in animals that are refractory to single agent therapy.

NONPROTEINURIC PATIENTS

Management of patients with CKD1 without proteinuria is less clear. A decrease in renal function without a progressive underlying disease may not warrant intervention. At present there is no means of predicting progression of CKD1 disease other than monitoring trends and/or identifying and treating the underlying cause if possible. Dogs and cats with congenital renal disease may or may not have progressive disease. For example, dogs with unilateral renal agenesis may not experience progressive CKD if the solitary kidney is otherwise structurally and functionally normal. On the other hand, patients with unilateral renal lymphoma often develop it in both kidneys and so CKD is progressive. Dogs and cats with Fanconi syndrome are often diagnosed as CKD1; however, this is usually progressive disease. The question is in patients without identifiable specific diseases does a diagnosis of CKD1 warrant intervention? There are 3 studies in dogs and 2 studies in cats that have been published. In a study of 210 client-owned dogs with normal creatinine concentrations, 18 dogs had an increased SDMA at baseline or during a 6 month crossover dietary study. Feeding a heat processed dry food containing fruits and vegetables, egg protein, wet chicken meat, lipoic acid, α-tocopherol, vitamin C, carnitine, and omega-3 fatty acids compared with a control was associated with a decrease in SDMA in 8 of 9 dogs compared with 4 of 9 dogs when consuming the control diet. A study of 81 geriatric research dogs were compared with 30 mature adult research dogs consuming a control diet and 2 foods that were similar to the aforementioned diet; however, one of the test diets contained additional fruits and vegetables and egg protein and did not contain corn gluten meal.(9) The geriatric dogs had lower GFR than the mature dogs. GFR increased and SDMA decreased in geriatric dogs when fed the test foods; however, the effect was greater in the geriatric dogs eating the second test diet. None of the dogs had GFR or SDMA values outside of the normal range, however. A longitudinal study of 36 client-owned dogs with CKD1 diagnosed by having abnormal kidneys, dilute urine of renal origin, proteinuria of renal origin, or combinations evaluated the influence of consuming a therapeutic renal diet over a 3 to 12 month period. Of the 36 dogs, 20 had dilute urine, 6 had persistent proteinuria, and 10 had both; 50% had an elevated SDMA at baseline. Serum creatinine, BUN, and SDMA decreased from baseline to 3 months and remained decreased from baseline in the 20 dogs that completed the 12 month study. Proteinuria was reduced in 12 of 16 dogs with proteinuria. Thirty-two geriatric research cats were fed two diets containing low phosphorous and protein with added omega-3 fatty acids, and L-carnitine; the second test diet contained median chain triglycerides. Over a 6 month feeding period, SDMA decreased slightly, but not significantly and GFR increased in cats fed the second test diet but decreased in the cats fed the first test diet; however, results were not significantly different from baseline. A study of client-owned cats has also been published. Cats consuming owner's choice foods showed significant increase in SDMA at 3 and 6 nonths whereas in cats consuming a test diet (described above), SDMA did not change. During the study 23 cats had an increased SDMA including 17 cats in the owner's choice food group and 6 in the test diet group. In the 6 cats fed the test food, SDMA decreased or remained stable in 4 cats, and increased in 2 cats, whereas in the 17 cats in the owner's choice food group, SDMA increased in 13 cats and decreased or remained stable in 4 cats. Results of these studies suggest that nutritional intervention may be beneficial in some older dogs and cats with CKD1 and that SDMA is a useful biomarker; however, there is variability in SDMA and trends in changes in SDMA may be more important in managing patients with CKD.

Drugs used to managed dogs and cats with CKD

Class	Drug	Dosage for dogs (D) or cats (C)
H2 blocker	Famotidine	D, C: 1-2 mg/kg PO q12h
	Ranitidine	D, C: 1-2 mg/kg PO q12h
Gastroprotectant	Sucralfate	D: 0.5-1 gm PO q8-12h; C: 0.25-0.5 gm PO q8-12h
Proton pump inhibitor	Omeprazole	D, C: 0.7-2 mg/kg PO q12-24hr
	Esomeprazole	D, C: 0.7 mg/kg PO q12-24hr
Serotonin antagonist	Mirtazapine	D: 15-30 mg PO q24h; C: 1.875-3.75 mg PO q72h- can give q48h with CKD
	Ondansetron	D, C: 1) 0.5 mg/kg IV; then 0.5 mg/kg/hr constant rate infusion 2) 0.1-0.2 mg/kg IV slowly q6-12h prn 3) 0.5-1 mg/kg PO q12-24h
	Dolasetron	D, C: 0.6-1 mg/ kg PO, IV q12-24h
NK-1 inhibitor	Maropitant	D, C: 2-4 mg/kg PO q24h
PGE2 analogue	Misoprostol	D: 2-7.5 mcg/kg PO q8-12hr; C: 5 mcg/kg PO q8hr
Medicine rhubarb	Rubenal	D: < 3kg: 37.5 mg; 3-6kg: 150 mg: 6-12kg: 150 mg; 13-25kg: 300mg; 26-45kg: 600mg; >45kg: 900 mg PO q12h C: <2kg: 37.5mg; >3kg: 75mg PO q12h
Amino acids / peptides	RenAvast, AminAvast	C: 1 capsule with food
Potassium	Potassium citrate	D, C: initial: 75 mg/kg PO q12h
Probiotics	Azodyl	D, C: < 2.5kg: 1 capsule PO q24h; 2.5-4.5 kg: 1 capsule PO q12h; > 4.5kg: 2 capsules PO in AM and 1 capsule PO in PM with food
	Visbiome	D, C: 100 billion organisms/kg q24h
Phosphate binder	Aluminum hydroxide	D, C: 15-45 mg/kg PO q12h with food
	Calcium acetate	D, C: 60-90 mg/kg PO q12h with food
	Sevelamer hydrochloride	D, C: 400-1600 mg PO q12h with food
	Lanthanum carbonate	D: 5-20 mg/kg PO q12h C: 1 ml (1 pump) PO q12h (Renalzin)
	Chitosan + calcium carbonate	D, C: 1 g/kg PO q12h 3-5kg: 1 scoop; 10kg: 2 scoops; 15kg: 3 scoops; 20kg: 4 scoops PO q12h (Ipakitine)
Vitamin D	Calcitriol	D, C: initial:2-2.5 ng/kg PO q24h; maximum: 5 ng/kg PO q24h Alternatively, 9-12 ng/kg PO q3.5d
Erythropoietin	Erythropoietin	D, C: 100 ug/kg SQ 3X/week initially
	Darbepoetin	D, C: 1.5-1.0 ug/kg SQ 1X/week initially
Calcium channel blocker	Amlodipine	D: 0.1-0.4 mg/kg PO q24h; C: 0.625-1.25 mg PO q24h
ACE-I	Enalapril	D, C: 0.25 mg/kg PO q12h initially
	Benazepril	D, C: 0.25 mg/kg PO q12h initially
Angiotensin receptor blocker	Losartan Azilsartan Irbesartan Telmisartan Valsartan	D, C: 1 mg/kg PO q12h D: 0.1-1.0 mg/kg PO q12h D: 5 mg/kg q12-24h D, C: 1 mg/kg PO q24h D: 80-160 mg PO q24h
Aldosterone receptor blocker	Spironolactone	D, C: 1-4 mg/kg PO q12h-24h

Summary including KEY "TAKE HOME" POINTS

1. CKD occurs commonly and is progressive over some length of time in dogs and cats
2. Early diagnosis may aid in early intervention with potential of slowing progression
3. Treatment is aimed at correcting excesses and deficiencies induced by CKD including nutrition,, electrolytes, acid-base, proteinuria, hydration, and neuroendocrine imbalances
4. Serial monitoring is vital to successful management of patients with CKD

Summary

CKD implies irreversible renal failure that remains stable for a period of time, but ultimately progresses. Although many things can cause chronic kidney disease, by the time chronic kidney disease is diagnosed the cause(s) is/are not present and not treatable. Treatment is aimed at correcting excesses and deficiencies induced by CKD including nutrition, electrolytes, acid-base, proteinuria, hydration, and neuroendocrine imbalances. Serial monitoring is important and aids in altering treatment in order to maximize response.

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THE GOOD, THE BAD, AND THE UGLY OF PET NUTRITION
What we know, what we think we know, what we thought we knew, what we don't know

Joe Bartges, DVM, PhD, DACVIM, DACVN
Professor of Medicine and Nutrition
The University of Georgia
jbartges@uga.edu

NOTE: The majority of these notes is based primarily on a very good, scientifically sound review article[1] and not on a more popular opinion-based article.[2]

Veterinarians, veterinary technicians, and the general public are inundated with information, some accurate and some not, on pet nutrition. Put in "dog nutrition" or "cat nutrition" in Google, and you get 13- 17,000,000 hits. Add to this, articles and books in print, advice from neighbors and store employees, and commercials on TV and in print, and the information is overwhelming. Discussing pet nutrition is sometimes akin to discussing religion or politics; people are passionate about their beliefs. There are many "fallacies and facts" that could be discussed; however, I will focus only on a few.

Despite advances in pet nutrition, one of the more popular 'conspiracy theories' is that pet food companies are poisoning pets. It makes no sense from a business perspective for a business to produce a product that would be detrimental to its consumers. Unfortunately, pet food toxicities and imbalances have and continue to make the news. Recalls for bacterial and aflatoxin contamination as well as vitamin-mineral imbalances such as thiamine deficiency and vitamin D excess exist, which heightens unease amongst pet owners. Distrust in veterinarians and veterinary health professionals results in owners acquiring information from other and often less knowledgeable sources. The perception is that veterinarians are loyal to larger companies because they make money on selling their food, are provided perks by these companies, and that nutritional education was provided by representatives of large companies. Unfortunately, some of this is not incorrect.

DIET-ASSOCIATED DCM
INTRODUCTION

In July 2018, the FDA issued a statement relating dilated cardiomyopathy (DCM) in dogs to consumption of certain diets that have pulse ingredients and potatoes as main ingredients. Pulse ingredients are a subset of legumes harvested as a dry crop with low concentrations of lipid. They are fine powders created from fractions of peas, lentils dry beans, and chickpeas, and made without the use of processing aids or chemical compounds.[3] They are used in food and feed-grade products and are marketed as offering natural solutions to increase the nutritional value of foods without altering flavor, aroma and color properties, and they have been used as ingredients in dog food for their protein and fiber for over 20 years.[4]

The FDA release stated:

> "*We are concerned about reports of canine heart disease, known as dilated cardiomyopathy (DCM), in dogs that ate certain pet foods containing peas, lentils, other legumes or potatoes as their main ingredients. These reports are highly unusual as they are occurring in breeds not typically genetically prone to the disease," said Martine Hartogensis, D.V.M., deputy director of the FDA's Center for Veterinary Medicine's Office of Surveillance and Compliance.* ***"The FDA is investigating the potential link between DCM and these foods****. We encourage pet owners and veterinarians to report DCM cases in dogs who are not predisposed to the disease*." – FDA, July 2018

The media attention this received resulted in what could be described as widespread panic (with respect to Athens' own rock band) and denouncing of all grain-free diets. The original concern was presumed to be DCM occurring secondary to low circulating taurine concentrations as had been found previously.[5,6] This was, in part, based on the relationship of low circulating taurine with dilated cardiomyopathy in cats,[7,8] and in part from the fact that pulses are generally high in lysine and low in methionine. The result of this speculation was an increased need for dietary taurine or its precursor methionine due to higher gastrointestinal fermentation of taurine and thus greater fecal excretion with higher dietary fiber intake.[9,10] Whether this has any link to dietary pulses or inclusion of pulses in grain-free dog food has not been proven. In one 24-week study evaluating graded concentrations of soybean meal up to 17% (as-fed basis) in dog foods, nutrient status of dogs was unaffected;[11] however, in another study, inclusion of more

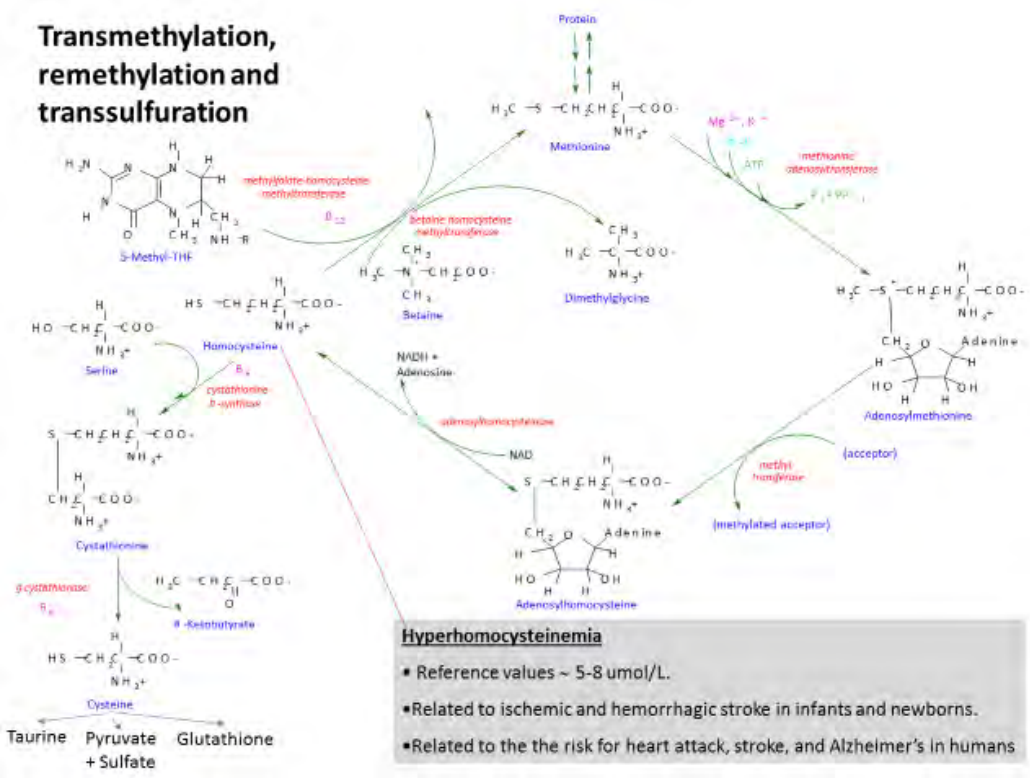
than 15% soybean meal (dry-matter basis) decreased crude protein digestibililty.[12] It is possible, that some dog foods exceed this at levels (more than 40%) resulting in taurine and/or methionine deficiency; however, these high levels have not been investigated. As dogs often are fed a single diet for extended periods of time, consumption of a diet that is marginally replete or deficient in one or more nutrients due to formulation may result in clinical disease over time.

Keep in mind that nutrient profiles established by the Association of American Feed Control Officials (AAFCO) are above known minimal requirements often using purified diets as found in publications by the National Research Council (NRC), but still represent minimal nutritional requirements that pet food companies must meet in order a diet to be considered "complete and balanced" and "providing adequate nutrition when fed as the sole source of nutrition" to dogs or cats in one or more life stages. Also, feeding trials are relatively short when compared to the length of time a dog may consume a diet. For example, an adult dog feeding trial lasts 6 months and requires minimal evaluations. Furthermore, the lead product in a family line of products may undergo feed trial testing; however, the other products in the family as long as they are not substantially different from the lead product may carry a feeding trial nutritional adequacy claim but would not have actually been through an AAFCO feeding trial. Thus, an AAFCO feeding trial nutritional adequacy claim would not likely detect marginal deficiencies (or toxicities).

DILATED CARDIOMYOPATHY AND LINK TO TAURINE DEFICIENCY

Dilated cardiomyopathy is a disease characterized by increased chamber diameter and possible arrhythmias as well as sudden death. Development appears to be slow and clinical signs may be non-existent during development, progressing to lethargy, anorexia, respiratory effort, cyanosis, and syncope, and possibly congestive failure (ascites, pleural effusion, or pulmonary edema) and death, which may be the only clinical sign. There is a predisposition in certain breeds, primarily larger breeds of dogs, and there are known genetic mutations in some dog breeds. Doberman pinschers, Boxers, Great Danes, Newfoundlands, Irish Woflhounds, English and American Cocker Spaniels, Golden Retrievers, and Portuguese Water Dogs have a high prevalence. When dogs who are not known to be genetically predisposed are diagnosed then diet and physiology or other factors are thought to be associated with the disease.

The first link between taurine deficiency and DCM was demonstrated in cats, which was shown to be reversible with supplementation.[8] In dogs, DCM diagnoses related to low whole blood taurine concentrations have been reported in Cocker Spaniels, Dalmatians, Boxers, Newfoundlands, Portuguese Water Dogs, English Setters, Alaskan Malamutes, and Scottish Terriers.[5,6,13-16] Supplementation with taurine improved cardiac function. Interestingly, when compared with cats, dogs can endogenously synthesis taurine from methionine and cysteine.[1]



These studies do not establish that decreased taurine intake is the mechanism for development of DCM. Dietary supply of precursor AAs necessary for taurine synthesis (i.e., methionine and cysteine), metabolic intermediates, and cofactors (such as methyl donors) cannot be ruled out as factors that contribute to the susceptibility of dogs to developing genetic and diet-related DCM. When DCM is diet-related, the formulation and the provision of all nutrients, including indispensable AAs, to facilitate optimum health and wellbeing of dogs should be considered.

The FDA report suggests canine DCM in dogs that are not genetically predisposed is related to consumption of legumes; however, this is at present unproven. Dogs have no minimum or maximum requirement for ingredients and ingredients provide nutrients, which do have such requirements. Thus, animals have nutrient requirements and not ingredient requirements. In diets that have nutrient deficits, imbalances, or exceed maximums, the final nutrient composition of the diet, not the ingredients, should be critiqued. In addition, animal nutritionists should consider that the nutrient concentration of ingredients can vary, nutrient availability is not 100%, and diets formulated to marginally meet requirements could actually be deficient.

Taurine is a dispensable and not essential amino acid in dogs and is synthesized from methionine and cysteine (see figure above). Taurine is a beta-sulfonic amino acid and is not incorporated into proteins. It is used as a mediator for various biological processes and is the most abundant intracellular amino acid. In the heart, taurine represents @ 60% of the total free amino acid pool. This may explain the association of taurine deficiency with DCM. Taurine may contribute to the reabsorption of calcium by the sarcoplasmic reticulum and increase the sensitivity of the myofilaments to calcium.[17] Thus, low dietary taurine intake and/or reduced synthesis of taurine from methionine and cysteine can deplete calcium pools in the cardiac cells and impede proper contraction of the cardiac muscle tissue, resulting in DCM in dogs.

There are no recommendations on minimum dietary concentrations of taurine for dogs reported by the NRC or AAFCO for dogs. The lack of regulation on minimum taurine concentrations in commercial dog foods suggests that endogenous synthesis of taurine can meet the metabolic needs in all dogs and at all life stages. This assumption may not be accurate as studies have determined that synthesis of taurine is related to the size of dog. [18] Thus, larger dogs may be at higher risk for intracellular taurine depletion. Additionally, lower blood taurine concentrations in rats in humans is associated with obesity, and nearly half of the dogs in North America are overweight or obese.[19] Obese dogs may have an increased

requirement for sulfur containing amino acids necessary for endogenous taurine synthesis. Age and gender may also play a role and it has been shown that male rats have higher cysteine sulfonate decarboxylase activity, the enzyme responsible for taurine synthesis, and kittens have higher activity of this enzyme compared with young adult cats; this has not been evaluated in dogs. Overall, these studies suggest that, despite some capacity for endogenous synthesis, physiological need of taurine can be heavily dependent on breed, age, sex, and physiological status. These physiological factors could help us to predict the risk for developing DCM when genotypic and environmental factors, such as diet, are simultaneously considered to ensure that dogs maintain adequate concentrations of taurine and other sulfur amino acids.

Whole blood samples, and not plasma samples, should be used to assess circulating taurine concentrations. In plasma, free taurine concentrations are much lower compared with intracellular taurine. This suggests that the plasma pool is not representative of taurine in other pools. In platelets, taurine concentration is high and is considered a marker of taurine status. Taurine concentration in platelets is captured when whole blood is analyzed. However, platelet count may vary and affect whole blood taurine concentration. Thus, whole blood taurine concentration likely does not totally reflect intracellular, particularly cardiac myocyte, concentrations.

Given that there are no recommendations for the minimum concentration of taurine in dog food, the concentration of taurine in dog foods can vary substantially depending on the ingredients used. Taurine is very low in plant-based ingredients but is higher in some algae and fungi species and is found ubiquitously in animal tissues, especially in the heart, brain, and white blood cells. Animal by-product is actually high in taurine relative to other sources. Many grain-free and/or legume-based dog foods limit use of animal by-products. In the context of providing adequate and preventive nutrition, dog foods should include organ meat or animal byproducts or be fortified with taurine and/or its precursors (methionine and/or cysteine) to ensure the delivery of sufficient levels of taurine.

Dietary fiber has been shown to affect the taurine status in dogs. For example, commercial diets formulated with lamb meal and rice bran were shown to cause taurine deficiency in part because of low bioavailable cysteine from lamb meal and possibly more importantly due to the effects of rice bran fiber on gastrointestinal metabolism of taurine.[20,21] It has been hypothesized that high-fiber diets can increase susceptibility to taurine deficiency by 2 mechanisms of action linked to obligatory bile acid conjugation with taurine in dogs and reliance on enterohepatic circulation for the reabsorption of bile acids and taurine. First, high-fiber diets may increase fecal output and losses of taurine-conjugated bile. This would require higher synthesis rates of bile in the liver, and consequently, higher utilization of taurine.[22] Second, high consumption of fermentable fibers may increase the abundance of microbial populations that degrade taurine in the intestinal lumen. Either alone or together, increased excretion or degradation of taurine from high-fiber diets may decrease enterohepatic circulation and recycling of taurine. Given that taurine is the only amino acid used for bile acid conjugation in dogs, over time, high-fiber diets could increase the risk of taurine insufficiency in dogs and lead to DCM. Dog foods with high concentrations of dietary fiber should be accompanied by higher supplies of taurine or sulfur AAs for endogenous taurine synthesis. Overall, the digestibility and bioavailability of taurine in ingredients used and the effect of other nutrients in taurine metabolism should be considered to avoid taurine deficiency and the development of DCM.

Although taurine is considered a dispensable amino acid in dogs, endogenous taurine synthesis requires an adequate supply of bioavailable sulfur AA precursors cysteine or methionine (Figure). Thus, providing marginal concentrations of these 2 sulfur amino acids, or providing sources with lower bioavailability, could increase the risk of taurine deficiency and facilitate the development of DCM. Contrary to taurine, methionine cannot be synthesized endogenously in dogs. Therefore, dogs depend on the provision of dietary methionine to meet daily sulfur amino acid requirements, which includes production of taurine. From an ingredient perspective, methionine and lysine are usually the first or second limiting amino acids in dog diets formulated with soybean meal and rendered meats. In addition, methionine is particularly susceptible to damage, and subsequent reduction in bioavailability, secondary to heat processing.

Just as understanding the inherent nutritional characteristics and the interaction between ingredients is important for preventing nutritional imbalances in pet foods, the effects of processing on these factors are equally important. Raw cereals and legumes contain antinutritional factors such as trypsin inhibitors, phytates, hematoglutinins, and polyphenols that can decrease protein digestion, nutrient absorption, and/or cause illness. Some of these antinutritional factors are thermolabile and, under the right conditions, can be effectively destroyed during the extrusion process improving the overall quality of plant-based ingredients and the final diet. Recent reviews across a variety of legumes and

legume-derived ingredients show that the activities of trypsin inhibitor, chymotrypsin inhibitor, and hemagglutinating activity were decreased by up to 95% across a variety of thermal treatment conditions, including extrusion.

It is important to mention that, while temperature and pressure processing can greatly decrease antinutritional factors, they can also negatively affect bioavailability of AAs. The Maillard reaction is a well-known example of heat-damaged protein.[23] In this reaction, lysine interacts with reducing sugars present in the diets forming the Maillard product. The complex formed can be digested and absorbed by the animal but cannot be utilized for metabolic processes (e.g., protein synthesis). Thus, in heat-damaged proteins, digestibility of AAs can greatly overestimate bioavailability.

CARNITINE DEFICIENCY AND RISK OF DCM

Carnitine is not nutritionally indispensable since it is endogenously produced in the liver and kidneys from lysine and methionine; it can also be attained exogenously from animal-based products. Carnitine is highly abundant in skeletal and cardiac muscles. Together, these represent >95% of the total carnitine in the body. Carnitine is essential for metabolism of fatty acids used for energy production.[24] In the heart, where 60% of the energy is derived from fatty acid oxidation, carnitine facilitates the uptake of free fatty acids into the mitochondria to produce ATP. Plant-based ingredients do not contain carnitine. Therefore, in commercial dog foods with reduced inclusion of animal-based ingredients, intakes of carnitine could be decreased if diets are not fortified. Reduced dietary carnitine intake translates into increased reliance on endogenous synthesis to meet physiological requirements.

Given that carnitine is required for sufficient energy production in cardiac muscle, it is not surprising that carnitine deficiency is associated with DCM. In 1991, a family of Boxers diagnosed with DCM were also diagnosed with carnitine deficiency.[25] However, carnitine deficiency as a causative factor in the development of DCM or a consequence of cardiac malfunction remains as a subject of debate. Despite the interest in this metabolite, little progress has been made on determining the effect of carnitine supplementation on alleviating risk of DCM. However, both taurine and carnitine are often supplemented in supraphysiological concentrations once DCM is diagnosed. This practice is supported by positive clinical outcomes, albeit without comparison groups.[14,26] Concentrations of carnitine in the plasma are relatively insensitive to dietary carnitine, and more invasive techniques (biopsies) are required to determine the concentration of carnitine in muscle tissue.[25] The invasive nature of testing for carnitine status is likely the reason why carnitine is rarely explored when investigating possible causes of canine DCM.

WHAT EVIDENCE IS THERE?

Several recent papers, both original research and reviews, likewise highlight the unknowns surrounding grain-free diets (typically legume or pulse-based, but sometimes also with “exotic” ingredients such as kangaroo, bison, or wild boar) and DCM.

One study examined 91 dogs of which 48 dogs of many breeds were included while 43 were excluded (including dogs eating vegan or vegetarian diets). These dogs were diagnosed with DCM and had a known diet history.[27] Twelve of the included dogs were eating grain based diets (2 were of ‘unusual breeds’) at the time of DCM diagnosis, and 36 were eating grain free diets (5 were of ‘unusual breeds’). Of the grain free dogs, 14 were eating one specific grain free diet, and 22 were eating other grain free diets. There were two pairs of unrelated housemates included (both eating the one specific grain free diet, which was California Natural owned by Mars PetCare) and one pair of related housemate dogs included in the grain free group. Seven brands of grain based diets were represented (Pedigree (Mars), Purina (NF, Maintenance, Beneful, ProPlan), Iams (maintenance, intestinal), Royal Canin (hydrolyzed protein, rabbit and potato, duck and potato, adult mini), and Science Diet (advanced fitness adult), and 12 brands of grain free diets were represented (NutriSource, 4Health, Acana, Blue Buffalo (intestinal, Wilderness, Basics), Zignature, Earthborne Holistic, Whole Earth Farms, Fromm, Iams, and Science Diet). The number of dogs eating each grain based diet brand ranged from 1 to 3. The number of dogs eating each grain free diet brand ranged from 1 to 5 for other grain free diets with 14 dogs eating one specific grain free diet. A diet change from grain free to grain based diets manufactured by a major brand pet food company with veterinary nutritionists on staff was consistently recommended for all dogs in the grain free group after June 2017 but was not recommended for the grain based group and inconsistently recommended for the grain free group before this time. Two dogs in the GF group were switched to a major brand food that was grain free. All but one dog in the grain free group received supplementation with taurine (30 mg/kg twice daily) after diagnosis and diet change, even if whole blood taurine

concentrations were within or above the reference range. Interestingly, only 2 dogs had low blood taurine concentrations and they were consuming grain based diets. Two dogs switched from that diet to other grain free diets showed improvement in their DCM. This suggests that grain-free composition per se may not be the root cause of DCM.

Another recently published case series of 24 Golden Retrievers with DCM and known diet histories were evaluated, and an association between grain-free diets and DCM was suggested.[28] Client-owned golden retrievers with documented blood taurine deficiency and DCM met inclusion criteria and were enrolled in a multicenter, prospective, observational study to evaluate dietary factors that may contribute to this condition as well as describe their clinical response to treatment. Echocardiography was performed by a board-certified cardiologist. A total of 40 dogs were considered for study inclusion. Sixteen dogs were excluded from the study due to one of the following reasons: inadequate imaging to assess DCM status (n = 7), no evidence of dilated cardiomyopathy based on echocardiographic remeasurement by investigator (n = 8), taurine concentrations that were categorized as normal regardless of whether or not they had underlying cardiac disease (n = 1), incomplete diet history (n = 0), or concurrent cardiac disease that was considered hemodynamically significant (n = 0). A total of 24 client-owned golden retrievers diagnosed with taurine deficiency and DCM were enrolled in the clinical portion of this study. Diets consumed by these dogs included 9 grain-free brands (Acana = 15, Taste of the Wild = 1, 4Health = 2, Zignature = 1, Instinct = 2, NutriSource = 1, Kirkland Signature = 1, Fromm = 2, and Orijen = 1). Of interest, 23 of 24 dogs with known diet amount consumption were consuming less than estimated maintenance energy requirements. At baseline, 11/24 dogs were diagnosed with congestive heart failure and prescribed diuretic therapy (furosemide). Nine out of these 11 dogs had resolution of congestion at time of follow-up. Of these nine dogs, five had successful discontinuation of furosemide therapy, and four had a reduction of their maintenance furosemide dose by 50–56%. In the two remaining dogs, one dog remained in congestive heart failure and the other was lost to follow-up. The remaining 13 dogs were considered to have occult DCM and no diuresis was prescribed. At baseline, 24 dogs were prescribed taurine supplementation at a median dose of 1500mg orally twice per day (daily dose range of 2000-4500mg) and thirteen dogs were additionally prescribed L-carnitine supplementation at a median dose of 2000mg per day (range 500- 6000mg). Additional medications included pimobendan (Boehringer Ingelheim Vetmedica, Inc., Duluth, GA, USA) (n = 13), enalapril (n = 7), benazepril (n = 4), spironolactone (n = 6), and diltiazem (n = 2). Follow-up data was available in 16 dogs. One dog was successfully removed from all cardiac medications and remained on only taurine and L-carnitine supplementation. In addition, taurine and L-carnitine supplements were successfully discontinued in four dogs based upon echocardiographic resolution of DCM.

A control group of healthy Golden Retrievers were also evaluated. Mean whole blood taurine concentration in samples obtained from 52 apparently healthy golden retrievers (mean age 5.1 +/- 2.8; mean weight 27.7 +/- 4.6 kg) was 279.1 +/- 51.5 (min 164 nmol/ml, max 382 nmol/ml). Forty-three of 52 had complete diet histories available. Twelve of 52 dogs had whole blood taurine concentrations of 200–250 nmol/mL, and 4/52 dogs (7.7%) had whole blood taurine concentrations < 200 nmol/mL. Interestingly, all 4 dogs with whole blood taurine concentrations < 200 nmol/mL, and 10 dogs with whole blood taurine concentrations between 200–250 nmol/ml in which complete diet histories were available, were on diets that were legume-rich, and/or were grain-free. None had DCM.

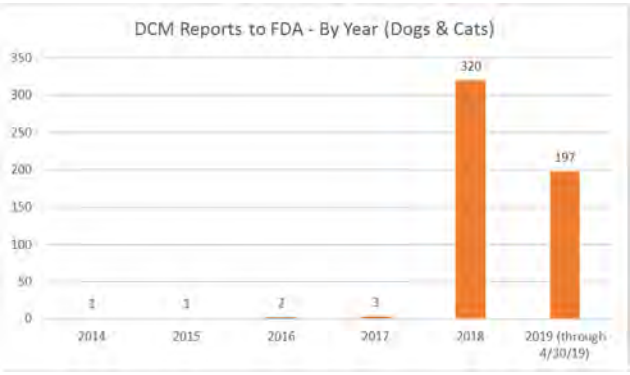
Most dogs in the study with DCM (15 of 24) were fed a single diet (Acana), which was significantly associated with low blood taurine concentrations, again suggesting that specific diet formulation may play an important role. However, as in the previous study, soluble vs. insoluble fiber concentrations were not available for the diets, nor were taurine, methionine, or cysteine concentrations, meaning that the true nutrient profiles of the diets could not be assessed and reinforcing the point that diet formulation for nutrients—not ingredients—is essential. It also suggests that nutrient requirements may vary widely based on breed, diet, and other phenotypic data. Indeed, most of the dogs with DCM in the previously described study were consuming less energy compared with their predicted requirements. It also bears pointing out that the numbers in both studies were very low (representing less than 100 DCM affected dogs between them), which surely represents a fraction of the dogs consuming grain-free, pulse-based diets.

SO WHAT INFORMAITON IS PROVIDED BY THE FDA REPORT OF JUNE 2019.

In July 2018, the FDA announced that it had begun investigating reports of canine dilated cardiomyopathy (DCM) in dogs eating certain pet foods, many labeled as "grain-free," which contained a high proportion of peas, lentils, other legume seeds (pulses), and/or potatoes in various forms (whole, flour, protein, etc.) as main ingredients (listed within the first 10 ingredients in the ingredient list, before

vitamins and minerals). Many of these case reports included breeds of dogs not previously known to have a genetic predisposition to the disease. The FDA's Center for Veterinary Medicine (CVM) and the Veterinary Laboratory Investigation and Response Network (Vet-LIRN), a collaboration of government and veterinary diagnostic laboratories, continue to investigate this potential association. Based on the data collected and analyzed thus far, the agency believes that the potential association between diet and DCM in dogs is a complex scientific issue that may involve multiple factors.

For the purposes of this investigation, the FDA defines a "case" as an illness reported to FDA involving a dog or cat that includes a diagnosis of DCM. Many of the reports submitted to the FDA included extensive clinical information, including echocardiogram results, cardiology/veterinary records, and detailed diet histories. The numbers below only include reports in which the dog or cat was diagnosed with DCM by a veterinarian and/or veterinary cardiologist. We did not include in these numbers the many general cardiac reports submitted to the FDA that did not have a DCM diagnosis. However, this case information is still valuable, as it may show heart changes that occur before a dog develops symptomatic DCM. Although the FDA first received a few sporadic reports of DCM as early as 2014, the vast majority of the reports were submitted after the agency notified the public about the potential DCM/diet issue in July 2018.



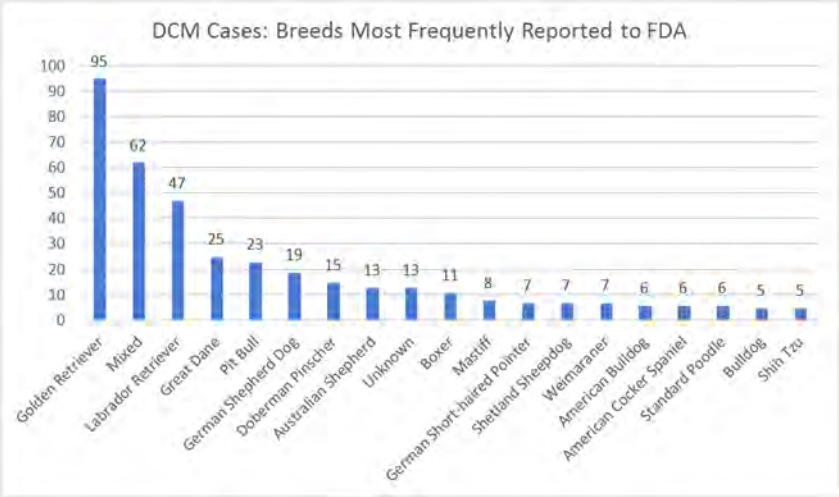
Between January 1, 2014 and April 30, 2019, the FDA received 524 reports of DCM (515 canine reports, 9 feline reports). Approximately 222 of these were reported between December 1, 2018 and April 30, 2019 (219 canine reports, 3 feline reports). Some of these reports involved more than one affected animal from the same household. The breakdown of reported illnesses below reflects the number of individual animals affected. The American Veterinary Medical Association estimates that there are 77 million pet dogs in the United States. Most dogs in the U.S. have been eating pet food without apparently developing DCM. It's not known how commonly dogs develop DCM, but the increase in reports to FDA signal a potential increase in cases of DCM in dogs not genetically predisposed.

Animal numbers in DCM Reports received between January 1, 2014 and April 30, 2019

	Number of reports	Number of animals affected	Number of deaths
Dogs	515	560	119
Cats*	9	14	5

*Cats are generally more likely to develop hypertrophic cardiomyopathy (a heart disease)

Dilated cardiomyopathy is recognized as a genetic condition in dogs, typically in large or giant breeds, such as the Doberman Pinscher, Great Dane, or the Irish Wolfhound. It is also seen in Cocker Spaniels associated with taurine deficiency. It is believed to be less common in small and medium breed dogs. We suspect that cases are underreported because animals are typically treated symptomatically, and diagnostic testing and treatment can be complex and costly to owners. FDA has observed a reporting bias for breeds like Golden Retrievers due to breed-specific social media groups and activities that have raised awareness of the issue in these communities and urged owners and vets to submit reports to FDA. Because the occurrence of different diseases in dogs and cats is not routinely tracked and there is no widespread surveillance system like the Centers for Disease Control and Prevention have for human health, we do not have a measure of the typical rate of occurrence of disease apart from what is reported to the FDA.



Additional breeds with more than one report include Afghan Hound, Australian Cattle Dog, Beagle, Belgian Tervueren, Border Collie, Boston Terrier, Bull Terrier, Chihuahua, Dalmatian, English Cocker Spaniel, English Springer Spaniel, Flat-coated Retriever, French Bulldog, Gordon Setter, Hound (unspecified), Irish Setter, Irish Soft-Coated Wheaten Terrier, Jack Russel Terrier, Maltese, Miniature Schnauzer, Old English Sheepdog, Pomeranian, Portuguese Water Dog, Pug, Retriever (unspecified), Rhodesian Ridgeback, Rottweiler, Rough-haired Collie, Saluki, Samoyed, Schnauzer (unspecified), Shepherd (unspecified), Staffordshire Bull Terrier, Standard Long-haired Dachschund, Vizsla, Whippet, and Yorkshire Terrier.

Genetic forms of DCM tend to affect male large and giant breed dogs beginning in middle to older age. DCM cases reported to FDA CVM have involved a wide range of dog breeds, ages and weights. There have been a greater proportion of males than females, consistent with what is seen in genetic forms. The significance of this is unknown, but it may be that some cases are genetic in origin or a combination of diet and genetic tendencies.

Table 1: Mean Age and Weight - DCM Cases in Dogs Reported to FDA-CVM

Dogs	Mean	Range
Age (years)	6.6	0.4-16
Weight (lbs)	67.8	4-212

Table 2: Mean Age and Weight - DCM Cases in Cats Reported to FDA-CVM

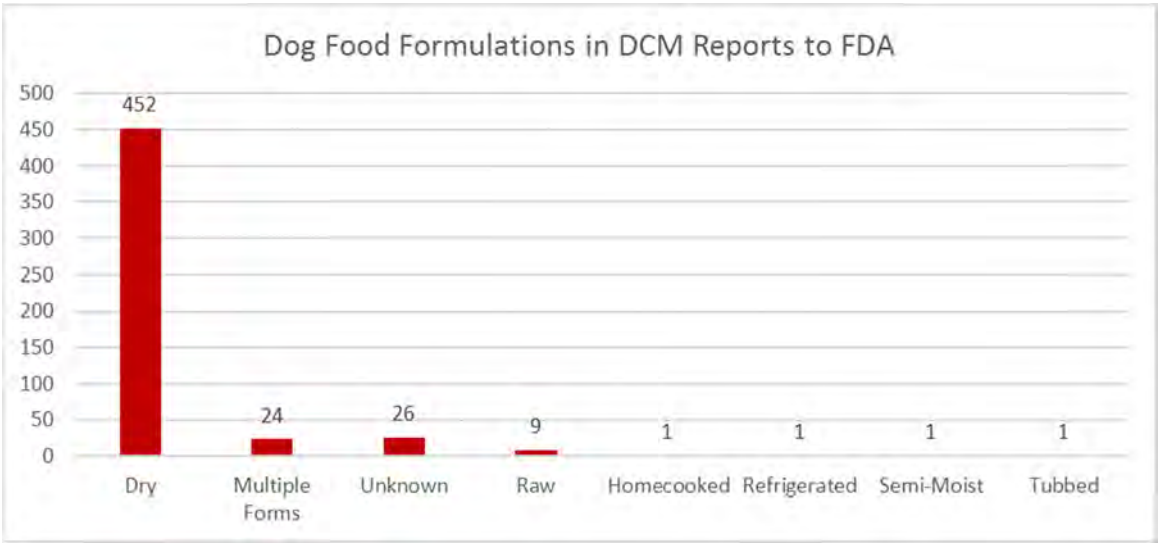
Cats	Mean	Range
Age (years)	6	0.4-17
Weight (lbs)	10.7	7-13

Table 3: Sex of DCM cases reported to FDA-CVM by species (%)

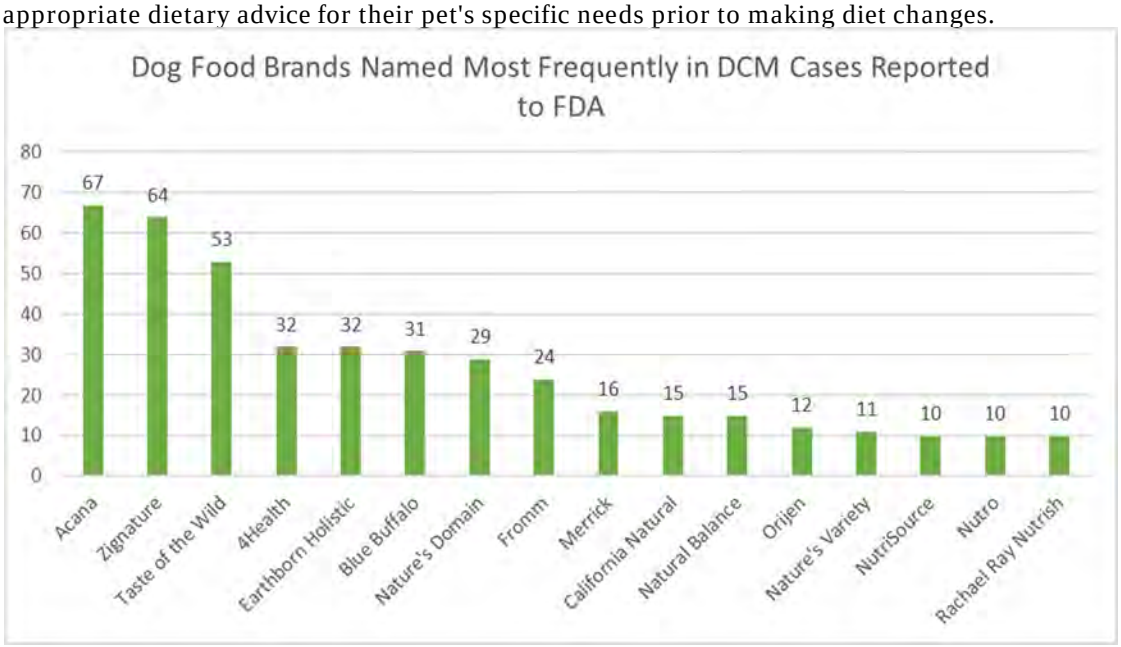
Sex (%of cases)	Male	Female
Dogs	58.7	41.3
Cats	62.5	37.5

Diet Information from Reported Cases

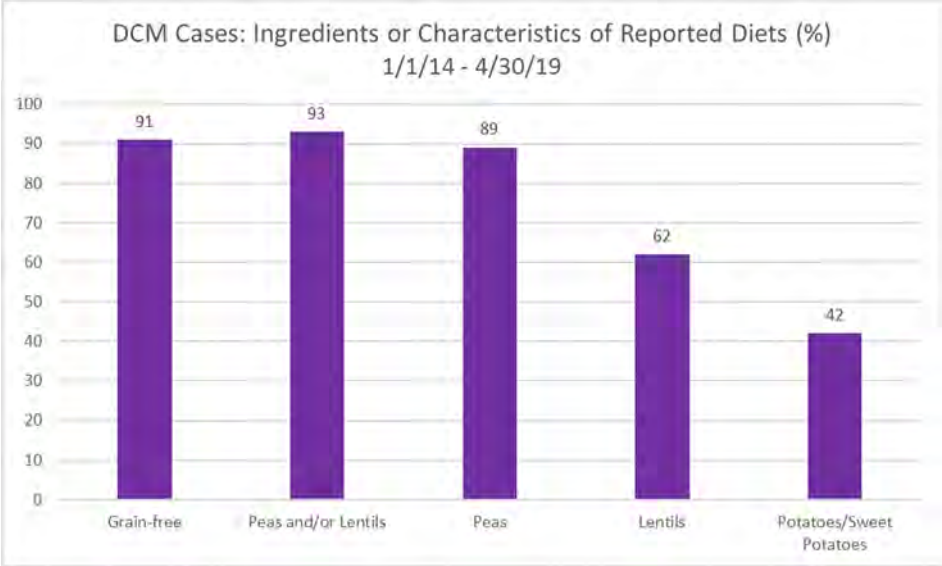
Review of the canine reports shows that most reports were for dry dog food formulations, but raw food, semi-moist food, and wet foods were also represented.



When examining the most commonly reported pet food brands named in DCM reports submitted to the FDA, it is important to note that the graph below is based on reports that included brand information and that some reports named multiple brands. Brands that were named ten or more times are featured below. For a granular, case-by-case breakdown of DCM reports submitted to the FDA, see Canine Dilated Cardiomyopathy Complaints Submitted to FDA-CVM Through April 30, 2019. FDA urges pet owners to work with their veterinarians, who may consult a board-certified veterinary nutritionist, to obtain the most appropriate dietary advice for their pet's specific needs prior to making diet changes.



To better characterize diets reported in DCM cases, product labels were examined to determine whether the product was grain-free (did not contain corn, soy, wheat, rice, barley or other grains), and whether the products contained peas, other lentils including chickpeas and beans, or potatoes (including sweet potatoes). Because so many products contained peas and/or lentils, a category was created for "peas and/or lentils". More than 90 percent of products were "grain-free", and 93 percent of reported products had peas and/or lentils. A far smaller proportion contained potatoes.



Animal protein sources in the reported diets varied widely, and many diets contained more than one protein source. The most common proteins in the reported diets were chicken lamb and fish; however, some diets contain atypical protein sources such as kangaroo, bison or duck. No one animal protein source was predominant.

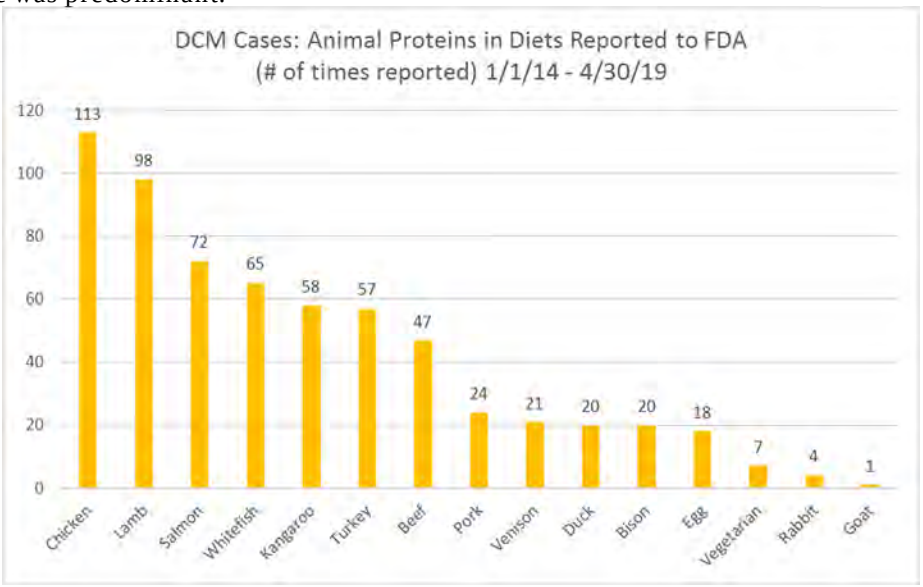


Table 1. Average values for grain-free and grain-containing products shown on a dry matter basis

Measurement	Average Grain-Containing	Average Grain-Free
Protein	28.8 %	29.6 %
Fat	15.2 %	16.6 %
Total Taurine	0.13 %	0.14 %
Total Cystine	0.3 %	0.29 %
Total Methionine	0.59 %	0.55 %
Total Methionine-Cystine	0.89 %	0.84 %
Total Dietary Fiber	8.6 %	12.1 %
Crude Fiber	2.5 %	4.6 %
Insoluble Fiber	7.2 %	11.7 %
Soluble Fiber	1.46	<1.41
Starch	37.4 %	26 %
Resistant Starch	<2.15 %	<2.15 %
Choline Chloride	3289 ppm	2731 ppm
Choline	2453 ppm	1979 ppm

One hundred seventy-six dogs and 3 cats (including both DCM and non-DCM cases) had both a taurine measurement and an echocardiogram (Table below). Approximately 64% of dogs with DCM had a taurine measurement. Of the pets diagnosed with DCM and tested for taurine, approximately 42% had at least one low blood taurine value. Golden Retrievers represented approximately 37% of all dogs with low taurine and DCM and approximately 48% of all dogs with low blood taurine regardless of type of cardiac findings.

Table 1. Number of pets with various taurine levels (either whole blood and/or plasma) and echocardiogram changes based on medical record review for dogs with a taurine test.

Taurine Status	DCM	Non-DCM Cardiac Changes	Normal Heart
Low	53 (51 dogs, 2 cats)	21 dogs	10 dogs
Normal	38 dogs	10 dogs	4 dogs
High	28 (27 dogs, 1 cat)	1 dog	
Mixed*	6 dogs		
Unknown	8 dogs		

*Mixed values include a normal whole blood taurine with low plasma taurine, increased whole blood taurine with normal plasma taurine, or normal whole blood taurine with increased plasma taurine.

Table 2. Pet breeds grouped by taurine (Tau) status and echocardiogram changes for dogs with a Tau test.

Taurine Status	Diagnosis	Number of Pets by Breed (dogs) of Species*
Low	DCM	Golden Retriever (19), Labrador Retriever (4), Samoyed (3), 2 each: Cocker Spaniel, Goldendoodle, Mixed Feline, Pitbull Mix; 1 each: American Staffordshire Terrier, Australian Shepherd, Blueheeler Mix, Bluetick Coonhound, Doberman Mix, Boxer, Boxer mix, Coton de Tulear, French Bulldog, Golden Retriever Mix, Great Dane, Great Dane Mix, Maltese, Pitbull, Rhodesian Ridgeback Mix, Sheepadoodle, Standard Poodle, Standard Schnauzer, Viszla Mix

Normal	DCM	Doberman Pinscher (5), Golden Retriever (4), Great Dane (4), Labrador Retriever (3), 2 each: Labrador Retriever Mix, Pitbull, Shetland Sheepdog; 1 each: Australian Shepherd Mix, Boston Terrier, Bulldog Mix, German Shepherd, German Shorthaired Pointer, Goldendoodle, Hound Mix, Miniature Schnauzer, Newfoundland Mix, Rhodesian Ridgeback, Shih Tzu, Standard Poodle, Wheaten Terrier, Welsh Terrier, Yorkshire Terrier, Yorkshire Terrier Mix
High	DCM	Great Dane (4), 2 each: American Staffordshire Terrier, Australian Cattle Dog, German Shepherd; 1 each: Akita, Australian Shepherd, Basenji, Beagle, Beagle Mix, Boston Terrier, Collie Mix, French Bulldog, German Shepherd Mix, Golden Retriever, Labrador Retriever, Mastiff, Miniature Australian Shepherd, Miniature Schnauzer, Mixed Feline, Pug, Unknown Crossbreed, Yorkshire Terrier
Mixed^	DCM	1 each: Boxer Mix, Catahoula Leopard Dog, German Shepherd, Great Dane, Pitbull, White Shepherd
Unknown	DCM	1 each: Beagle Mix, Chesapeake Bay Retriever, English Setter, German Shorthaired Pointer, Miniature Schnauzer, Pitbull Mix, Pomeranian, Unknown Crossbreed
Low	Non-DCM Changes	Golden Retriever (13), 1 each: American Staffordshire Terrier, Doberman Pinscher, Flat Coated Retriever, Golden Retriever Mix, Labrador Retriever Mix, Poodle-Wheaten Terrier Mix, Rough Collie, Wire Hair Pointing Griffon
Normal	Non-DCM Changes	Golden Retriever (4), Doberman Pinscher (2), 1 each: Chihuahua, Pitbull Mix, Shetland Sheepdog, Whippet
High	Non-DCM Changes	German Shepherd Mix (1)
Low	Normal	Golden Retriever (7), 1 each: Corgi, Goldendoodle, Labradoodle
Normal	Normal	Golden Retriever (4)

*Cats only
^ Mixed values include a normal whole blood taurine with low plasma taurine, increased whole blood taurine with normal plasma taurine, or normal whole blood taurine with increased plasma taurine.

Table 3. Taurine results by blood sample measured for dogs and cats with confirmed DCM.

Taurine Status*	Whole Blood only	Plasma only	Whole Blood and Plasma	Whole Blood with normal Plasma	Plasma with Normal Whole Blood	Unknown
Low	25 dogs	16 dogs, 2 cats	8 dogs		2 dogs	2 dogs
Normal	26 dogs	2 dogs	7 dogs			3 dogs
High	14 dogs	6 dogs	7 dogs, 1 cat	1 dog	3 dogs	
Unknown						7 dogs

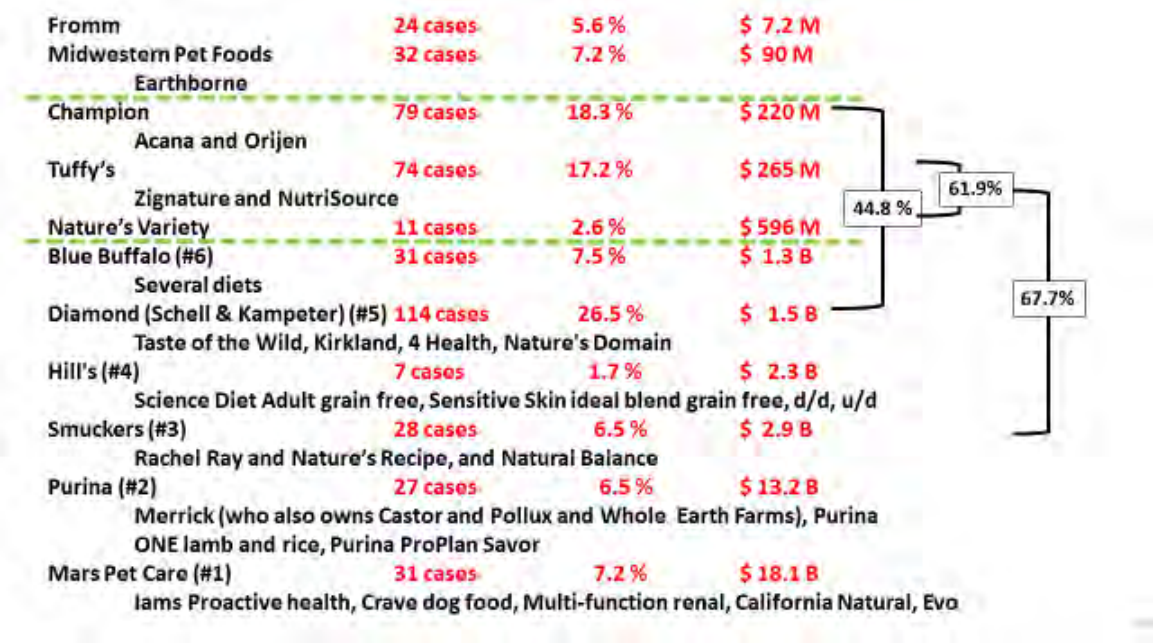
*The taurine status is based on reference ranges used by the laboratory that performed the test.

When you evaluate the FDA information further, including the 80 or so page document of specific case details, you find the following:
Association with certain diets:

- 90% grain free
  - 10% grain-based
- > 90% of grain-free diets were legume based (lentils, peas, chick peas, etc)
- > 64% contained conventional protein sources
  - Add in fish, duck, egg, vegetarian, rabbit, goat – 87.5%
- grain free diets were higher in fiber (total - primarily due to insoluble fiber)
- majority were dry foods (84%),
  - 16% or so were not
- very few cats in the list

- Many were breeds with a predisposition to DCM

Furthermore, the graph shown above of the top 16 brands list some specific brands but also groups brands in the report made by one company without separating the brand from the company. If you evaluate the full data, you find:



So what is my take on things:

There is association with certain grain free diets:

90% were grain free – but - 10% were grain-based

> 90% of grain-free diets were legume based (lentils, peas, chick peas, etc)

not just "grain free' but "grain free and legume-based"

> 64% contained conventional protein sources

Add in fish, duck, egg, vegetarian, rabbit, goat – 87.5%

grain free diets were higher in fiber (total - primarily due to insoluble fiber)

majority were dry foods (84%), 16% or so were not

very few cats in the list

why? Perhaps cat foods are supplemented with taurine?

> 65% were breeds with a predisposition to DCM

WHAT HAVE LEARNED SINCE THE FDA REPORT?

Taurine

A study was published evaluating feeding a commercial grain free diet to Labrador retrievers, which shoed over the course of 6 ½ months and actually increased plasma taurine concentrations while maintaining plasma concentrations of amino acids.[31]

Retrospective study

In November 2020, a retrospective study[32] was published in the Journal of Veterinary Internal Medicine. In this study, records were evaluated between January 1, 2014, and September 30, 2018, at an academic referral hospital. Dogs had to be newly diagnosed with DCM based on a fractional shortening of < 25%, normalized left ventricular internal diameter in diastole > 1.8, and normalized left ventricular internal diameter in systole > 1.2. Lab work was evaluated in addition to echocardiographic parameters. The dog's main diet (ie, the diet providing the majority of the dog's calories) at the time of diagnosis was recorded, as was whether or not the diet was changed and each dog's final main diet. For the purposes of the study, diets were classified as traditional when they were grain-inclusive extruded diets that did not contain peas, lentils, or potatoes as main ingredients (ie, top 10 ingredients on the ingredient list), and the manufacturer of which met the World Small Animal Veterinary Association (WSAVA) Global Nutrition Committee recommendations. Nontraditional extruded diets were defined as those that were grain-free,

contained nontraditional ingredients (eg, peas, lentils) as main ingredients, or whose manufacturer did not meet the WSAVA Global Nutrition Committee recommendations. Seventy-five dogs were included and the number increased each year; however, 71 dogs were included in the final analyses. Fifteen of the dogs were eating a traditional diet and 56 were eating a non-traditional diet. Breeds included Doberman pinscher (n=18; 5 eating traditional diet and 13 eating non-traditional diet), Great Dane (n=16, 2 eating traditional diet and 14 eating non-traditional diet), Boxer (n=6, 3 eating traditional diet and 3 eating non-traditional diet), Golden retriever (n=5, 0 eating traditional diet and 5 eating non-traditional diet), Labrador retriever (n=5, 2 eating traditional diet and 3 eating non-traditional diet), mixed breed (n=4, 1 eating traditional diet and 3 eating non-traditional diet), French bulldog (n=3, 0 eating traditional diet and 3 eating non-traditional diet), Other breeds (n=14, 2 eating traditional diet and 12 eating non-traditional diet including German shepherd (2), Pit bull (2), and 1 each of Australian Cattle dog, Bull Mastiff, Caucasian Shepherd dog, German shorthaired pointer, Irish Wolfhound, Mastiff, Miniature Schnauzer, Portuguese water dog, Saint Bernard, and Samoyed). There were no differences in echocardiographic parameters between dogs eating traditional diets and dogs eating non-traditional diets. Fifty of 71 dogs had congestive heart failure; 7 of 15 eating traditional food and 43 of 56 eating non-traditional food. Plasma taurine was measured in 20 of 71 dogs including 19 in the non-traditional diet group and 1 in the traditional diet group. Four dogs of the 19 had low blood taurine concentrations. Dogs were treated with various medications and 30 dogs received taurine supplementation while 3 dogs received carnitine supplementation. For the 56 dogs in the non-traditional diet group, 31 had their diets changed whereas 25 did not. For the 15 dogs in the traditional diet group, 6 had their diets changed whereas 9 did not. Forty-five of 71 dogs (63%) had follow-up echocardiographic information at least 90 days after diagnosis. Diet group and presence of congestive heart failure were associated with a significant change in normalized left ventricular internal diameter in systole; other echocardiographic parameters were not different between diet groups whether diet was changed or not. At time of data analysis, 11 dogs were alive; 10/31 dogs in the nontraditional diet group who had diets changed, 0/25 in the nontraditional diet group that did not have their diets changed, and 1/15 dogs in the traditional diet group. Of the 60 dogs that were no longer alive, 21 dogs died suddenly, 38 dogs were euthanized due to worsening congestive heart failure or other causes, and 1 dog died of unknown cause(s). In all dogs and in dogs with congestive heart failure there was a longer survival time in the non-traditional diet group that had a diet change when compared with those in the non-traditional diet group that did not have a diet change; survival curves were identical. There was no difference, though, between dogs eating traditional diets and the other 2 groups.

This study does not provide much supportive evidence concerning “non-traditional diets” and DCM. Bear in mind that the number of cases increased between 2014 and 2019, which could coincide with the FDA announcements and that the majority of dogs were consuming non-traditional diets implying that these dogs were either referred or recruited resulting in an over-representation of dogs with DCM eating such diets. Despite this, survival and response to treatment including diet change were not found except in those dogs who were eating a non-traditional diet and had a diet change. Unfortunately, diet information is not provided and so it is difficult to draw other conclusions.

DCM Symposium at Kansas State University

There were several presentations by the FDA, representatives of food companies, cardiologists, and other academicians. Pulse ingredients have been around for decades and the FDA stated “no clear evidence indicating that grain-free foods with pulse ingredients are inherently dangerous”. Further, the FDA acknowledged reports of non-hereditary DCM associated with dogs eating grain-containing diets. One study showed no increase in incidence from 2000 to 2019 with only a slight but not significant increase in small and mixed-breed dogs from 14 cardiology practices across the US. Data was also presented that showed that feeding grain-free diets was not associated with changes in taurine, carnitine, or amino acids in Labrador Retrievers. One presentation suggested a possible link to infectious agents such as viruses in some dogs. Another study suggested a possible link to subclinical anemia. The FDA concluded that DCM is a scientifically complex, multi-faceted disease, and it has a clear genetic component and other potential factors may contribute. The FDA feels that this is not a regulatory issue and so no recalls have occurred and further specific brand names will no longer be released.

Here are some thoughts:

all grain free diets are not bad – actually most are good

boutique and exotic are arbitrary and have no actual defined or legal definition or meaning

avoid feeding high risk dogs kibble grain free diets that are legume based and contain more than 5% fiber – such as Golden retrievers

we do not know the mechanism of the associated heart disease but it is likely a combination of factors as mentioned

taurine likely has some role – even if not measurable whole blood or plasma likely at a tissue level – it is not an essential nutrient for dogs per AAFCO and NRC but perhaps it should be

but if want to feed a grain free diet – monitor closely including echocardiography – consider supplementing with taurine regardless – cheap and safe – may not help, but probably won't hurt

consider use of therapeutic diets – I like "joint" and "intestinal" diets as many are supplemented with taurine (and carnitine and methionine – even though taurine and carnitine are not "essential") and despite recent recalls of some they tend to have more defined formulas and better quality control over the formulation and production of the product (although as mentioned there are a few therapeutic diets that do show up on the FDA list). Many also carry AAFCO feeding trial adequacy statements for adults and for growth.

you can measure plasma or whole blood taurine if there are concerns or if you see dogs with heart disease

Do not discount the possible association of any diet with DCM – ALL cases of DCM and associated diet – grain-free, grain-based, whatever – should be reported to the FDA so that the investigation does not become target-fixated.

Admit that "we don't know what we don't know"

Be brand neutral – your reputation and possibly career is resting on a specific company or brand

Business is business

We may never figure this out as it complex and likely a combination of nature and nurture

When you only look for what you want to see, you will only see what you wanted to look for

This is not a grain-free issue – this is a pet food industry and FDA issue

This is also not likely to go away. People understand imbalances, contaminants, toxins, deficiencies, etc. but this issue is "dogs dying of heart attacks related to diet", which many people relate to. This is likely at least in part for the emotion elicited with this.

A recent thoughtful review supports these conclusions by reiterating the crucial need for plant-based diets for dogs to be formulated with sufficient quantities of bioavailable methionine and cysteine to support adequate taurine synthesis.[29] This can be achieved with the addition of purified AAs and other sources that are readily available.[30] Finally, a recent commentary carefully concludes that a true cause-and-effect relationship between grain-free diets and DCM has not been proven, and other factors may ultimately be more important.[2] Taken together, these recent publications may point to faulty nutrient formulation in some, but not all, grain-free diets.

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Presentation and diagnostics of perforating wounds

Marije Risselada, DVM, PhD, DECVS, DACVS-SA
Purdue University, West-Lafayette, Indiana. mrissela@purdue.edu

1. Introduction

The external appearance might not always be indicative with the amount of internal injuries. Similarly, the repercussions of missing an internal injury might be severe – and might in some cases outweigh the morbidity of a negative explore.

2. Types of wounds/injuries:

Broadly, perforating wounds can be divided by two big classification systems: by body area or by etiology. Perforating wounds to the head (brain), neck, thorax, abdomen and pelvis/perineum all have their individual concerns and diagnostic focus – this series focuses on neck, thorax, abdomen and perineum. Similarly, different etiologies, such as blunt trauma, bite wounds, or impalement injuries might dictate a different course of action in the diagnostic and treatment plan.

3. Location:

Although often multiple structures will be involved simultaneously, and can extend into different body areas, they will be addressed separately below under 4 main areas: cervical, thoracic, abdomen, and perineal.

Cervical:

Bite wounds occur frequently in the neck, and often external appearances do not seem to extend beyond puncture wounds, and bruising. The trachea is a relatively fixed and rigid structure, and puncture wounds after a fight can occur. These tend to be in between tracheal rings, but are sometimes more extensive. Cervical hyperextension (Lawrence et al, 1999) can lead to tracheal avulsion, either cervical or intrathoracically. Cats can present acutely, or a pseudotrachea can form, connecting the 2 parts – and symptoms can develop later due to narrowing of the lumen.

Air can be seen tracking along the cervical fascial planes into the mediastinum, radiographically outlining the esophagus and large vessels, but does not necessarily involve a pneumothorax. In caudal wounds, there might be a direct connection to the thoracic cavity, leading to pneumothorax. Tracheal air leakage should be distinguished from external air trapping from the wounds themselves.

Esophageal perforation can occur in conjunction with, or separately from, tracheal damage, but due to the esophagus' collapsed state and relative mobility, esophageal injuries might occur less frequently than tracheal injuries. The trachea is a more rigidly open, less mobile tube, and puncture wounds can occur.

Muscle trauma, sometimes very extensive, can occur in bite wounds, due to shaking and the animals trying to get away, tearing several muscles. Any asymmetry, or discontinuity or indentations on palpation should raise suspicion for more extensive muscle damage that might require repair.

Thorax:

Intercostal muscle tears can occur both with blunt and sharp trauma, but are most commonly associated with bite wounds. Several intercostal spaces can be involved, creating palpable defects, and widening of the IC spaces on radiographs. The skin can be seen sucked internally upon inspiration (pseudoflail chest, figure 1). If multiple rib fractures are present, with a freely moving segment of ribs, the entire segment will move upon inspiration (flail chest, figure 2).

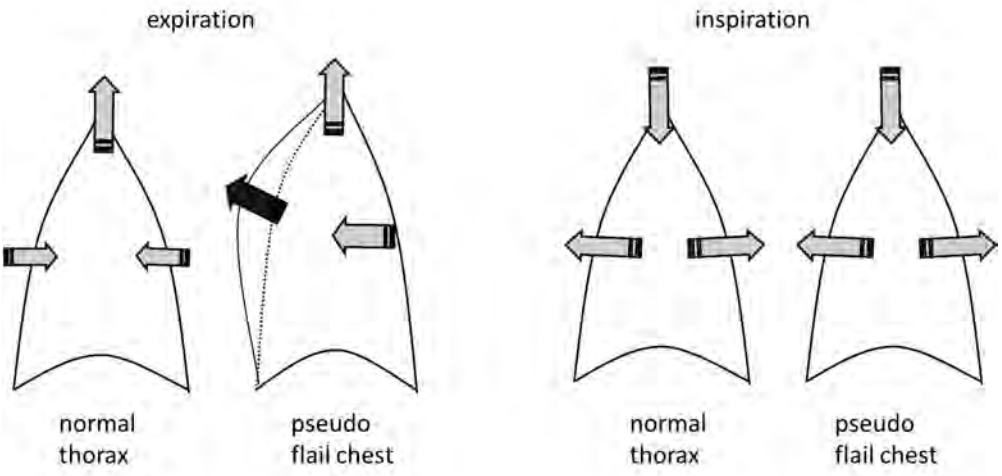

Figure 1: thoracic movement in pseudoflail chest

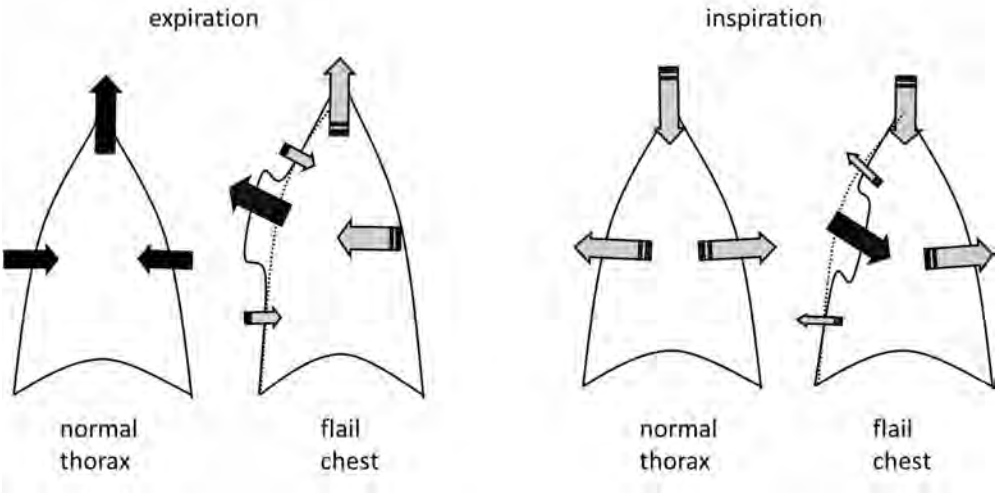

Figure 2: thoracic movement in flail chest

Diaphragmatic rupture/tear: this can occur as a standalone, or together with other trauma, and can be diagnosed based on survey thoracic radiographs.
Direct damage to the lung in conjunction to chest wall trauma appears infrequent (Risselada et al, 2008) – most likely due to the lung collapsing and retracting from the body wall at the time of loss of negative pressure. However, inspection of the lungs at time of chest wall repair is recommended.

Abdomen:
Body wall damage can be extensive, and can range from multiple puncture wounds with bruising and crushing, to muscle tears and avulsions. Careful palpation might lead to an index of suspicion, but bruising & edema might interfere with full identification of muscle interruptions, and imaging might be of help to fully assess discontinuity of the abdominal wall.
Damage to hollow viscera, such as the intestinal tract or the bladder should be ruled out in any perforating abdominal wound. Damage to vascular organs such as liver, spleen, kidney can occur, and can be self-limiting, but assessment for ongoing bleeding in an acute presentation might be prudent.

Perineum:
While bite wounds in the perineal area occur less frequently than in the cervical area, they can occur. Inspection of this area, together with a rectal exam, and inclusion in abdominal radiographs might allow early identification of rectal damage.
Urethral damage can similarly occur secondary to pelvic fractures, or bite wounds, and might not be immediately evident. Sometimes signs only occur after urine extravasation causes local swelling and cellulitis if the tear is small.

4. Etiology:
Various etiologies exist, and can be broadly categorized into sharp (impalement, gunshot wounds, bite wounds, other fight wounds) or blunt (car accidents, fall), or can be a combination (bite wounds).

Impalement & projectiles/gunshot wounds
Impalements can occur due to a fall (or jump) onto a fence, or by spear or arrow. There is a fair risk of perforation or injury to an internal organ. Ideally, the impaling object is only removed in a controlled fashion in order to stop bleeding or leakage immediately. If possible, the external part should be cut short, or if difficult/impossible, the external portion should be immobilized, to minimize the chance of additional trauma during transport caused by movement of the object. If the object has barbs, removal will cause additional injury, and a surgical extraction is preferred.
Gunshot wounds, and their damage will depend on the distance and type of projectile, and signs can range from incidental finding on radiographs, to GI perforation, or to extensive appendicular bone & muscle damage.

Bite wounds
The bite wounds with most damage are the large animal vs small animal ones, where a hold +- shake occurs. Cervicothoracic injuries are common, and life-threatening damage (lung, heart, trachea) should be ruled out.

Other
Car accidents
These wounds are not commonly perforating unless an extensive shearing injury occurs, but can be accompanied by avulsions and muscle tears. Crushing/Shearing injury can also be associated with car accidents, due to the animal being dragged over the road. If over a body cavity, these can involve evisceration.

Some other causes might be a migrating foreign body, or a traumatic catherization with urethral perforation. Migrating foreign bodies can be via airway inhalation, external migration, or migration from the GI tract, and more often lead to chronic draining tracts or intermittent fistulation and non-healing wounds. Traumatic catherization can occur in de-obstructing male blocked cats, and most frequently occurs dorsally at the junction of perineal to pelvic urethra.

4. Wounds
External part
Bite wounds follow the ‘tip of the iceberg’ principle, where only bilateral puncture wounds (typically of the canines) might be visible, in conjunction with bruising. Clipping the fur (widely) around any areas with bleeding or discharge will typically lead to discovery of more wounds. Lavage and flushing without surgical exploration carries the risk of carrying debris, hair and contamination further in the wound.

Body wall injury
These might be small (puncture wounds only) or might be more involved, and lead to functional impairment, or organ herniation. Findings on physical examination will lead to a high index of suspicion, although sometimes further imaging might be needed. The need for surgical repair (and urgency) is based on the extent, suspicion of underlying organ damage, and the general status of the patient.

Internal injuries
Additional diagnostics typically are needed to evaluate the extent of organ damage. This can include ultrasound, CT, contrast studies, and centesis for fluid cytology and analysis.

5. Diagnostics:
Laboratory tests
CBC & chemistry will help establish a baseline for further patient care, as well as what stabilization might be needed prior to anesthesia, such as fluid therapy or blood products.
If free fluid is present, a sample for cytology and/or creatinine analysis might help diagnose GI perforation or urinary tract trauma respectively. A fluid creatinine 2 times the serum value is diagnostic for an uroabdomen.

Imaging
AFAST/TFAST upon admission will help with triaging and establishing larger air/fluid pockets, and can guide where to focus additional diagnostics. These methods should not replace a full abdominal ultrasound, and this might still be indicated if organ damage is suspected.
Radiographs are a helpful baseline diagnostic tool, and can establish whether there was a penetrating body cavity injury when intra-abdominal air is present, raise suspicion for a perforating thoracic injury in the case of a pneumothorax (although lung, and tracheal trauma must be ruled out). Often times muscle disruption, widening of IC spaces (indicating muscle tears), tracheal disruptions, as well as other injuries can be identified as well.
Horizontal beam radiographs might be helpful in identifying small pockets of abdominal air and can be considered in addition to a 3 view abdominal study.
If available, a trauma CT can be performed, imaging all major body areas. If indicated, a contrast study can be performed to evaluate urinary leakage, as well as to identify the location of the urine leakage (ureter vs bladder vs urethra).
Endoscopy might be used to identify tracheal and esophageal lesions, although there is a risk for a false negative study, and care must be taken not to exacerbate the leakage by insufflation. If contrast is used to assess the esophagus, ideally agents should be used that are non-toxic for subcutaneous tissues, such as diluted IV contrast media. Smaller tears are easily missed if the lumen is not distended enough to obtain sufficient pressure to fully ‘leak’ test.

6. Stabilization/immediate management
The immediate management open wounds is focused on stabilization, including protecting the airway, negative thoracic pressure and hemostasis.
Cervical wounds: a temporary bandage can be placed, protecting the wounds from the clinic environment and to compress/stop ongoing bleeding. However, this might cause leakage of contents from the esophagus to accumulate in the tissues, similar to air trapping from a tracheal tear.
Thoracic wounds: a protective temporary bandage might be needed for open chest wounds. A sterile layer is placed first after introducing a thoracostomy tube through the existing defect. The remainder of

the bandage can be placed routinely, and after placement of the bandage the thoracostomy tube can be emptied and checked intermittently as needed.
Abdomen: a soft padded bandage with a sterile first layer can be placed to protect the abdominal organs from further damage.
While cleaning the area prior to bandaging is ideal, caution must be exercised in order not to introduce foreign bodies or bacteria into the wound or body cavity by probing the wound, lavage or flushing.

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Surgical management of perforating wounds in the cervical, thoracic, abdominal and perineal area, p1: cervicothoracic

Marije Risselada, DVM, PhD, DECVS, DACVS-SA
Purdue University, West-Lafayette, Indiana. mrissela@purdue.edu

1. Introduction

The goals of this lecture are to discuss immediate wound management options, and different surgical options for various different cervical and thoracic wounds. Management and surgical options will be split by body area.

2. Immediate wound management

The primary goal should be to protect the wound from further contamination and damage. In some cases care should be taken to maintain or re-establish an airway, or avoid creating a pneumothorax.

Cervical

Airway

If there is damage to the larynx, (potentially including fractures of the hyoid bones), then luminal narrowing might be severe enough to warrant a temporary tracheostomy.

A tracheal laceration in combination with external (open) wounds typically does not cause severe respiratory signs – similar to allowing a tracheostomy to heal by second intention – but bandaging over the site might exacerbate or cause dyspnea by trapping air in the subcutaneous tissues. If the patient's breathing pattern changes after applying a bandage (even a loose bandage), then removal of the bandage is indicated and a suspicion of a tracheal injury should be considered.

Tracheal transection: it is possible that the peri-tracheal tissues provide enough of a pseudotrachea that a tracheal transection or separation of the tracheal rings can exist while still allowing airpassage. Intubation might cause the more distal piece to be pushed away by the tip of the endotracheal tube. If the patient decompensates/desaturates post intubation, this might be a differential. Reintubation w a smaller diameter tube or temporary tracheostomy at the transection site/intubation via a ventral cervical incision might be needed.

Bleeding

Severe hemorrhage from the carotid arteries or jugular veins can occur, but most often the crushing injury of the bite wound will cause sufficient vasospasm, inflammation and thrombosis that blood loss is limited. If the bleeding continues, the carotid or jugular can be clamped or ligated. The vagus and left recurrent laryngeal nerves lie close to the carotid, and care must be taken to separate these prior to hemostasis. One – potentially both – carotid arteries can be sacrificed in the dog (Holmberg et al, 1997), but not in the cat (Gillilan, 1976). One (or both) jugular veins can be ligated.

Esophagus

Esophageal injuries are difficult to detect without a direct surgical explore, and do not need immediate intervention prior to surgery, given its mostly empty state. If longer stabilization is needed prior to surgery, bypassing the esophagus (NE or NG tube) might be appropriate.

Thoracic

Open chest

Initial care: Protecting the wound with a bandage will protect further contamination of the wounds. By providing support, it might also increase comfort level, especially if rib fractures are present, as well as decrease subcutaneous bleeding. Ideally all wounds are treated with sterile gloves and a sterile

protective layer, and the tissues in the wound bed protected from desiccation by applying either Triple Antibiotic ointment or sterile KY. Even if there was not time yet, or the animal was not stable, or analgesed/sedated enough for clipping and cleaning, providing coverage might help protect the tissues.
If a clear communication to the chest cavity can be visualized, placing a thoracostomy tube prior to placing the bandage will prevent a tension pneumothorax, and will allow continued emptying of the thoracic cavity while the animal is stabilized.
If only puncture wounds are present, smaller bandages, just protecting the wounds might be appropriate while awaiting confirmation of presence of a pneumothorax, as this would allow access to perform a thoracocentesis or place a thoracostomy tube, and place a more encompassing bandage later.
Larger wounds: if the wound is large enough that the cavity can be visualized, and/or lung tissue herniates out, and approximating the skin with staples can help protect the lung from abrasion from the primary bandage layer. A temporary thoracostomy tube can be inserted through the wound for ease & efficiency, as further wound care/exploration will be needed after stabilization, and a tunneled thoracostomy tube can be placed afterwards.

Pre-surgical assessment & management: Clear external debris and contamination is removed. Presence of a similar level of debris in the wound is an indication for surgical explore of the wound. Lavage of the deeper structures is done at that time, as opposed to lavage with only a smaller entry site, as this would risk pushing debris into the deeper tissues.
Puncture wounds often only have a small external wound, and the deeper damage is due to tearing and crushing of the tissues. This can lead to a large subcutaneous pocket, or to larger wall defects. A large subcutaneous pocket, without evidence of major organ or thoracic wall trauma could be managed conservatively by compressing the tissues & preventing the pocketing from re-occurring. The animal should be checked for development of skin necrosis due to devascularization, or for site infection.
Presence of larger defects in the thoracic wall, unstable rib fragments or recurring pneumothorax or bleeding are indications for a targeted or more extensive explore.

Rib fractures
Presence of rib fractures is not necessarily an indication for surgical exploration, but having an unstable segment or severely displaced fractures with risk for trauma to lungs could be. Oftentimes, the chest cavity is entered at the time of skin incision, or removal of the bandage, and the patient should be intubated with ventilatory support prior to bandage removal & start of surgery.
If feasible with regards to pulmonary contusions and saturation, managing the patient flail side down might help minimize pain by reducing the motion of the ribs. Similarly, analgesia can help counteract the decreased respiratory effort caused by pain, although titrating of the best analgesic dose while avoiding respiratory depression is advisable.

Damage to other structures: Bandage support might help increase comfort as well, although if the bandage is too rigid or too compressive it might push any segments or fractured ribs inward.

Bleeding
Any intrathoracic bleeding (heart, large vessels) should be stopped as part of initial stabilization – but realistically, if these are damaged, the patient might not survive long enough to reach emergency care.
Intercostal arteries and the internal thoracic artery (along the sternum) can bleed profusely, but potentially intermittently due to blood pressure – and these sites should be checked if bleeding continues or occurs after initial stabilization.

3. Surgical options

Cervical

Surgical dose/surgical extent:

The subcutaneous pocketing typically is extensive, and potentially is connected across different areas. If not, a connection can be established, to allow using only one negative suction drain. This would require all individual puncture wounds to be closed.

A full explore of the neck might necessitate both a dorsal and a ventral approach. However, all important structures can be targeted via a ventral approach. The left and right lateral skin can be lifted in a 'hanging' prep by using a towel clamp to elevate the skin – this will allow more dorsal draping, and thereby more dorsal access for bilateral puncture wounds.

Muscle trauma

Muscles are re-attached and reapposed to help restore the function and continuity. If the ends are clearly devitalized, debridement is performed, but given robust blood supply, minimal resection is typically necessary. If larger areas are removed, muscles can be tacked down in place, rather than a repair under high tension, as this is most likely to fail – given the lack of holding strength of muscle tissue without a fascial component.

Thoracic

Body wall reconstruction

Rib fractures and dislocations can occur, and the primary goal is to stabilize the rib fragments and relieve the pain during breathing by shortening the ribfragments to the point where the ends do not touch. The easiest, and preferred, surgical stabilization is a 'basket weave' pattern, by placing circumcostal sutures around several adjacent ribs, two at a time, similar to normal intercostal thoracotomy closure. An external bandage can be used to provide additional support and for comport, but external splints are typically not necessary. Use of external splints without surgical exploration has been described, but can further compromise already traumatized skin. If respiratory compromise and pain are severe enough to warrant intervention, surgical stabilization might be a safer option in the long term.

Intercostal muscles are often damaged over several intercostal spaces. If possible, suturing can be attempted, but is often not possible due to the size of the animal. The latissimus dorsi can be elevated and moved over larger defects to provide additional coverage.

Use of Negative Pressure Wound Therapy (NPWT) to treat a thoracic wall defect is not commonly used in veterinary medicine, but has been described in a dog (Nollf 2016).

Targeted explore

This can be done by visual inspection of cardiovascular structures, the esophagus, lungs, combined by submerging the lungs to check for air leaks. Overinflating of the lungs beyond 10-15cm H_2O and/or a prolonged breath hold is not needed, and is ideally avoided as this might create additional damage to the pulmonary parenchyma.

If a leak is detected, a partial or total lung lobectomy is performed. Severely bruised lung is inspected, and could be preventatively removed, if no insufflation is detected during the explore – but erring on the side of caution if only areas of a lung are under insufflated is preferred.

Full Explore

A full explore of the thoracic cavity can be either done by median sternotomy, or by bilateral lateral thoracotomies. Given the additional anesthesia & surgical time, as well as additional destabilization of the chest cavity by a median sternotomy in the case of bilateral rib trauma, this is best avoided. The side

of most trauma, and/or suspicion of underlying lung damage should be targeted first, and the second side can be addressed next, or potentially in a separate anesthetic event.

Combined

If multiple areas are involved, the benefits must be weighed of being able to reach multiple areas even if this compromised ideal positioning, versus having to reposition and redrape for each site individually. Similarly, if multiple sites are involved, and repositioning is necessary, an order of priority is chosen, and the site with the most life-threatening injuries addressed first, in case the animal is not stable enough under anesthesia for the additional surgical interventions. Generally, while the explore should be thorough, it should be targeted in order to minimize anesthetic time.

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Surgical Management of Perforating Wounds in the Thoracic Area

Marije Risselada, DVM, PhD, DECVS, DACVS-SA
Purdue University, West-Lafayette, Indiana. mrissela@purdue.edu

1. Introduction

The goal of this lecture is to discuss monitoring options to recognize potential postoperative complications early, or avoid them. Secondarily, managing complications will be discussed for each separate body area.

2. Drains

Intracavitary

Closed suction drains are the preferred method to monitor postoperative abdomens for development of peritonitis. Drains are protected from the environment, and handled aseptically to provide bacterial contamination within the grenade, and potentially tracking into the abdomen.

Abdominal monitoring: Drain production is monitored throughout the postoperative period by grenade emptying and noting the removed fluid. Ideally drain production should be less than 1-2 ml/kg/24hrs, but might not reach that target in inflammatory circumstances or if the patient is hypoalbuminemic. A sustained downward trend is another indicator for improvement. Calculating the daily production can be started immediately after surgery, but as there still will be lavage fluid present in the initial readings, starting the calculation at the morning TPR check the following morning might be preferred. This will allow a repeatable time point, and the perioperative fluid will have been removed by that point, so the readings will be more representative of actual fluid production.

A second parameter is daily fluid cytology – this can be done daily, but if concerns arise can be checked more frequently. Acellular samples can be spun down prior to slide preparation. Saving all the daily cytology slides can be helpful to check the progression of cellularity, type of cells, degenerative nature of the neutrophils. Any postoperative patient with abdominal surgery, and especially cases where extensive peritoneal inflammation was present at time of surgery, are expected to have cellular samples with neutrophils postoperatively. If bacteria are seen, the following questions & flow diagram can be followed for decision making (Fig 1).

Using fluid glucose and lactate are a well established method for differentiating bacterial septic effusion from non-septic effusion (Levin et al, 2004). However, using fluid glucose and lactate in postoperative abdomens might not provide a reliable indicator for peritonitis as it would in preoperative settings, as found by Szabo et al in non-septic, routine postoperative abdomens (Szabo et al, 2011).

Thoracostomy tubes: These should be monitored for air, fluid and cytology. Presence of air postoperatively can occur due to: entry site leakage/tube dislodgement issues, lung parenchyma leakage, necrosis and leakage through the body wall. The thoracostomy tube itself is trouble shot first, and replaced if needed, after which tissue leakage should be investigated. If the air production is minor, the leak might be treated conservatively at first, such as continuous suction by Pleurovac, or blood pleurodesis can be considered (Oppenheimer et al, 2014). Blood pleurodesis is performed by drawing 5-10 ml/kg of uncoagulated autologous blood from an intravenous line, and administering through the thoracostomy tube after removing any existing effusion (generally treatment is started with 5ml/kg, and can be repeated with another 5ml/kg). The patient is then alternated between left and right recumbency and kept quiet in the hope of the blood forming a seal over the area of leakage. While the reported success rate by Oppenheimer et al was 62%, which corresponds with my personal experience of ~50% of attempts working in cases of spontaneous pneumothorax, it is a benign intervention that might help.

If the air leakage is sudden, and of a high volume, tissue necrosis – either of the lung or the bodywall would be the top differential, and re-explore should be considered (see below).

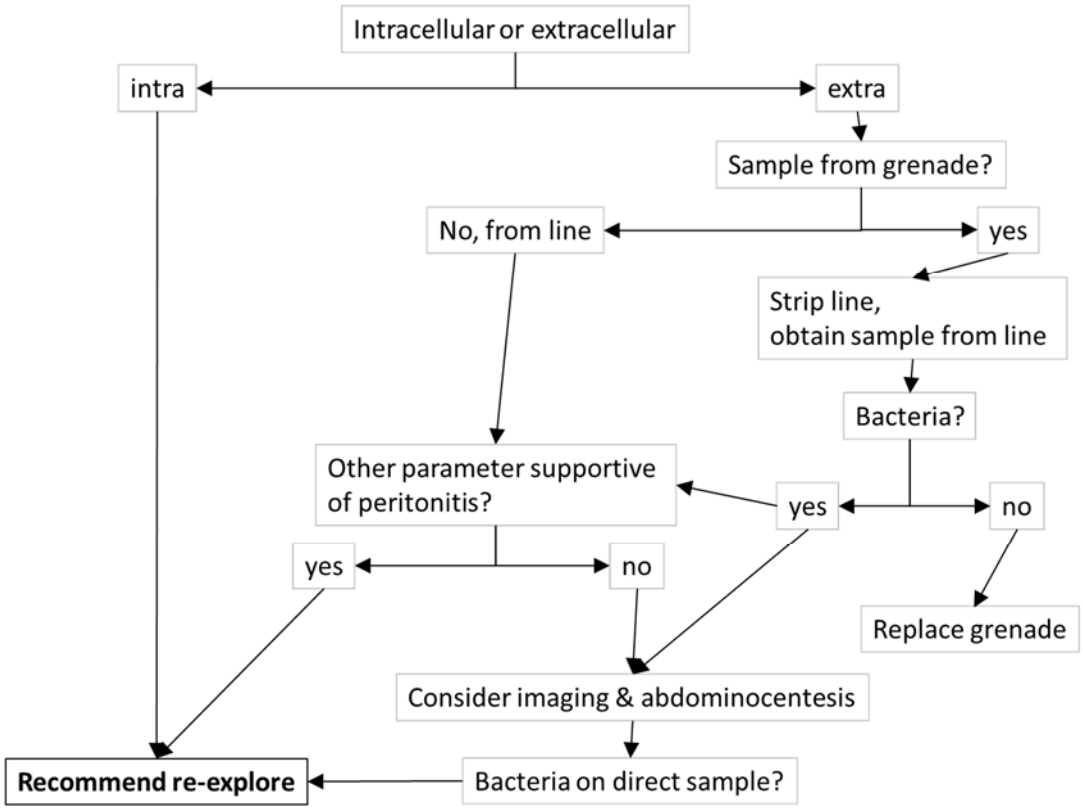

Figure 1: flow chart for drain cytology

Subcutaneous drains: a similar monitoring set up (fluid production, and drain cytology) can be used, although fluid production generally will be lower in subcutaneous drains than abdominal drains. Placing a drain should be weighed against the risk of presence of a foreign body, if the level of contamination is still questionable, it might be preferred to wait another day, and re-lavage the wound prior to closure.

3. Tissue necrosis

Skin

Debridement of skin should be performed serially, and with discretion initially. Often after 2-3 days a clear demarcation line will become evident. As long as skin is still of questionable viability, treating the wound 'open' might of benefit, as this will allow preservation of the most amount of skin (which will make reconstruction easier) and will prevent loss of negative pressure (over a drain), or incision site dehiscence.

Thoracic wall

Typically, these wounds are closed immediately, as closure of the thoracic wall should be re-established. If tissues appear of questionable viability postoperatively, the thoracostomy tube should be left in place, to monitor fluid production and air, and as a method to remove air, should the thoracic wall dehisce. Any defects in the thoracic wall that develop postoperatively should be treated surgically by aggressive removal. Devitalized tissues should be debrided en bloc, and exposed bone (from ribs) taken down

further. This might create a large defect that might need a mesh or advanced reconstructive technique (such as a diaphragmatic advancement) to close.

Abdominal wall
Aggressive debridement at time of surgery is possible due to the more 'stretchy' nature of the abdominal wall vs the thoracic wall. Postoperative defects in the abdominal wall generally don't manifest as immediate tissue necrosis due to its muscular and well vascularized nature, but are rather seen as long(er) term complications, such as herniations, and failure to fully heal.

Organs
Any questionable lesions ideally are treated at time of surgery, but sometimes issues only declare themselves postoperatively. Drains can be used to monitor for organ lesions, by checking drain cytology, fluid & air production, or chemistry (for example creatinine or bilirubin) can be run.

4. Support
Nutrition
Any postoperative trauma patient, and especially in the case of a large amount of tissue damage or ongoing effusion will be in a negative metabolic state, and aggressive nutritional measures might be needed. Placing a feeding tube at time of surgery, or the following day, should be considered. Placing a larger lumen tube, such as E tube, or PEG/G-tube might be advantageous over a smaller lumen NE or NG tube, but if the patient is not stable enough for a second anesthesia, or prolonged anesthesia, then an NE or NG tube that do not require full anesthesia might be preferred.

Antibiotics
Broad spectrum antibiotherapy should be started while awaiting culture results, and the patient's postoperative progression should be weighed against adding/changing antibiotic coverage, either while awaiting results, or after receiving the antibiogram. After the results become available, antibiotics are tailored to those results to choose the one (or a combination) that treats the cultured bacteria, while not over treating.

Analgesia
These patients are extremely painful due to the amount of tissue trauma, and opioid analgesia, as a base, is mixed with other options. One of such options are NSAIDs – especially due to the amount of tissue inflammation, but their use should be instituted carefully and only if the general state of the patient allows. Gabapentin can be considered as an adjunctive medication as well, especially as a TGH medication if use of NSAIDs is prohibited. For critical patients, adding Ketamine and Lidocaine to a fentanyl CRI might be needed to get the initial, immediate pain controlled.

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Surgical management of perforating wounds in the cervical, thoracic, abdominal and perineal area, p2: abdominal & perineal

Marije Risselada, DVM, PhD, DECVS, DACVS-SA
Purdue University, West-Lafayette, Indiana. mrissela@purdue.edu

1. Introduction

The goals of this lecture are to discuss immediate wound management options, and different surgical options for various different abdominal and perineal wounds. The main focus will be abdominal wounds. Management and surgical options will be split by body area, and type of management. Postoperative management will be discussed in the next proceeding/lecture.

2. Immediate wound management

The primary goal should be to protect the wound from further contamination and damage, and prevent evisceration or organ damage. This can be done at time of initial assessment and stabilization. Further inspection and cleaning can be done as the patient is more stable and has received analgesics and/or sedatives – potentially following imaging.

Abdominal

Initial management:

Protecting the wound with a bandage will protect further contamination of the wounds. By providing support, it might also increase comfort level, as well as decrease subcutaneous bleeding. Ideally all wounds are treated with sterile gloves and a sterile protective layer, and the tissues in the wound bed protected from desiccation by liberal application of either Triple Antibiotic ointment or sterile KY ointment. Even if there was not time yet, or the animal was not stable, or analgesed/sedated enough for clipping and cleaning, providing coverage might help protect the tissues. This will be even more important if any intraabdominal structures can be seen in the wound.

Herniated organs are gently lavaged with warmed isotonic saline to clear off gross contamination. If herniated fat is necrotic, it can be ligated prior to repositioning, but care must be taken that no vital vascular supply is compromised. In case of doubt, repositioning is preferred. The skin can be temporarily stapled closed to protect the herniated organs, and to prevent additional contamination of the abdominal cavity during clip and prep.

If herniated organs are visible, then the animal is managed until stable enough to undergo exploratory laparotomy. If need be contrast imaging of the urinary system can be performed prior to surgery to identify the location of a urinary leak, but the value of these diagnostics must be weighed against the delay in exploring the abdomen and decreasing the contamination by copious lavage of the abdominal cavity.

Pre-surgical assessment & management:

The level of contamination is an important factor in deciding whether surgical intervention and exploratory surgery is needed. In any instance where devitalized tissue is seen or debris is stuck to the tissues, surgical exploration of the wound is recommended. However, the approach might differ, whether or not a full exploration of the abdomen is concurrently performed.

Using the externally visible extent of the wound might be deceiving, as deeper parts might exist. If a large tissue flap isThe extent of the visible wound

Assessment of the deeper damage and decision making for amount of surgical explore.

Perineal

Recognition of rectal or urethral involvement is more important than full coverage. Keeping the area clean might be more effective than trying to place and maintain a bandage over the side adjacent to the anus.

The urethra can be temporarily bridged using a urinary catheter (or urinary diversion) during stabilization, but rectal perforation repair is best left until the definitive repair in surgery given the fragility and relative lack of tissues.

3. Surgical options

Abdominal

Regardless of the extent of the abdominal surgery, placement of an intra-abdominal closed suction drain should be considered to monitor for effusion, peritonitis or uroabdomen due to organ compromise. Open abdominal management, either by partial closure of the linea and a bandage, or by using intraabdominal Negative Pressure Wound Therapy (NPWT) can be considered if there is high concern for vascular compromise of the GI tract with potential for necrosis, or gross contamination that cannot be removed, or to reconstruct large defect (Nolff 2015) Both strategies will require extensive monitoring due to increased loss of fluids and proteins, and any bandage removal/change needs to be performed under general anesthesia in aseptic conditions. Open abdominal management has largely been abandoned for abdominal NPWT as maintaining sterility in the site is easier in a closed system. Another option is to fully close, but plan for a second look abdominal exploratory surgery and lavage 24 hrs later. In this setting, the first surgery is deemed damage control, and more involved measures can be left for the second procedure after additional stabilization. Similar measures have been used for traumatic bleeding from the liver, by placing towels or laparotomy sponges to tamponade bleeding in human trauma surgery with the intent to re-explore when the patient is more stable.

Local management can be performed if the perforating part of the injury is not deemed to have major intra-abdominal components, or if the body wall damage itself is only limited to a minor puncture wound. Additionally, if the owners decline abdominal exploratory surgery (limited or full), a more conservative approach can be taken by only treating extra-abdominally.

Limited, or *targeted abdominal explore*. This approach can be taken in terms of assessing the local intra-abdominal structures when exploring the wound at the area of the body wall damage itself, without fully opening the abdominal cavity with a midline incision. For example, if the patient is not stable enough to reposition during anesthesia and the local wound is far enough away from the ventral midline to address both in one field.

Full abdominal explore is the recommended extent of assessing the abdominal cavity for any potential damage. Different strategies can be used to systematically assess all structures: by quadrant, by body system, or even a different method – as long as all organs are checked. After the full abdominal explore, the defects in the body wall can be closed.

Body wall reconstruction: this can range from closing pinpoint defects, to herniorraphy by apposing muscles, using muscle flaps, or in extreme cases by using mesh. If only pin point defects are visible, they can be closed in 1 or two layers. Closure intra-abdominally will allow the suture line to be away from a larger wound – especially if there is devitalized tissue, or potential for ongoing wound management.

Extra-abdominal wound management:

The intra-abominal component is taken care of first – and after the body wall continuity is restored, the peripheral wound(s) are addressed. If both can be addressed through one incision that would be preferred, but if not, but approaches should be chosen so that the tissue between the two surgery sites will not become compromised or devitalized. While debridement and removal of grossly contaminated and devitalized tissue is recommended, it might be worth considering to remove skin in a conservative manner, and plan to do serial debridements/wound treatments until all skin devitalization had declared

itself. Aggressive removal of skin might lead to a difficult to close defect, with a lot of tension, or need for reconstructive procedures. Premature closure w/o aggressive removal of questionable skin might lead to dehiscence and surgical site complications due to ongoing skin necrosis.

Perineal

Wounds w/o perforation into rectum, or urethra: treatment will depend on the extent and size of the wound. If only a small tooth-size perforation, the wound might be able to be left to heal on its own. Larger wounds can be lavaged, the edges trimmed, and apposed to avoid additional contamination due to proximity of the anus. If closure is performed in an interrupted pattern, the ventral aspect of the wound can be left open to allow drainage to occur to decrease the risk for full suture line breakdown or abscessation.

Perforation into rectum: This site ideally should be addressed last, if multiple areas are involved. While ideally a purse string should be in place to decrease additional soiling of the surgery site, it might be easier to have access to the anus & rectum, and use gauze (placed aborad) to get visualization of both sides of the perforation (intra- and extraluminally). Sutures can be placed with knots intra- or extraluminally, but if enough tissue is present, a two layer pattern could be considered (with a small diameter rapidly absorbable monofilament intraluminally and a more slowly absorbable monofilament extra-luminally). Oftentimes, only one layer can be placed. Debridement is done judiciously and only with obviously devitalized tissue, in order not to increase the risk for stricture formation. Similar to enterotomies, closure can be redirected in the opposite direction as the defect (ie transverse closure for linear defect) if there are concerns for luminal narrowing. After reconstructing the rectal wall, the extraluminal site is copiously lavaged (a purse string can be placed temporarily at this time to help decrease contamination), and open wound management should be considered, or if not possible, drainage options should be used (whether closed drainage, or partial closure) given the highly contaminated surgery site.

Urethral perforation: unless the damage is extensive (requiring a permanent urethrostomy, or direct repair) placing an indwelling urethral catheter for 5-7 days to allow 2nd intention healing of the defect is typically recommended. Both 2nd intention healing as well as direct repair will carry a risk for stricture development. However, addressing a stricture at a later time point after full wound healing and in the absence of inflammation might be a preferable approach to performing a definitive surgery immediately. Options could include stenting, dilation or a permanent urethrostomy more proximal if possible.

Combined

If both the perineal and abdominal areas need to be addressed, the animal can be positioned with legs pulled forward, and the hind end/perineal area flush with the edge of the table. To preserve sterility of the cleaner areas, the initial drapes can be placed incorporating both sides, with a second drape or towels covering the perineal area and the field drape only cut enough to allow access to the intial area. Access can then be increased to both areas as needed without need to reposition.

Timing of surgery

If gross contamination (either external or from internal sources) is present, or abdominal wall defects within a wound with organ herniation, definitive surgery is ideally performed as soon as the animal is stable enough for anesthesia, and further stabilization can be performed while the patients is under anesthesia during surgery for source control.

In the absence of gross contamination or evisceration, further patient stabilization prior to anesthesia might be preferred to decrease the anesthetic risks.

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Diagnosis, surgical management of chronic draining wounds

Marije Risselada, DVM, PhD, DECVS, DACVS-SA
Purdue University, West-Lafayette, Indiana. mrissela@purdue.edu

1. Introduction

Chronic non-healing wound can be broadly divided in two large categories: 1) recurrent infections with fistulas and draining tracts, and 2) non healing wounds due to subcutaneous pocketing and/or localization in high motion areas.

2. Fistulas & draining tracts

After surgery

Fistula formation after a total ear canal ablation with a lateral bulla osteotomy (TECABO) is a well-known complication, and is caused by remnant epithelium. If a TECA without an LBO was performed, this might also occur, and the bulla itself might be lytic due to ongoing pressure and infection, with potential for draining tracts ventrally in addition to extending laterally from the external acoustic meatus. These complications can occur years after surgery. Any local swelling and/or draining tracts after TECABO should be investigated using advanced imaging, and excision of the draining tract and exploration of the bulla with removal of any epithelial remnant. Decreasing the inflammation & infection by pretreating with antibiotics prior to surgery might decrease the risk for surgical complications such as Facial nerve paralysis. The nerve, due to its location ventrolateral to the external meatus, is typically adhered to scar tissue, inhibiting easy identification and retraction. Concurrent inflammation of the tissues will make identification even more difficult, and will also carry the risk of increased bleeding during surgery further increasing the risk of nerve damage. Even if the draining tract temporarily closes, its fibrous remnant can still be traced to the bulla – or lacking a clear tract, the bulla is approached directly as the etiology of the swelling/draining tract.

Implants, such as orthopedic implants, or mesh, are also well documented causes of draining tracts. Removal of the implant is the only option to fully control the recurring infection. In some cases the state of bone healing must be weighed against the need for implant removal.

Due to foreign bodies

Via the *gastrointestinal tract*

Some foreign bodies that perforate a loop of bowel will instigate a draining tract with chronic signs such as (intermittent) fever, neutrophilia or non-healing wounds. This can occur at any point along the GI tract, but the cervical and peri-abdominal areas are most common.

Cervical foreign bodies most commonly are sticks that get pushed through the wall of the pharynx/esophagus as the dog runs into something. If a dog presents with an acute stick injury, it might be worthwhile to warn the owners of any signs to look out for long term (fever, swallowing abnormalities, swelling, wounds) in case of any remaining pieces. If the stick has a very irregular appearance, and there is concern for bark, debris or splinters, advanced imaging can be pursued, but often is has low yield for smaller pieces, while an explore in this area without solid evidence on imaging (dorsal to the pharynx) has limited visualization with a risk for nerve damage.

If a draining tract develops, a fistulogram (radiographs or CT) might help direct to the nidus and site where a surgical explore should be focused to find remaining pieces. If a clear tract is present, methylene blue can be injected to help guide dissection during surgery as well. Care must be taken to

not over-inject and spill into the surrounding tissues, and if the tract is damaged, it might leak into the surrounding tissues and coloring everything blue.

Perforating foreign bodies from the abdominal part of the GI tract can migrate into the thoracic cavity, through the body wall, or into the pelvic & thigh musculature. Any draining tract or non-healing wounds in this area should include imaging of the GI tract/abdomen to rule out FBs from this area.

Via *airway/lungs*
Pyothorax, spinal involvement, and thoracic wall draining tracts have all been described secondary to inhaled grass awns. Advanced imaging is needed to investigate the etiology and find the area of most concern/nidus, although trying to pinpoint foreign bodies often proves difficult.
In pyothorax cases, a full thoracic exploratory surgery with removal of mediastinal tissues, and any macroscopically unhealthy looking lung tissue in addition to long-term targeted anti-microbial and anti-fungal therapy might be needed to adequately treat the disease. Often an inciting foreign body is not found.

External
Any area can be involved, such as distal limbs – especially the interdigital area, but also the thoracic inlet, lateral chest and flank area. Any pinpoint lesion that occurs without a known fight/bite incident immediately prior, should raise suspicion that a foreign body might be embedded, especially if it recurs after medical management.

Imaging can be performed, such as ultrasound, CT, contrast studies or MR. Air in the tissues/draining tract will interfere with a focal ultrasound, and localizing a potential FB will be operator dependent. Plain radiographs will be low yield, with a CT preferred over radiographs. A contrast study, especially a fistulogram can lead to the site of origin and be very helpful. MRI has been described in cases where the potential FB will be small and the involved area is very localized, such as the footpads or interdigital skin.

3. Chronic non healing wounds: Pocketing & high motion areas
Subcutaneous pocketing can occur after closure of a chronic wound, or develop secondary to a seroma, especially if the seroma gets traumatized or infected. High motion areas such as the axilla, inguinal area, thighs are predisposed.
A combination of a closure of a chronic wound over JP drain in a high motion area will put the incision site at increased risk of forming a chronic non-healing pocket. The drain will avoid fluid accumulation while in place, but the wound pocket remaining close post drain removal will depend on a fibrinous and fibrous tissue connection, that might not be strong enough to withstand a lot of movement.

Some precautions that could help decrease the risk of wound healing complications are:
- Freshen tissue, especially in mature granulation tissue. This can range from gently abrading with a dry gauge to excising mature fibrous tissue and scars.
- Consider tacking sutures instead of a JP drain. Reducing dead space and sliding of surfaces on
- Consider compression/immobilization by bandage to prevent excessive motion, together with strict exercise restriction. Immobilization does not need to include a splint level of rigidity – it is mostly to keep the two surfaces (wound bed and overlying skin) together during the initial stages of wound healing.

Recurrent pocketing/non healing wounds:
- investigate for fungal, mycoplasma, or Pythium. Pythium and fungal disease are difficult to cuture: diagnosis might be easier by identifying hyphae on cytology or histology. Or in the case of Pythium, serology/serum titers can be considered as an alternative to get a diagnosis.
- consider omentalizing the wound, if cultures come back negative. This can be performed by a small grid approach to the abdomen, and subcutaneous tunneling. This option proved helpful in in a case series in cats with axillary nonhealing wounds (Lascelles et al, 2001).

4. References

Lascelles BD et al. Combined omental pedicle grafts and thoracodorsal axial pattern flaps for the reconstruction of chronic, nonhealing axillary wounds in cats. Vet Surg 2001;30(4):380-5.

GOING BEYOND COMPASSION FATIGUE TO FEEL BETTER

Julie Squires, BA, CCFS, Life Coach

"Everything can be taken from a man but one thing; the last of the human freedoms — to choose one's attitude in any given set of circumstances, to choose one's own way."
-Viktor Frankl, M.D., Ph.D., Neurologist, Psychiatrist and Holocaust Survivor

Compassion Fatigue

While the term compassion fatigue was first coined in 1992, it existed eons before that. For as long as humans have provided care to others, compassion fatigue has been omnipresent.

It is the profound emotional, physical, psychological and even spiritual exhaustion and depletion that we feel when we are continually bearing witness to the pain and suffering of others. In veterinary medicine the "others" includes patients, clients and your co-workers and colleagues.

The very thing that you're hard-wired to do, help others, is the very thing that creates the conditions for compassion fatigue.

It makes sense to think that the solution to compassion fatigue is to leave the work or shut down emotionally or even to self-medicate but those are short-term fixes that miss the opportunity to more deeply understand yourself.

Burnout

The term burnout is often used synonymously with compassion fatigue but it's different. It's different in it's causes and solutions.

Burnout is a workplace-specific issue. It is the result of an unbalanced work: resource dynamic over time. Too much of one thing (work) and not enough resources to meet that demand (time, staff, appointment slots, etc.).

Signs and symptoms of burnout and compassion fatigue can be similar: apathy, physical and mental exhaustion, anxiety, depression, self-medicating, insomnia or wanting to sleep more, physical ailments, hopelessness, anger and problems in personal relationships, to name a few.

Sometimes burnout can be solved with time away from the work. This is often not the case with compassion fatigue.

Those suffering from high levels of compassion fatigue can find relief through:

- getting support from a coach or therapist that specializes in compassion fatigue
- understand and practice self-care like it's their job
- explore meditation and mindfulness practices

- journal daily
- monitor and select positive influences over negative
- learn how to reframe situations so you can see all sides
- make yourself and your mental health/wellbeing a priority

Mind Management

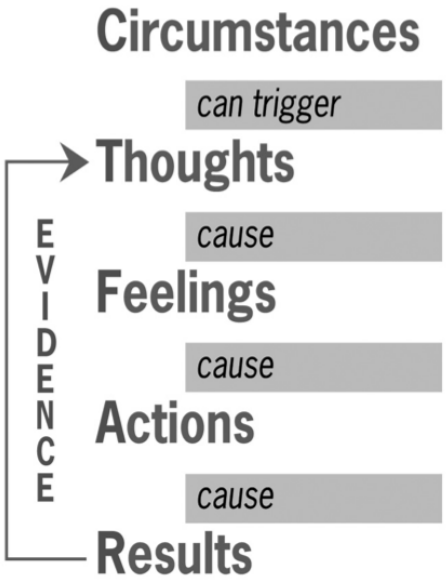
Our thinking can create a lot of self-induced suffering. We as humans are very hard on ourselves and self-criticism can really chip away at our self-esteem and ultimately our happiness. And then there are our thoughts about clients and our patients and our doctors and co-workers. One negative interaction can literally ruin our day!

How easily can we let go of things we can't control? Do we beat ourselves up when things don't go the way we want them to? Can we let ultimately let go of the unrealistic expectations we have for ourselves? Can we let go of the sadness and pain when the outcome is not what we desire?

Managing our minds is a skill that provides us freedom from suffering. Our emotions can become overwhelming, damaging and breed rumination.

The Self-Coaching Model

This self-coaching model can offer relief. Based on cognitive psychology, this model gives us the ability to identify thoughts that are creating negative emotions and then, and only then, we can change those thoughts to ones that make us feel better.



This model is based on the following truths:

- We cannot control the world
- Nothing outside of us has the power to make us feel good or bad
- It is not the circumstances, but our thoughts about the circumstances that create our experience
- We attract what we think about
- Emotions lead to action
- We can't permanently change our results without changing our thoughts
- We don't have to get anything to feel better; we can feel better right now

Circumstances: Things that happen in the world that we cannot control.

Thoughts: Things that happen in your mind. This is where you self coach.

Feelings: Vibrations that happen in your body—caused by thoughts, not circumstances.

Actions: Behavior—what we do in the world. Caused by feelings, determined by thought.

Results: What we see in the world (our lives) as an effect of our actions. The result will always be evidence for the original thought.

Being conscious and choosing our thoughts are the most important components to feeling better.

How to Feel Better

Step 1. Understand that you are not your mind.
Step 2. Realize that your thoughts are not facts.
Step 3. Become aware of what you are thinking.
Step 4. When you don't like the way you feel, change your thought.
"How can I see this differently?"
"How else can I interpret this situation?"
"How else can I think about this?"

Criteria for changing your thoughts.
1. Change your thought to one you believe.
2. And one that feels better when you think it.

RADICAL SELF-CARE

Julie Squires, BA, CCFS, Life Coach

Self-Care: What Actually Is It?

Self-care is hugely misunderstood. We think it's something we have to go to the mall and buy or put in our shopping cart on Amazon.

It might be. But there's a lot more to self-care than consumerism.

Self-care enhances our wellbeing. It's an intention, activity or practice that we do on a regular basis to reduce stress and support our wellbeing.

Self-care is actually pretty basic. Essentially it's taking care of ourselves.

Want to know the best way to feel better? Take care of yourself.
There are 5 areas of self-care:
Mental
Physical
Spiritual
Social
Emotional

Mental

Mental self-care is the balance between stimulating your mind and giving it a break.

Examples:

- Reading a book
- Taking a class
- Listening to music or a podcast (check out the Rekindling podcast!)
- Disconnecting from social media for a while
- Journaling
- Meditation
- Netflix/movies

Physical
Physical self-care relates to our health, nutrition and physical wellness.

Examples:

- Exercise
- Daily lotion
- Staying hydrated with water (at least 64 oz/day)

- Eating healthy
- Going to bed early, taking naps, getting at least 7-8 hours sleep/night
- Dancing when no one is looking(or when they are!)
- Washing your face at night
- Shaving your legs
- Regular doctor and dentist visits
- Taking your meds

Spiritual

This is your personal practice that allows you to follow the values and beliefs that give you purpose. Connecting to something greater than yourself (God, Higher Power, etc). It may be religious or it may not be.

Examples:
- Being in nature
- Yoga practice
- Going to church
- Meditation
- Volunteer work
- Prayer
- Reflecting on your why

Social
Although most associate self-care with being alone, social connection can also be a form of self-care. It can create a sense of belonging and acceptance.

Examples:
- Exercising with others (e.g. spin class)
- Walking with friends
- Starting a book club
- Organizing a family get together
- Taking a sip-n-paint class with co-workers
- Attending a conference
- Meet-ups

Emotional

Emotional self-care helps us understand ourselves more, cope with challenges and develop healthy relationships. When we tend to our emotional needs we cultivate a greater sense of compassion, kindness and love for ourselves.

Examples:

- Journaling about how you're feeling
- Identifying what you're feeling (Use the Feelings & Sensations handout!)
- Saying no to things that aren't good for you
- Saying yes to things that are
- Working with a therapist or coach
- Feeling your feelings
- Connecting with a friend

Think self-care is selfish? How can taking care of yourself be selfish?

What if instead of it being selfish it's our *responsibility* to take care of what we've been gifted. A mind, a body and a spirit.

Start small. Start with the most basic needs you have. And you don't need a lot of time. Think 5-10 minutes.

HOW TO GO FROM JUDGMENTAL TO COMPASSIONATE IN 3 STEPS

Julie Squires, BA, CCFS, Life Coach

Releasing Our Judgment of Self & Others

Humans are judgmental. We are all judging each other and the situation all the time. And there's a very good reason for it, it helps keep us safe. Our primitive brain is designed to keep us safe and alive and therefore judges, s*afe or dangerous.*

There's safety in believing someone else is wrong and you are right, at least according to your primitive brain. If I believe you are "wrong" about something, it makes me "right" and in being "right", there's a perceived gain.

We judge clients and are judged by clients.

They shouldn't be so mean to us.
Clients should be more appreciative of how hard we work to save their pets.
Clients should not complain about how much veterinary care costs.
They shouldn't have a pet if they can't afford it.

Probably sounds familiar. It's a common narrative in veterinary medicine.

There is a cost to being judgmental. It feels horrible it's exhausting and extremely depleting. It for sure leads to compassion fatigue. You can't possible feel compassion and judgment at the same time. And where there is no compassion, there is no connection. We have to connect to pet owners in order to help their pets.

When our brains sense negativity, it creates a stress response in which adrenaline and cortisol are secreted and circulated throughout the body. These hormones are designed to make us run away from a threat not connect. Instead we have to get our brains to a feeling of safety so we can create a connection and thereby serve at the highest level.

Being judgmental is a choice and one we can choose differently. Instead of jumping to conclusions we can take a step back and open our minds up to other possibilities.

When we are pointing our finger at another, there's actually 3 fingers pointing back at us.

Here's how to get yourself out of judgment and into compassion and understanding.

1. Self-compassion
2. Recognize the story your brain is creating.
3. Practice empathy and see where you're "just like them"

WOUND EVALUATIONS AND BANDAGING

Kathleen Ham, DVM, MS, DACVS
Associate Professor, University of Florida

Introduction: Wound healing is a complex event that consists of phases that will occur simultaneously as well as continuously. Most of what is occurring is microscopic, but it is important to try and associate certain aspects of the wound healing process that we can see to these microscopic events. Cytokines, growth factors, and key cell types are going to initiate, maintain, and modulate the healing process. The goal of this lecture will be to review the microscopic events, describe the macroscopic changes, and then apply this information to open wounds and their evaluations.

There are 3 main phases in wound healing; inflammation, repair, and maturation. The inflammatory phase is characterized by vascular permeability, chemotaxis, and cell activation. The proliferative or repair phase has fibroblast proliferation, angiogenesis, and epithelialization. Last the maturation or remodeling phase is hallmarked with reorganization of the collagen and gain in strength. We will discuss each phase in depth.

Following an injury, capillaries and lymphatics are damaged which results in bleeding and lymph fluid accumulation. The vessels will undergo vasoconstriction for about 5-10 minutes after the insult in response to catecholamines, serotonin, histamine, and bradykinin. Next there is vasodilation that occurs with an increase in vascular permeability. This results in transudation of plasma and cells. The most important cell in this stage is the platelet which helps to organize the blood clot and releases cytokines and growth factors. The blood clot acts as a hemostatic plug, barrier to infection and fluid loss, and a substrate for organization of the wound (provisional extracellular matrix, ECM).

The term extracellular matrix is used to refer to the wound bed at various stages in the healing process. Initially in the inflammatory phase the provisional ECM is composed of fibronectin and fibrin that has binding sites for the white blood cells and connective tissue cells that will be migrating into the wound. Later this ECM reorganizes into the granulation tissue during the repair phase. It is composed of collagen (initially type 3, then type 1 predominates), hyaluronan, laminin, and proteoglycans. Lastly the ECM will become the scar that will undergo collagen reorganization in the maturation phase.

The inflammatory phase is characterized by neutrophils and macrophages that enter the wound and provide debridement. Fibrinogen is converted to fibrin which releases fibrinopeptides; these serve as chemoattractants for neutrophils. Neutrophils migrate into the wound within 6 hours. They phagocytize bacteria and debris and release toxic oxygen species that kill bacteria, degrade macromolecules, denatured ECM and damaged cells. Pus or exudate that we see is from the wound fluid, degrading neutrophils and denatured tissue.

Monocytes predominate in older wounds (by 5 days), because neutrophils are short lived. Monocytes are essential cells and become the wound macrophage, can become multinucleated giant cells, or evolve into epithelial cells and histiocytes. Macrophages produce mediators that modulate wound healing; fibroblast growth factor (FGF), epidermal growth factor (EGF), platelet-derived growth factor (PDGF), transforming growth factor alpha and beta (TGFα, TGFβ), tumor necrosis factor (TNF), interleukins, and matrix metalloproteinases (MMPs).

Early the macrophages provide debridement with phagocytosis and later the macrophages help modify the provisional ECM into granulation tissue. Macrophages have good survivability and become the directors of wound healing. They modulate fibroplasia, angiogenesis, and stimulate collagen production with the release of various cytokines and growth factors. The inflammatory phase usually lasts about 3-5 days and is sometimes called the lag phase. This is because there is no gain in strength during this phase.

The transition from the inflammatory phase is marked by invasion of fibroblasts and subsequent accumulation of collagen into the wound. The provisional ECM transitions into granulation tissue by 3-5 days after the injury. Epithelialization and wound contraction also occur. The repair phase is typically from days 5-21, but this is dependent on many other factors.

Granulation tissue fills the defect, protects the wound, is a barrier to infection, provides scaffolding for epithelialization, and contains myofibroblasts. The myofibroblast, a specialized fibroblast, is responsible for contraction of the wound. Granulation tissue can be dark red all the way to pale depending on the amount of angiogenesis and collagen deposition.

Early in the repair phase there will be an in-growth of new capillaries from pre-existing vessels at the wound edges, endothelial sprouting. Macrophages produce FGF, VEGF, and TGFβ which are the main mediators of angiogenesis. Low oxygen tension and increased lactic acid can also stimulate angiogenesis within the wound.

Fibroplasia will occur as mesenchymal cells migrate into the wound. The nearby fibroblasts will proliferate and via expression of integrin receptors migrate. Fibrinolysis occurs as collagenases are needed to help clear the path as the fibroblasts are migrating. Another cell type that was already discussed was the myofibroblast which contains contractile filaments that allow for contraction. The fibroblasts then begin synthesizing the ECM by producing collagen, proteoglycans, and glycoproteins. The collagen is unorganized and accumulates the most during days 7-14 after wounding. The wound gains strength during this phase as the collagen is deposited.

The epithelial cells at the margin will begin to move and migrate using the granulation tissue as a scaffold. These cells will then proliferate working to cover the wound. Epithelialization can be seen as soon as 4-5 days, but it starts 1-2 days after the injury. This process takes place under the scab, which is why scabs fall off. Re-epithelialization may not be complete depending on the wound and host factors (discussed later).

Contraction is visible after 5-9 days after the injury. This is the reduction in wound size. There is intussusceptive growth as the surrounding skin stretches, centripetal advancement. This is predominately due to the myofibroblast and TGFβ1, TGFβ2, and PDGF. The collagen in the wound is not responsible for contraction. The wound will continue to contract until there is negative feedback from contact inhibition or excessive tension. Contraction can result in contracture which is the shortening of the scar tissue which will result in a distortion of function over certain areas of the body, i.e. a joint.

An incised wound that was sutured closed may have complete epithelialization by 24-48 hours. A partial thickness wound that has a variable portion of the dermis can heal by adnexal re-epithelialization which takes ~21 days. There will be immediate migration over the surface of the wound from the wound margins and adnexal structures left in the dermis. If there is a full thickness wound it will heal by wound

contraction and epithelialization. The adnexal structures won't regenerate, and pigmentation may be variable.

The maturation phase is hallmarked by the transition from ECM to a scar. The cellularity in the wound decreases and the collagen fiber bundles become thicker, increase cross-linking (making them stronger), and change orientation along lines of tension. This process takes months to years and the increase in mechanical strength is slow. The MMPs and tissue inhibitor of MMPs (TIMP) work to degrade the ECM, remodel, inhibit angiogenesis, and induce apoptosis.

The gain in strength in an open wound is slow. The most rapid gain in strength occurs between 7-14 days as collagen deposition occurs. An open wound has approximately 20% of its final strength within 3 weeks of the injury. A wound may never have the tensile strength of the original tissue; a scar is only 70-80% as strong as normal tissue. Interestingly, different tissues gain strength at different rates. The sutured wound of the intestine and urinary bladder is 80% of normal tensile strength at 10 days.

So if we approach this in a problem solving format, what are the types of goals we would need through each phase of wound healing? During the inflammation phase we would like to reduce contamination, prevent infection, and clean and debride the wound. Whereas during the repair phase we want to protect the granulation tissue and provide topical stimulants for wound healing depending on the events taking place (epithelialization and contraction). Lastly, during the maturation phase we want to protect the fragile epidermis as it begins to stratify.

As we consider the normal healing process, it is important to take into consideration the factors that can have an impact on the normal healing process. As we look to different treatment options and new methods of wound healing, we examine these factors and try interventions to optimize the healing process. There are 3 categories of factors that have an affect on wound healing; physical, endogenous, and exogenous factors. The physical factors may include things like oxygen tension or temperature. Endogenous factors include hypoproteinemia, altered microcirculation, uremia, liver disease, steroids, or infection. Lastly, exogenous factors such as vitamins and minerals, NSAIDS, steroids, cytotoxic drugs, radiation therapy, and antiseptics can affect wound healing.

Wound Classification Schemes and Wounding Mechanisms: The wound factors that should be listed with each injury include mechanism of the wound, duration, and contamination. We can describe the wound by how it was caused or the mechanism of the wound; mechanical, chemical/ toxin, thermal, radiation, pressure, draining tracts. Examples of mechanical wounds include abrasions, incision or lacerations, degloving or shearing, puncture or projectile wounds and bite wounds. Draining tracts are often the result of infection, sequestrum, implants, foreign body or immune mediated disease. The mechanism allows us to consider the forces that were exerted to create the wound with shear tension and compression forces being the most common. Shear forces usually are created with a small amount of energy to a small area. They tend to be more superficial, but if the object is sharp they can be deep with contamination. Tension forces occur when there is a striking force at an angle with high energy. This usually results in a flap of skin or avulsion with subsequent trauma to tissues from ischemia and contamination. Compression forces are from a striking force with high energy resulting in significant trauma to the tissues from ischemia, shredding and contamination. Breaking the wounds into mechanisms can allow us to characterize a group of wounds based on the effects that they cause including the size, depth, degree of contamination, damage to surrounding tissues and treatment strategies.

We also classify the wound based on the amount, type and time of contamination. Wounds can be considered clean, clean contaminated, contaminated or dirty and they are considered per acute within 4-6 hours, acute or chronic. In veterinary medicine we see mostly acute wounds and there is usually some level of contamination. The difference between a contaminated wound and a dirty wound is based on the amount of time the contamination has been there, if greater than 8-12 hours the contamination has likely progressed to an infection and is now dirty. This again allows us to stratify treatment options knowing that contaminated and dirty wounds should most likely be left open initially.

It is always important to consider patient factors when evaluating the mechanism of the wound. Patients with certain comorbidities may be more at risk of different types of wounds or wound healing complications. The medications list, travel history, nutrition, and past medical history should be carefully assessed. The way to treat a wound is very dependent on host or wound factors and host factors to consider would include diabetes mellitus, hyperadrenocorticism, hypoproteinemia, uremia, anemia, FeLV, or FIV. Once the wound factors have been characterized and the patient factors described a diagnostic wound assessment can be performed.

Diagnostic Wound Evaluation: The first part in doing a diagnostic wound evaluation is to have a clear definition of the anatomy of a wound and to have a wound evaluation form that is used consistently. If the wound is not being thoroughly documented, then subtle changes that indicate complication or lack of progression can be missed delaying wound healing. The parts of the wound include the wound edge, depth, base, surface of wound bed, structures in the wound, pocketing, periwound skin, and effusion.

A diagnostic wound assessment typically requires heavy sedation or anesthesia, so should eb done in patients that are stable. The wound should be covered with sterile lubrication and the skin clipped at least 6-10cm lateral to the wound. After the skin has been scrubbed aseptic technique is used during the assessment. The location of the wound should be recorded, and size is measured in cm; length (dorsal to ventral), width (cranial to caudal), clusters can be measured as 1 wound. The depth is probed in a circular fashion using a clock face to describe the location. The periwound skin is helpful in determining if the wound is infected and if it is sustainable for closure. Features to consider are color, texture, temperature, integrity and pain. Exudate is characterized by type and quantity. If present necrotic tissue can be characterized by type and what connective tissue is involved. The wound edges should be evaluated for adherence to the wound bed and the margins evaluated for thickness, rolling, and fragile epithelium. The wound bed lists exposure of supporting structures and the presence and characterization of the granulation tissue. Lastly, cofactors for non-healing or delayed healing should be assessed; tissue ischemia, infection, osteomyelitis, pressure, necrotic tissue, foreign material, neuropathy, neoplasia, atypical etiologies.

The 6 Goals of Wound Management: When treating a patient with an open wound we aim to heal the patient as fast as possible, have minimal complications, keep cost low, and have minimal morbidity for the patient. Some wounds can be closed right away while others may have a delayed primary closure, and some heal by second intention or with third intention healing. In any wound where closure is not selected immediately proper open wound management should be initiated. The main goal of open wound management is to establish a wound bed free of necrotic tissue or infection. There are 6 steps involved in open wound management; prevent further contamination, remove foreign contamination and debride necrotic tissue, lavage, provide adequate drainage, promote a vascular wound bed, and selection of the appropriate closure type.

When a patient is presented for open wound management they may have concurrent injuries. Therefore, patients with traumatic injuries need a careful patient assessment and the problems should be triaged. The life-threatening injuries should be addressed first. If there is an open wound, the wound should be covered to prevent contamination while the patient is stabilized. We can prevent further contamination by adhering to aseptic technique when addressing the wound. Keep the patients on clean tables and always wear gloves when handling the patient. The administration of broad-spectrum antibiotics is indicated in traumatic wounds.

The wound needs to be cleaned to remove any foreign material. Start by using a sterile lubricant placed directly into the wound. Clippers should be used to remove hair from the skin edges and about 6-8cm around the wound. Chlorhexidene or betadine scrub solution can be used to clean the skin around the wound, taking care not to get the scrub into the wound as it is cytotoxic.

Lavage is used to remove foreign material and bacteria from the wound and surrounding skin. For maximum efficacy the irrigant should be delivered at 8-12 psi. This was originally described using a 35cc syringe and a 19-gauge needle, but more practically a 60 cc syringe and 18 gauge needle, a pressure bag can be used, or bulb syringe and bowl. Lavage fluid should be an isotonic crystalloid fluid such as LRS or normal saline. In grossly contaminated wounds tap water can be used as lavage. The wound can be flushed with an antiseptic such as dilute chlorhexidine at a concentration of 0.05% (1:40 dilution) or povidone-iodine at 1% (1:10 dilution).

The third step is to provide debridement and assess the wound. Strict asepsis should always be employed when tending to open wounds. Instruments that have been autoclaved, sterile gloves, and drapes should be used to prevent nosocomial infection. This is usually when the diagnostic wound evaluation is also completed. An important thing to remember is that the degree of damage to the deeper tissue may not always be apparent from the superficial wound. If the wound penetrates the abdominal cavity, an abdominal exploratory must be completed. If the wound is contaminated or dirty a deep tissue culture should be obtained for culture as superficial swabs are not effective at providing accurate culture results. The criterion that is used for removal of necrotic tissue includes the color, vascularity warmth, and contractility of the tissue. While debriding it is important to avoid disrupting the subdermal plexus as this is the blood supply to the overlying skin. The level of tissue damage may not be apparent until after 24-48 hours, therefore a staged debridement may be necessary. Some wounds may require continual debridement. The types of debridement that can be provided include surgical, enzymatic, mechanical, interactive dressings, or larval.

If a wound is left open, then any effusion produced from the wound will drain onto the bandage. Contaminated and dirty wounds should be left open for this reason. In circumstances where the wound is closed and wounds with a large amount of dead space or a clean contaminated wound should also have a drain. The type of drain selected will depend on the location of the wound, amount of drainage expected, and availability. A passive drain such as a penrose can be used but it is important that the exit site is away from the wound and in a dependent location. Also, the exit site should ideally be covered by a sterile dressing at all times. Active drains such as a Jackson-Pratt drain should exit away from the wound and need to be emptied at routine intervals.

To promote a vascular bed that will be acceptable for wound healing appropriate bandaging techniques need to be employed. You should choose a bandage that will remove the exudates, provide

debridement, protect the surface, and stimulate granulation tissue formation and healing. There are various topical agents that can be used to stimulate healing at the various stages of healing.

Finally, wound closure will be determined based on the various factors previously described. The basic options for wound closure include first intention healing which is primarily suturing the wound edges together. Second intention healing takes place with the formation of granulation tissue, epithelialization and contraction. Third intention healing is delayed closure of the wound after granulation tissue has been formed.

Bandaging Open Wounds: The functions of a bandage include maintaining a moist environment, providing a local energy source, reducing edema, increasing growth factors, improving inflammatory response, improving oxygen content and improving blood flow. The two basic types of bandages used in open wound management include wet to dry bandages and moist wound healing bandages. Choosing the right type of bandage is dependent on the purpose of the bandage (soft tissue injury, orthopedic injury, surgical wound), animal factors (age, health, compliance), and wound factors (locations, depth, surrounding tissue, contamination, concurrent injuries, phase of wound healing).

When considering the different bandaging techniques of the open wound it is important to understand the components of a bandage. The primary layer is in contact with the wound and should be sterile. The secondary layer is the wicking layer and will collect the exudates produced by the wound. The tertiary layer essentially holds the bandage together. A splint or some form of immobilization can also be incorporated into the bandage.

The wet to dry bandage is a commonly used bandage for wounds in the inflammatory and early repair phases. It is useful for wounds that are contaminated or dirty with moderate to large amounts of exudate. It provides mechanical debridement of bacteria and necrotic tissue. The primary layer is a saline dampened gauze. The bandage is changed according to the amount of exudate, but it should be changed at least once a day. Once granulation tissue is present this type of bandage is usually not needed. The disadvantages of a wet to dry bandage are nonselective debridement, sedation is needed for bandage changes, it is painful, they need to be changes frequently, and bacteria can penetrate the gauze.

The moist wound healing bandages are used in wounds during the inflammatory and repair phases. The primary layer is a moisture retentive dressing (examples will be discussed). It promotes an environment where white blood cells survival and therefore provide a more selective debridement. There are decreased infection rates, lower oxygen tension, they are less painful, and there are less frequent bandage changes. Disadvantages of moist wound healing bandages include damage to peri-wound skin and the wound bed due to excessive exudate.

Primary layer options can be divided into 2 groups; nonadherent and adherent. Nonadherent layers are further categorized as occlusive or semi-occlusive and examples include calcium alginate, polyurethane foam, hydrocolloid, hydrogel, telfa pads, petrolatum-impregnated gauze - Adaptic. When to use the various primary layers is dependent on the type of wound, contamination level, phase of healing, and the amount of exudate. Non-adherent dressings are used in moist wound healing, surgical wounds, or

epithelializing wounds. Many of the primary layers can be purchased with topical agents already impregnated.

We can enhance the wound healing with the addition of various topical agents. There are many products available and many companies making new therapies for enhancing wound healing. Many of these products try to target goals such as maintaining moisture, providing an energy source, reducing edema, increasing growth factors, increasing the inflammatory response, and improving antibacterial properties.

A few that are used more commonly include silver, iodine, acemannan, honey, and sugar. Acemannan acts as a growth factor which stimulates macrophages, enhances fibroblast proliferation, increases neovascularization, enhances collagen deposition and epidermal growth. It can be used in partial or full-thickness wounds, burns, or ulcers. Honey (preferably unpasteurized) decreases edema, stimulates macrophage migration, accelerates sloughing of dead tissue, provides an energy source and promotes the development of a healthy granulation bed. Honey is also naturally broad spectrum and can be used in infected wounds. Granulated sugar can also be used in open wound management. It has very similar effects as honey. When using honey or sugar it is important to monitor for hydration or electrolyte imbalances due to the hydrophilic action of the agents.

A newer wound healing method being used is negative pressure wound therapy, also known as vacuum assisted closure (VAC). Vacuum-assisted therapy uses controlled subatmospheric pressure by applying intermittent or continuous negative pressure. This promotes wound healing and accelerates the formation of granulation tissue. The VAC was initially developed to increase wound healing by second intention in patients that were debilitated or those that were poor surgical candidates. An open cell foam sponge is cut to fit over the wound surface. An adhesive dressing is applied over the foam and skin to provide an airtight seal. The sponge is connected to a vacuum pump that transfers the fluid to a drainage canister. This type of bandage allows for moist wound healing.

The mechanism of action is thought to work by altering the physiologic and chemical environment of the wounds by removing fluid and causing mechanical stress. The vacuum removes the wound exudates, reduces tissue edema and enhances blood flow. The mechanical stress caused by the negative pressure increases cell proliferation which will increase granulation tissue formation allowing for migration of keratinocytes across the defect.

The VAC bandage can be applied in many clinical situations. This bandage can be used in the inflammatory or repair phases of wound healing. The VAC is safe to use over areas of exposed bone, tendon, or muscle. The VAC can be used in wounds that have moderate to mild exudate. This type of bandage can be useful in large wounds that are difficult to bandage. The VAC has also been applied in reconstructive surgery cases to aid in graft adherence. The disadvantages of using the VAC include expense, repeated sedation, hospitalization, and pain.

The last thing to consider when applying the bandage is the secondary and tertiary layers. These are dependent on the type of bandage that is being applied which is usually selected based on the location of the wound and the function of the bandage. For wounds on the extremities a Modified Robert Jones is a common and easy way to apply the bandage. If immobilization is needed than a splint can also be incorporated into the bandage. If the wound is on the truck or in an area that is difficult to bandage a “tie-over” bandage or an ioban bandage can be used.

The tie over bandage is very easy to apply and can be used with wet to dry or moist wound healing bandages. The patient should be sedated or anesthetized. Stay sutures are placed around the wound, the bandage is applied and then umbilical tape is used to tie-over the bandage to keep it in place. A protective layer of drape or ioban can also be used to protect the bandage from the environment.

An ioban bandage or a bandage utilizing an adhesive dressing as the tertiary layer is also useful in areas that are difficult to bandage. An adhesive spray can be used on the skin surface to help the dressing adhere. When changing an ioban bandage, leave the ioban adhered to the skin. Ioban can also be used to help relieve tension. The disadvantage of an ioban dressing is that anesthesia may be required for complete removal and the skin can become inflamed after removal.

Monitoring and Complications: It is important to monitor the patient for signs of complications and owners need instruction as to abnormalities to note. If the bandage is on an extremity than the toes can be monitored for spreading apart which indicates swelling. The skin can be monitored for coolness which may indicate ischemia. The bandage should be touched to see if it is wet which could be fluid from the wound, dog licking or external source, but would require a bandage change. If the patient is licking at the bandage it may indicate that there are pressure sores, irritation dermatitis, infection and it should warrant a bandage changes and careful consideration. Common complications include pressure sores especially over pressure points. The skin can develop dermatitis especially between the toes. If the bandage is too tight it will cause swelling and in worse case scenario ischemia whereas a bandage too lose may fall off. Careful consideration is needed at fractures, making sure to span the joint above and below so that the bandage doesn't act as a fulcrum at the fracture site. Skin maceration can occur in wounds that are highly effusive and aren't changed frequent enough. Careful monitoring and immediate action can usually prevent major complications from occurring.
Diagnostic Wound Evaluation: The first part in doing a diagnostic wound evaluation is to have a clear definition of the anatomy of a wound and to have a wound evaluation form that is used consistently. If the wound is not being thoroughly documented then subtle changes that indicate complication or lack of progression can be missed delaying wound healing. The parts of the wound include the wound edge, depth, base, surface of wound bed, structures in the wound, pocketing, periwound skin, and effusion. A diagnostic wound assessment typically requires heavy sedation or anesthesia, so should eb done in patients that are stable. The wound should be covered with sterile lubrication and the skin clipped at least 6-10cm lateral to the wound. After the skin has been scrubbed aseptic technique is used during the assessment. The location of the wound should be recorded and size is measured in cm; length (dorsal to ventral), width (cranial to caudal), clusters can be measured as 1 wound. The depth is probed in a circular fashion using a clock face to describe the location. The periwound skin is helpful in determining if the wound is infected and if it is sustainable for closure. Features to consider are color, texture, temperature, integrity and pain. Exudate is characterized by type and quantity. If present necrotic tissue can be characterized by type and what connective tissue is involved. The wound edges should be evaluated for adherence to the wound bed and the margins evaluated for thickness, rolling, and fragile epithelium. The wound bed lists exposure of supporting structures and the presence and characterization of the granulation tissue. Lastly, cofactors for non-healing or delayed healing should be assessed; tissue ischemia, infection, osteomyelitis, pressure, necrotic tissue, foreign material, neuropathy, neoplasia, atypical etiologies.

ABDOMINAL SURGERY

Kathleen Ham, DVM, MS, DACVS
Associate Professor, University of Florida

Introduction: It is important to have a good understanding of the anatomy of the gastrointestinal tract, understanding the blood supply, ligaments, and relation to other organs systems. This will help in deciphering the diagnostics and help in surgical decision making.

Diagnostics: When working a patient up with non-specific clinical signs such as vomiting, it can be challenging to interpret radiographs when there is not an obvious obstruction. By taking both lateral views the gas can redistribute highlighting a foreign object or you can follow loops of bowel easier. In some instances, contrast can be used to help differentiate functional obstructions from mechanical obstructions. The colon is often gas filled and can be confused with small bowel, a barium enema or a pneumocologram can be used to help delineate the colon. If the radiographs are inconclusive treat the patient medically: rehydration, antiemetics, gastroprotectants and recheck radiographs in 6-8 hours. Patients with gastroenteritis typically start to show improvement within that time, while patients with obstruction may

Localization: Sometimes the clinical signs can help in localizing the segment of bowel that is affected. Dogs that have diarrhea may have an obstruction that is located further aboral, closer to the ileum. Intussusception is also more likely to manifest with diarrhea versus other causes of obstruction. Patients with a pyloric obstruction may have projectile vomiting and a metabolic alkalosis due to the loss of gastric acids. Linear foreign bodies usually start as being partial with intermittent clinical signs and as the intestines plicate the obstruction becomes complete and the patients are sick.

Surgery: Patients should receive intraoperative broad spectrum antibiotics such as Unasyn at induction and every 90 minutes. Antibiotic are usually continued for 24 hours after surgery. A midline celiotomy is performed with an incision made from xiphoid to pubis. An exploratory laparotomy is performed, and the foreign body is located. The bowel is inspected for ischemic necrosis using subjective criteria; color, arterial pulsations, peristalsis, bleeding and texture. Sometimes a perforation will be obvious, if not then experience will help in determining if an enterotomy or resection and anastomosis is indicated. Always use more than 1 criteria to determine viability. If in doubt cut it out, is an expression used in these situations. If the bowel is healthy than a longitudinal enterotomy is made aboral to the foreign body in the non-obstructed bowel. If the bowel is ischemic or perforated a resection and anastomosis should be performed, removing the unhealthy bowel. Bowel may appear much healthier after the foreign object has been removed indicating congestion of the tissue. If the bowel stays dark in color after removal of a foreign object, then it should be removed. If the intestinal wall tears while removing the foreign object it is best to perform a resection and anastomosis.

Key principles to intestinal surgery include; prevent contamination, proper suture material, proper suture pattern, leak testing, and reinforcement. Isolate and bring the intestines out of the abdomen. If the foreign object is in the duodenal flexure, transect the duodenocolic ligament. Prior to creating an incision in the bowel, the segment to be operated should be isolated away from the abdomen; this is another reason why a larger incision may be beneficial. Laparotomy sponges are used to “pack-off” the site or keep separate the clean and contaminated areas. Set a separate set of clean instruments aside, these will be used to close the body one the GI surgery is complete. Ingesta should be milked away from the site of the incision and then the intestine atraumatically clamped using fingers or Doyen forceps.

Bobby pins can be purchased from the store, gas sterilized, and work great for atraumatic occlusion of the lumen.
When transecting bowel for a resection and anastomosis use an oblique angle to improve the blood flow to the antimesenteric edge and to adjust for luminal disparity. Suture material should be of a small gauge, absorbable, synthetic, with strength characteristics similar to the healing of the intestine. For most small animals 4-0 is a good choice, and a taper is preferable; I prefer 4-0 PDS using an RB needle. In small animal patients an appositional pattern with simple interrupted or simple continuous is recommended. A gambee pattern can also be used if there is excessive mucosal eversion.
Once the incision is closed a leak test is performed by inserting a small gauge needle (25g) on an angle into the intestine a few cm away from the incision and saline is injected into the lumen while occluding oral and aboral to the incision. A fluorescein stain can be placed into the saline to change the color to yellow making it more visible. Reinforcement using omentum or serosa can be used to patch the incision site. Some people also like to use sealants over the incision, although there is no evidence this prevents leakage. Staplers can also be used to close enterotomies and anastomoses.
Surgical gloves and instruments are changed to prevent contamination. The surgical site should be locally lavaged to remove any gross contamination prior to being placed back into the abdomen. The abdomen should also be lavaged in case of leakage or bacterial translocation. If the patient has septic peritonitis thorough lavage is recommended. Ideally suction is used to remove the fluid during and after lavage. The peritoneal cavity should have minimal fluid after lavage as the presence of fluid can prevent bacterial opsonization.
Linear foreign bodies: First release the object where is it lodged and then perform 1-4 enterotomies to remove the object without applying excessive tension which can cause perforation. Perforations occur on the mesenteric side of the bowel which is technically more challenging to evaluate due to fat. Up to 40% of dogs already have peritonitis at the time of surgery; therefore, they are more likely to require resection and anastomosis.
Intussusception: If the intestinal viability is good meaning no evidence of ischemia then a surgical reduction can be performed. When the lesion cannot be reduced, necrosis or tumor is present, then a resection and anastomosis is performed. The underlying condition should be treated as well. Enteroplication, a technique where the intestines are loosely sutured together to prevent a recurrence, can be performed if recurrence is suspected. Young patients should be dewormed and any tissue that is resected should always be submitted for histopathology.
If the obstruction is secondary to strangulation, incarceration or a tumor than a resection and anastomosis would be required. When a tumor is present, wide margins are preferred taking 6-10cm of healthy bowel on either side.

Post-operative Care: Patients recovering from intestinal surgery should continue to receive IV fluid therapy to maintain hydration and electrolyte status. Many patients have ileus following intestinal surgery due to the obstruction, surgical manipulation, pain management and electrolyte abnormalities. If the patient is having clinical signs such as regurgitation or inappetence then prokinetic drugs such as metoclopramide or cisapride should be used. In patients with evidence of ileus during surgery, a nasogastric tube can be placed prophylactically to help remove residual gas and fluid following surgery and the tube can also be used to provide enteral nutrition. When the patient is nauseous antiemetic drugs should also be administered. Patients should be managed with injectable opioids immediately following surgery for pain management. When eating they can be switched to tramadol or gabapentin, avoiding NSAIDS. Nutrition is an important aspect of recovery from intestinal surgery and patients are offered food within 24 hours.
A challenge of postoperative monitoring is differentiating major complications such as dehiscence and peritonitis versus ileus. Injury or contamination of the peritoneal cavity induces a profound

inflammatory reaction which starts with vascular permeability and movement of fluid into the peritoneal space. Mesothelial cells, mast cells, lymphocytes, neutrophils, and macrophages react-to and enhance this inflammatory reaction with the production of cytokines and recruitment of more inflammatory cells. Fluid losses into the peritoneal cavity can lead to hypovolemia and hypoproteinemia along with decreased function of the leukocytes resulting in impaired clearance. These changes can further lead to systemic alterations such as respiratory acidosis, decreased cardiac output, hypotension, tissue hypoxia and metabolic acidosis. Bacteremia, bacterial translocation and endotoxemia ensue in patients with a bacterial component to the peritonitis or in those patients with severe ileus, increased intraabdominal pressure, or impaired bile flow. The body's response to this profound inflammatory response can be as severe as pulmonary thromboembolism, disseminated intravascular coagulation, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome. Most animals with peritonitis are systemically ill presenting with anorexia, vomiting, abdominal pain and distention, and fever. There may be palpable abdominal effusion, the patients may be stable or could also present moribund and laterally recumbent. Abdominocentesis can yield fluid for analysis and cytology. An abdominal ultrasound can be used to help with abdominocentesis as well as an assessment of the architecture and echogenicity of the abdominal organs. The white blood cell count of abdominal fluid that has inflammation is >5,000 but is usually much higher with a septic exudate. The presence of intracellular bacteria confirms the diagnosis of septic peritonitis. The glucose concentration of the fluid should be compared to the blood glucose concentration and a finding of the blood-to-fluid glucose concentration difference of >20mg/dL is 100% sensitive and specific for septic peritonitis.
If septic peritonitis is diagnosed after surgery, usually 3-5 days afterwards, the patient should be stabilized with fluid resuscitation as needed. Starting antimicrobials therapy is initially empirical and should include bactericidal antibiotics effective against gram + and – aerobes and anaerobes. A culture and sensitivity should be performed on the fluid to further guide antimicrobial therapies.
Surgery is the mainstay of treatment and the area that is leaking will need to be resected.
The abdomen should be thoroughly lavaged with 200-300ml/kg as a minimum and more commonly lavaging until the returning fluid is clear. Prior to closure of the abdomen a closed suction drain can be placed to help evacuate the exudate that is produced from the inflammatory reaction that will continue. The post-operative care of patients with peritonitis can be intensive with a large amount of monitoring. Patients should continue to receive parenteral antibiotics until they are receiving enteral nutrition. Nutrition is an important concept during the healing process and placement of feeding tubes at the time of surgery should be considered. Fluid therapy needs to be maintained as these patients continue to have ongoing losses of protein rich fluid into the abdominal cavity. Peritonitis, ileus, and surgery all contribute to pain and therefore multimodal therapies are beneficial with local blocks given via epidural, continuous rate infusions of a pure mu opioid and Lidocaine are helpful. Some animals may require transfusions of plasma, red blood cells, or albumin.

Prognosis: The prognosis for gastrointestinal foreign bodies is usually good with reported dehiscence rates of 3-27.7%. Risk factors for dehiscence include low albumin, peritonitis, and a higher band neutrophil count. The cause of the obstruction will influence the prognosis in each case. If the patient has septic peritonitis the prognosis carries up to a 50% mortality rate.

Principles of Urinary Tract Surgery:

When performing surgery on the urinary tract it is very important to have gentle tissue handling. We can achieve this goal with the use of stay sutures and avoiding using thump forceps to grasp or manipulate tissue. If you are using thumb forceps make sure to use DeBakey forceps. The normal bladder heals very quickly with mucosal defects healing within about 5 days and full thickness defects

regaining 100% of normal tissue strength within 14 - 21 days. Monofilament suture material is recommended since it causes less tissue drag in the delicate bladder and fewer bacteria will adhere (as compared to multifilament suture). Nonabsorbable suture is not required and could contribute to cystic calculi formation. The urinary tract heals quickly and therefore it is important to select suture that wont stick around for too long. Suture material that is in the lumen of the bladder can be nidus for infection or stone formation. Ideal suture types include monocryl or byosyn. The holding layer of the urinary tract (excluding kidney) is the submucosa and therefore it is imperative that you capture the submucosa when closing an incision into the urinary tract. The ideal suture pattern to close the urinary tract in small animals is appositional with full thickness bites. Tissue will always heal best when directly apposed. There is no need to place an additional suture line as the urinary tract heals quickly and fully.

Prior to surgery most patients with urinary tract disease should undergo an extensive preopertive work up including thorough physical exam, CBC, serum biochemistry, coagulation panels, urine culture, and blood pressure. Full staging should be performed in animals suspected to suffer from neoplasia. Imaging including radiographs, ultrasound, CT, or MRI can be performed (with our without contrast) to further evaluate the structure of the kidney and other abdominal organs prior to proceeding with surgery. Whenever possible, uremia, blood pressure irregularities, and electrolyte abnormalities should be corrected prior to surgery. An indwelling urinary catheter may be placed to allow quantification of urine production. Maintenance of renal perfusion is critical so pre- and intra-operative hypotension should be prevented or corrected aggressively.

If a cystotomy was created a leak test can be performed to ensure no obvious leakage. This is done by injecting saline into the bladder with a catheter or with a needle and the putting gentle pressure on the bladder while observing the incision. If the patient had radiopaque bladder stones, a post operative radiograph should be performed to ensure all stones have been removed.

Post-operatively intravenous fluids should be continued to maintain renal perfusion and to prevent obstructive blood clot formation within the urinary tract. Analgesia should be provided and animals should be monitored for anemia, oliguria/anuria, and evidence of urinary tract obstruction. Early post-operative management may include serial RBC or platelet counts, serum biochemistry panels, weight and blood pressure measurement, and quantification of urine output.

Hepatic anatomy

It is important to review the anatomy of the liver prior to taking a patient to surgery for evaluation, biopsy or possible resection. The liver sits in the cranial abdomen where it is protected under the ribs. The liver is divided into lobes based on fissures that create these separations giving rise to individual sections. The left liver lobe is divided into the left lateral and the left medial lobes. The quadrate lobe sits on midline and creates ½ of the gallbladder fossa with the other ½ coming from the right medial liver lobe. The right lateral liver lobe and caudate liver lobe are usually fused at their base. The caudate lobe has a caudate process that is dorsal and touches the right kidney and a papillary process which crosses to the left side and is covered by the lesser omentum.

The liver has several ligaments that keep it situated alongside the other organs. The coronary ligament is an extension of the peritoneal covering of the liver and connects the liver to the diaphragm. More prominent are the left and right triangular ligaments attaching the respective lobes to the diaphragm. There is a hepatorenal ligament that attaches the caudate process to the right kidney. The hepatogastric ligament is part of the lesser omentum that connects the porta hepatis to the lesser

curvature of the stomach and the hepatoduodenal ligament connects the porta hepatis to the duodenum. The hepatoduodenal ligament is the ventral border of the epiploic foramen which contains the bile duct, hepatic artery, and portal vein. Temporary occlusion of the hepatic artery and portal vein by compressing the hepatoduodenal ligament is called the Pringle maneuver.

The liver has a dual afferent blood supply with the portal vein contributing 80% of the blood and the hepatic artery 20%. Animals can survive without a hepatic artery but not a portal vein! The hepatic veins drain the liver lobes and feed into the caudal vena cava with the right and central portions being within the hepatic parenchyma. The left hepatic vein can be within the parenchyma but is usually seen between the liver and the diaphragm where the vena cava passes through the diaphragm.

Biliary tract anatomy

Bile is secreted from hepatocytes into the canaliculi and then enters the bile duct system. Hepatic ducts exit the liver and converge to form the common bile duct. Bile is directed up the cystic duct into the gallbladder for storage and modification. Upon eating cholecystokinin is secreted and bile flows from the gallbladder into the cystic duct and then common bile duct through the sphincter of Oddi and out of the major duodenal papilla. The common bile duct passes through the right limb of the pancreas, enters the mesenteric duodenum and travels intramurally 1-2cm before the papilla, which is usually 3-6cm aboral to the pylorus. The cystic artery, a branch of the hepatic artery, supplies the bile duct and gallbladder.

Indications for liver surgery include PSS, elevated bile acids, AV malformations, hepatopathy, focal lesions, cavitary lesions, torsion, and very rarely trauma. Surgical liver disease can be grouped as being parenchymal, biliary, neoplastic, or vascular. We will discuss portosystemic shunts, although keep in mind there are other vascular anomalies of the liver such as portal vein hypoplasia or arteriovenous malformations. Examples of parenchymal diseases include vacuolar disease, necrosis, hepatitis, cavitary lesions (abscess/ cyst), torsion, or hyperplasia. Oftentimes surgical biopsies are obtained to rule out neoplasia and histologically define the types of nodules noted on ultrasound or during explore. Older animals with Diabetes and steroid induced hepatopathies often have vacuolar changes noted in the liver. Amyloidosis may be seen secondary to chronic inflammation or in Shar peis and oriental cats. Hepatitis is classified as being acute or chronic and determining the cellular infiltrate, presence of fibrosis, and mineral content can help guide the therapy. In many instances animals are considered to have a hepatopathy and histopathology is required to classify acute versus chronic, familial, drug associated, infectious, idiopathic, regenerative, or copper accumulation.

Hemorrhage is a large risk when performing hepatobiliary surgery and can be life threatening. Prior to liver surgery a CBC should be evaluated to have a baseline PCV and evaluate platelet counts. A coagulation profile is warranted and a routine part of the preoperative assessment. Depending on the type of liver surgery anticipated, a blood type or cross match should be done beforehand as well as having blood products or substitutes available in the hospital. Patients with impaired bile flow or impaired liver function may have vitamin K deficiencies and therefore vitamin K coagulation factor deficiencies. In some cases the patients may benefit from whole blood transfusions, fresh frozen plasma, and or vitamin K supplementation.

Special anesthetic considerations include the use of drugs undergoing hepatic metabolism should be avoided or reduced in dosage. Non-steroidal anti-inflammatories should be used cautiously as patients with hepatic disease may have increased gastrin levels. During large liver lobectomies, the diaphragm may need to opened; therefore the ventilation requirements would change to accommodate an open chest. The use of alpha 2 agonists should be avoided in patients with pancreatic disease.

Broad spectrum antimicrobials are recommended in patients undergoing hepatobiliary surgery. The most common isolates from the liver seem to enteric in origin. Recommended protocols would include metronidazole and potentiated penicillin, potentiated penicillin and a fluoroquinolone or clindamycin and a fluoroquinolone.

Some patients presenting with acute diseases such as hemoabdomen or biliary tract rupture may require more intensive therapies prior to surgery such as transfusions, fluid and electrolyte replacement, colloid support, glucose supplementation, and continuous monitoring of hemodynamic status with EKG and blood pressure measurements.

Liver biopsy techniques

The histologic assessment of the liver is needed for many diseases that affect the parenchyma of the liver. Ideally a liver biopsy should contain 6 portal areas and therefore need to be of a sufficient size. At least 2-3 liver biopsies should be procured sampling different liver lobes. It is also important to remember that the right division of the liver receives the first branches of the blood supply and therefore may represent a healthier portion of the liver especially when getting biopsies in which vascular anomalies are a concern. Prior to biopsy the technique should be determined and this can be a function of the location, patient stability, and concurrent diseases. Lastly, the number of samples obtained is influenced by the different diagnostic testing being performed. At least 2-3 samples should be submitted for histopathology, whereas a 1cm piece of tissue should be evaluated for mineral analysis, and a 5mm sample for culture, and occasionally a sample for toxicology can be submitted.

The different approaches for sampling include percutaneous, laparoscopy and open surgical. A tru-cut biopsy needle is used with ultrasound or CT guidance to obtain a percutaneous liver biopsy. A 14 or 16 gauge needle is recommended as the 18 gauge is too small. Advantages to this technique include the less invasive nature, usually only sedation or a short anesthetic period is required, no to minimal pain. Disadvantages include hemorrhage, equipment (needle and ultrasound) and small samples.

Laparoscopic liver biopsy is a less invasive technique compared to open surgical biopsies and allows for direct visualization of the liver with more targeted biopsies. Usually 5mm cup biopsy forceps are used to sample the liver from both the periphery and the central portions of the lobes. The scope can be used to observe the biopsy sites for visualization of clotting and if needed gel foam or surgical can be placed onto the biopsy sites to help promote clot formation and hemostasis. This technique may also detect smaller lesions that were missed with diagnostic imaging techniques alone. Advantages include the magnification, visualization, larger biopsy samples, and minimally invasive attributes. Disadvantages include the cost of equipment, need for anesthesia, risk of hemorrhage, and learning curve with the technique.

Open surgical biopsy techniques are maximally invasive but allow for a thorough examination of the liver and abdominal contents. This option is best for a focal disease that may require a liver lobectomy or if there are concurrent abnormalities that need to be addressed. The suture fracture technique or guillotine method is easy and commonly used. A monofilament, absorbable suture is selected and pre-tied with a surgeons throw. A peripheral portion of the liver lobe is placed through the loop and the loop is tied. This crushes through the parenchyma and compresses the vessels and ducts into the suture loop to stop bleeding. Smaller vessels may tear and ooze, but should stop with clot formation. Gel foam or surgical can be placed on the cut surface to aid in hemostasis. Alternatively a punch biopsy can be used to obtain samples from the central portion of the lobes. The punch is depressed into the parenchyma completely and then removed. The portion of liver tissue is removed, but may need to have the medial attachments cut with Metzenbaum scissors. Care should be exercised to not crush the tissue with forceps during manipulation. A piece of gel foam, the size of the punch, can be placed into the defect that was created in the liver to aid in hemostasis. If a larger piece is needed a partial or complete liver lobectomy can be performed. This can be achieved with dissection and ligation techniques, the use of vessel sealing devices, and thoracoabdominal staplers. Risks associated with bleeding are greater with larger liver resections.

Bile sampling

When evaluating a patient for infectious or inflammatory conditions the bile should be collected for aerobic and anaerobic culture and sensitivity. A 25 or 22 gauge needle can be used for sample

collection. This can be performed percutaneous, although much riskier, via laparoscopy or an open surgical approach. The needle is inserted into a healthy area of the gallbladder or it can be introduced through the liver and then into the gallbladder, transhepatic. Drain a large volume of bile to reduce leakage from the needle site. This should be avoided in instances where there is extrahepatic biliary obstruction of primary gallbladder disease. It has been found that bile cultures are significantly more likely to be positive than liver cultures. Potential risks may include gallbladder rupture, bile leakage, and vagal response. Smaller needle sizes help reduce the risk of vagal reactions and acute shock.

The liver has an amazing regenerative ability. About 70% of the liver can be removed, but care should when considering the function of the remaining liver. The hepatocytes undergo compensatory hypertrophy and hyperplasia following hepatectomy.

Acute inflammation is the response to injury of pancreatic tissue. The inflammation can progress to pancreatitis, but is usually reversible. Following the leukocyte infiltration fibrosis occurs and is also indicative of chronic conditions

Following biopsy patients should be monitored for potential complications. Hemorrhage is the most common complication following liver biopsy. The patients can receive crystalloid fluid therapy during recovery and potentially colloid fluids or transfusions as needed. The respiratory rate, heart rate, blood pressure and PCV/TP should be monitored. In patients with small drops in PCV/ TP and no other signs of hypovolemia, no additional surgery is generally required. Patients that are clinical for blood loss, are not responding to medical management, and continue to decompensate should be taken back to surgery to look for hemorrhage. Patients undergoing pancreatic biopsy should be monitored for signs of pancreatitis.

SURGICAL COMPLICATIONS: CLASSIFICATIONS AND DECISION MAKING

Kathleen Ham, DVM, MS, DACVS
Associate Professor, University of Florida

Introduction: Talking about complications is not a very enjoyable discussion. It usually implies some sort of technical error, patient or owner compliance issue, or poor decision making and planning. But despite the best pre-operative planning, compliance, technique, and peri-operative care, complications do happen. Complications can lead to patient morbidity, death, and increased cost, loss of trust of the client and for these reasons everything should be done to avoid surgical complications. There are many different complications and they are often dependent on the type of operation, various patient factors, surgeon factors, and post-operative care. Taking the potential complications into consideration is imperative as a surgeon and although this topic is not fun, it is very important.

Infection: An infection is the invasion of pathogens into the tissue and post-operative infections are a risk for any patient undergoing surgery. About 10^5 bacteria per gram of tissue are needed to establish an infection. An infection can develop in the skin or subcutaneous tissues, at the body wall, within any organ, in bone, on an implant, or within a body cavity or space. The Centers for Disease Control, CDC, uses specific criteria for defining surgical site infections (SSIs). They are divided into superficial incisional, deep incisional and an organ/space SSI. A superficial incisional SSI is within 30 days of surgery is localized to the skin or subcutaneous tissue and clinically has purulent discharge, pain, swelling, redness, heat, and has a positive culture if performed. A deep incisional SSI occurs within 30 days to 1 year (if an implant is present), is in the deep soft tissue (fascia, muscle), and clinically has purulent discharge, the patient may have a fever, pain, abscess, fistula, and a positive culture if performed. An organ/ space SSI occurs within 30 days to 1 year (if an implant is present), is present in the area that was operated, has purulent discharge, abscess, and a positive culture if performed.

The risk factors for developing infections can be categorized as patient factors, procedure factors, pre-operative factors, surgical factors, or post-operative factors. The patient factors would include things like endocrinopathies, pyoderma or distant infections, or obesity. The type of procedure can be defined using the wound classification system. Pre-operative factors can play a role such as early hair removal and the use of peri-operative antibiotics. Surgical factors that may contribute to the development of SSIs include the operating room environment (number of people, ventilation), asepsis, surgical technique, and the duration of surgery. Lastly the post-operative care of the incision can contribute to infections; if the wound is not properly cared for or if the patient is in an environment that is dirty.

The best ways to avoid developing an SSI is with proper pre-operative planning. Careful review of the risk factors should result in actions that will reduce the development of a post-operative infection. Good client communications will result in good owner compliance as well.

Treatment of SSI's require antimicrobial therapy. The antibiotic can be selected empirically, but it's recommended that a culture and sensitivity be performed to guide the therapy for a more efficient and effective course. Depending on the location and condition of the infection, open wound management may need to be employed. Remember the principles of open wound management; prevent further contamination, remove foreign contamination, debride necrotic tissue, provide adequate drainage, promote a vascular wound bed, and selection of the appropriate closure type.

Implants are ideally inert and therefore do not cause any reaction in the body. Unfortunately, if bacteria contact an implant they can cause chronic infections. Bacteria secrete a biofilm while on foreign material in the body which is impenetrable by systemic administration of antimicrobial agents. Therefore, when using an implant, proper planning and review of the risk factors associated with infections must be understood prior to surgery.

Dehiscence: Dehiscence is the breakdown and subsequent opening of an incision. Risk factors for dehiscence include technical error, patient factors, infections, neoplasia, exogenous factors, and self mutilation. Although not intentional, lack of experience and knowledge can have a significant correlation to iatrogenic causes of incisional dehiscence. The type and size of suture should be selected based on the healing anticipated, the tissue, presence of infection, and the size of the animal. Remember that the rectus fascia is slow healing and therefore the suture should degrade slowly. Examples of appropriate suture include polydioxanone (PDS®), polyglyconate (Maxon®), or non-absorbable sutures. Closing the appropriate tissue layers and knowing the holding layer of the tissue being closed is especially important. Remember that the holding layer for the body wall is the external rectus fascia, and if the fascia is not correctly identified subcutaneous tissue may be closed instead resulting in a body wall hernia. Poor knot tying can result in dehiscence which can be more catastrophic if a continuous pattern was used. It is also important to take adequately sized bites of tissue and appose the tissue properly without the suture being left loose or instead being tied too tight. If the sutures are being tied tightly they can cause necrosis and tissue breakdown, in the skin they can cause irritation and potentiate self-trauma. Knowing the appropriate distance between suture placement is important and will vary on the tissue being closed. Clamping or grabbing suture material with instruments will make it weaker and could lead to suture failure.

The appearance of dehiscence of the skin incision will vary depending on the cause and the presence of infection. Occasionally just 1 or 2 sutures may fail, and a small area may be open. Alternatively, the entire incision may be open. The treatment will be directed at closure of the wound which could be immediate or require open wound management first. Dehiscence of the body wall will result in a body wall hernia. The size of the hernia will depend on the cause of failure. Smaller hernias may allow only omentum to herniate which could lead to serosanginous fluid at the incision. Larger hernias can look rather obvious and palpation will reveal the defect in the closure. Occasionally an animal may self mutilate because of the hernia or they may cause the hernia. This leads to evisceration and some animals then consume parts of the viscera. The treatment of a body wall hernia is directed at closing the external rectus fascia. Depending on the severity, the animal may require a simple closure, lavage of the abdomen, or an intestinal resection and anastamosis. Pre-operative planning, experience and practice and good client communication about post-operative care will contribute to avoiding dehiscence.

Dehiscence of an incision of the gastrointestinal tract will lead to leakage of the intestinal contents into the abdominal cavity and septic peritonitis. Other causes of septic peritonitis include contamination from leakage at the time of surgery, contamination or rupture of infected fluid from the uterus (pyometra), urinary tract, or biliary tract, ascending infections, migrating foreign objects, or retained sponges in the abdominal cavity. Septic peritonitis is a severe and life-threatening disease and therefore careful and planned techniques when operating within the abdominal cavity are very important. Take the time to use laparotomy sponges to isolate the structure, use stay sutures and retracting devises to prevent spillage. Local lavage to the contaminated tissue is beneficial and then the entire abdomen is lavaged.

Dehiscence of an incision into the urinary tract results in an uroabdomen and chemical peritonitis. If the urine is infected, then it is also septic. The holding layers for the gastrointestinal tract and bladder are the submucosa. Remember gentle tissue handling, preservation of blood supply, and appropriate suturing techniques.

Seroma: A seroma is the formation and accumulation of sterile fluid under the skin or within a tissue. A seroma may lead to additional complications such as infection or dehiscence, therefore it is important to understand how a seroma may form. The factors contributing to seroma formation include dead space, inflammation, foreign material or irritants, high activity or high motion. Sometimes these are inherent in the location or type of surgery. Techniques that will minimize seroma formation are gentle tissue handling, closure of dead space, minimizing inflammation by keeping tissues moist during surgery and reduced surgery times, using suture material that is appropriate for the tissue type. Good post-operative care with exercise restrictions are also paramount. The treatment for a seroma is exercise restriction, e-collars, bandaging and warm compresses. Occasionally the seroma should be drained (needle versus surgical drain), but care should be used as introduction of bacteria is a risk. A seroma may be very slow to resolve and can take up to 4 weeks.

Hemorrhage: A hematoma is a collection of blood within a tissue or organ whereas hemorrhage is the ongoing loss of blood into a space or body cavity. Hemostasis is a necessary part of surgery as the effects of a hematoma or continued hemorrhage can have serious effects on the patient. The different techniques for hemostasis are covered in another lecture. All attempts should be used to stop any active bleeding at surgery. Unfortunately, bleeding may occur during recovery due to improper ligature technique, dislodgement of clots due to increases in blood pressure, an unrecognized bleeding disorder, or development of a coagulation defect. Prior to surgery review the anatomy and be aware of the blood supply in the area, review hemostasis techniques and have hemostatic agents available if needed, and properly assess the patient for any bleeding disorders or risks of developing bleeding disorders after surgery. The treatment of a hematoma may include pressure bandaging and supportive care. Hemorrhage can be treated with supportive care with fluids, blood transfusions, pressure wraps or re-operation. The decision for medical versus surgical treatment will vary in each case.

Pain: Pain management is an important part of the post-operative care that a surgeon provides. Remember that there are four different classifications of pain (somatic, visceral, sympathetic, and neurogenic) and we have many medications that can be used to target different pain pathways. Patients will have pain from the incision, manipulation of viscera, ileus, implants, sutures, inflammation, as well as many other reasons. Unfortunately, it can be difficult to assess pain in animals. Some signs to watch for include crying, excessive panting, elevated heart rate, tenderness, and reluctance to walk or lay down. Cats tend to hide or become less social. Choosing the right pain management plan will depend on the type of surgery, the patient's ability to tolerate particular medications, and the route and ability to administer the medications.

Ileus: Ileus is the lack of peristalsis of the intestinal tract that can lead to a functional intestinal obstruction. In people, ileus is a common problem after surgery. In dogs and cats, it is less of a problem but may be seen after intestinal surgery, pancreatitis, peritonitis, hypokalemia, intestinal obstruction, drugs (opioids, anticholinergics) or after intra-abdominal surgeries with extended duration. A cat or dog may not have a bowel movement immediately after surgery and this is acceptable for up to 7 days after surgery. Ileus can present a clinical problem if it is resulting in severe abdominal pain or intestinal obstruction. Signs of ileus may include vomiting, abdominal pain, lack of borborygmus on auscultation,

and inappetence. Good surgical technique and pre-operative planning can help reduce the incidence after surgery. Treatment for severe cases includes supportive care with fluid therapy, prokinetics, correction of any underlying problems (potassium), or discontinuing medications. Walking can also be helpful to stimulate normal gastrointestinal motility.

Stricture: A stricture is the narrowing of an opening, lumen, or passageway. A stricture is usually due to poor wound healing (ischemia) and improper technique. Certain anatomic locations are more prone to developing strictures such as the esophagus or ureter. Techniques to help decrease the chance of stricture formation include gentle tissue handling, proper suture selection and utilization, and good tissue apposition. There are also techniques of closing incisions to improve the diameter of the lumen versus narrow the lumen. Signs of a stricture will depend on the location of the stricture. Treatment can be either medical or surgical and this decision will be based on the clinical signs, location and previous surgery. For example, a perineal urethrostomy performed in cats with urethral obstruction can lead to stricture formation. This is usually due to inadequate dissection, too large of suture, failure to close the mucosa to the skin or self-trauma following surgery.

Disease/Procedure Specific: There are certain cancers that can cause either local or systemic effects that may alter a surgical plan. For example, a thyroid carcinoma will release cytokines that cause a local coagulopathy which can lead to substantial blood loss during surgery that may require a blood transfusion. A hemangiosarcoma can cause cardiac arrhythmias that require careful monitoring during and after surgery. Inflammatory diseases (pancreatitis, peritonitis) can cause systemic effects and lead to severe complications after surgery such as hypotension, SIRS, DIC, ARDS, and MODS. Dogs undergoing upper airway surgery are at risk of pharyngeal swelling and aspiration pneumonia post-operatively; therefore, we must monitor them closely for normal respiratory patterns.

Checklists and damage control: For decades many industries, for example the airline industry, have used checklists to lower error and complication rates and ensure safety. Checklists have recently been introduced into both human and veterinary medicine and have been found to improve patient safety and reduce medical errors. Implementing a checklist in practice can be practical and adaptable to the needs of the clinic. When errors or complications occur, it is very important to manage these directly and openly. Team meetings and morbidity mortality rounds can be very beneficial to creating an environment that favors open discussion rather than punishment. Meeting to discuss complications leads to making changes and alterations in procedures to improve outcomes.

Mindful Communication

Laura J. Smallwood, DVM DACVIM (SAIM) RYT-200

What we say, why we say it, and how we say it matters. Every time we communicate, whether by speaking or in writing, we either foster connection or cause harm. The words we choose, our intention in communicating, and the tone of how we deliver those words all play out in the effect that those words have on others, on ourselves, and on our families, and communities. In the words of Desmond Tutu "Language is very powerful. Language does not just describe reality. Language creates the reality it describes."

In his book, *Say What you Mean: A Mindful Approach to Nonviolent Communication*, Oren Jay Sofer writes that **"True dialogue is more than the mere exchange of ideas. It is a transformative process based on trust and mutual respect, in which we come to see another in new and more accurate ways."** This statement points to the possibility that communication, any communication, offers the possibility of strengthening our connection to one another in ways that create understanding—even when we disagree.

At its simplest, mindful communication means bringing full attention to our conversations—full attention to our own thoughts and emotions, to the words and emotional cues of the other person, and to our typical inclinations when we are engaged in conversation.

In this workshop, participants will have the opportunity to explore communication from the perspective of mindfulness. Topics to be explored include:

- The role of presence in communication and strategies for staying focused and present.
- Curiosity and care as attitudes that foster mutual understanding
- The role of needs and emotions in conflict and communication
- Strategies for working with nervous system activation in the context of communication
- The role of self-awareness in aligning verbal, paraverbal and non-verbal cues
- Ethical considerations in communication

Mindfulness and Communication

Mindfulness can be defined as the capacity to pay attention to one's moment to moment experience with openness and curiosity. Mindfulness is a natural capacity that we have as humans and also a skill which can be strengthened with a bit of patience and practice. As it turns out, not only is mindfulness a key communication skill but communication also provides many excellent opportunities to practice mindfulness!

In his book, *Say What you Mean: A Mindful Approach to Nonviolent Communication*, Oren Jay Sofer offers a three-step guide to cultivating mindfulness in the context of communication.

1. Lead with Presence
2. Come from Curiosity and Care
3. Focus on What Matters

Each of these skills is supported by the state of mindfulness which is, as defined by Jon Kabat Zinn, is "…paying attention in a particular way: on purpose, in the present moment, and without judgement." Paying attention in this way supports presence, a curious and caring mindset, and the capacity to skillfully focus on what is most important in the moment.

Presence

Giving someone our full attention is the most precious gift we can give another person and having someone give us their full attention is the most precious gift we can hope to receive. However, when we begin to pay attention to the quality of our conversations, we might notice that full attention is as rare as it is precious. How often do we find ourselves having a conversation with someone who is checking their phone or glancing behind us to see what else is going on in the room? How often are we that person?

Being present for conversations and sustaining that presence isn't easy. The environments in which we live and work are filled with distractions. There are also many internal distractions that can come into play. In communication, some of the ways distraction might show up include mind-wandering, multi-tasking, or thinking about what to say next while the other person is talking. When we are aware of this, attention oscillates back and forth and does so at a cognitive cost. We miss important details that can lead to missteps in our conversation, misunderstandings, and even conflict.

Oren Jay Sofer defines presence as the "embodied awareness of our direct sensory, mental and emotional experience". In other words, when we are fully present, we are able to monitor what we are observing, what we are thinking and what we are feeling in real time. This is made possible by the capacity of meta-awareness which is what allows us to observe what we are thinking, what we are feeling, and what we are sensing—separate from the raw experience of thinking/feeling/sensing. Meta-awareness is what underlies not only our capacity to be self-aware but also the capacity to be aware of when we are not aware—when we are lost in thought, or flooded by emotions, or not paying attention to pertinent sensory information. Meta-awareness lets us know when we've "left the building", as it were, so that we can course-correct and return to mindful presence.

But presence is hard when we are face to face with another person. Some of the reason for this include:

- We feel very vulnerable when we are face to face with another human being because our evolutionary biology is gauging the situation— is this friend, foe or mate?
- Social engagement activates the sympathetic nervous system—triggering fight or flight and causing us to lose focus and connection.

- We are often distracted, which also causes us to lose the connection. Sometimes the distraction is by choice, such as when we multi-task with communication—checking text messages during a meeting or continuing to work on a medical record while responding to a question from our nurse for example. Sometimes it is difficult to maintain focus because of general distraction in the workplace—ringing phones, movement. Sometimes we are distracted by our thinking minds, lost in thought and only halfway paying attention.

To "Lead with Presence" requires a commitment to mitigate distractions—setting aside/turning off, retreating to a quiet environment when possible and a commitment to arrive brining focused attention to the encounter.

Curiosity and Care

According to Oren Jay Sofer, the second step in mindful communication is to "Come from Curiosity and Care". This step has to do with the intention we set for our interaction with others. This intention is also explicit in the practice of mindfulness which asks us to be curious and open to whatever arises—sensations, emotions, thoughts—without judging the experience. In the same way, we can be curious in the context of communication, loosening up on the tendency to see what we expect (or want) and making space for the unexpected and unwanted. Letting go of judgement is difficult, especially since many of our judgments percolate below the level of consciousness and the brain is wired to make judgments based on past experience. Curiosity, however, offers an antidote to this past conditioning by offering a freshness to experience.

Care can be viewed as goodwill connected to empathy. Oren Jay Sofer writes that "Care means that we are open to being affected by what we learn, that we are committed to seeing the other person's humanity, and that we are willing to include their needs in the situation rather than be rigidly fixated on getting what we want in exactly the way we want it". Care is imbued with the attitudes of kindness and compassion. Kindness can be defined as the heartfelt wish for the well-being of another that emanates from a world view that all beings, without exception, are deserving of wellbeing. Compassion can be defined as kindness in the face of hardship and suffering where the wish for the other is that they be free of suffering. The capacity to view another as someone with whom we have a great deal in common (99.9% of our genetic make-up for starters!) is key to both kindness and compassion. When we recognize all that we have in common—a finite time on earth in these human bodies, our shared needs, and lives that are a progression of ups and downs/laughter and tears,

What Matters Most?

The third step of Oren Jay Sofer's approach to Mindful Communication is to focus on what matters most. Identifying what matters most in the context of dialogue is challenging and, moment to moment, what's important may vary. It might be the activation of the nervous system. It might be the arising of emotional cues. It might be the arising of empathy and connection.

In the context of the non-violent communication framework, however, there is one aspect of human experience that underlies much of what arises in the context of communication. That aspect of human experience is needs.

Needs reflect universal human values that we all share and all actions, including words, can be viewed through the lens of our longing to fill a need. Needs can be categorized in various ways. Most of us are familiar with Maslow's Hierarchy of Needs: physiological needs, safety, love, esteem and self-actualization. However, needs can have far more nuance than this this hierarchy would suggest and despite what was previously thought, the needs that we are trying to meet actually span this hierarchy. An extensive (though not exhaustive!) inventory of human needs is published on the Center of Non-Violent Communication's website. https://www.cnvc.org/training/resource/needs-inventory

Even though needs are universal, needs that are met and unmet are constantly in flux and, even if two people are trying to meet the same need, it is likely that their strategies will vary. This is key. Needs are integral to our shared human experience, but out strategies to meet those needs are personal. We might think of strategies (the things we think, say, and do) as the visible tip of the iceberg with needs lying below the water. We often mistake strategies for needs. "I need chocolate" is a strategy. Comfort is a need. "I need you to be more attentive" is a strategy. Connection is a need. Strategies are not always skillful, particularly if we are woefully unaware of what need we are actually trying to meet. And it is at the level of strategy that conflict and a sense of separation from others arise.

The good news is that between needs and strategies lie our emotions. Emotions arise in response to something that we perceive as important and are keys to our needs. Emotions like contentment, appreciation, happiness, playfulness, and enthusiasm arise in response to needs being met. Emotions like anger, discouragement, fear, boredom, confusion arise in response to needs not being met. When we meet our emotions with awareness, they provide vital clues that allow us to discern what need underlies their arising. For an inventory of human emotions in the context of met and unmet needs, visit https://www.cnvc.org/training/resource/feelings-inventory .

Likewise, when we employ empathy in the context of communication, the emotional experience of another can provide vital clues to the unmet needs that underly their actions. Becoming familiar with our own emotions and underlying needs is key to being able to connect to the feelings and needs of others.

Empathy and Mindful Communication

Empathetic listening includes perspective taking, letting go of enjoyment, brining attention to emotions and underlying needs, and communicating one's understanding in some way. This can include simply giving someone your wholehearted undivided attention, paraphrasing back to them your understanding of what they are sharing, offering an empathic reflection or guess

as to what they might be feeling, offering an empathic expression of how their words are landing for you, or offering empathic support—physical touch for instance.

There are many common communication habits that can shut down empathic connection. Among them are criticizing, lecturing, giving advice, probing, and one-upping. Some habits that seem like empathy but also can shut down empathic connection include giving advice, praising, reassuring, and sharing a similar experience. All of these get in the way of empathy because they shift the focus from other to ourselves. They also compromise agency—sending the message that I know better than you, I can't trust you to figure this out for yourself. That said, advice, praise, reassurance, or sharing of a common experience can be an empathic response if offered in a timely way with the explicit or implicit consent of the other. In other words, once you have insured that the other person feels fully heard, and is open to an empathic response, one of these may be appropriate.

Mindful Guidelines for Communication

In addition to cultivating the mindful communication skills of presence, curiosity, care, and the capacity to discern needs, there are some additional guideline around the choices we make around what we communicate that can be helpful. These include.

1. Speak the truth. Do not speak falsehoods.
2. Speak with the intention of being helpful, not divisive.
3. Speak with kindness—without harshness or cruelty, non-harming.
4. Speak with sensitivity to context—timely, and not idle.

Speaking the truth applies to the spoken word, the written word, and what we share on social media. The viral spread of misinformation and disinformation makes speaking the truth an especially critical guideline. Before you speak, write, or share, ask yourself, "Do I know, really know, that this is true?"

Speaking with the intention to be helpful, asks us to question whether the intention behind what we write, say, or share is the intention to foster understanding and connection.

Speaking with kindness does not mean that we don't have difficult conversations. We can bring truth and the intention to create understanding to a difficult conversation in a way that is also kind. An example of this might be a difficult conversation during a performance review. Or with a significant other.

Speak with sensitivity to context. This asks us to consider "idle" talk—the things we say without much thought, the chatter that can make up much of our day. How often do we unintentionally say something that may not be true or that is divisive (i.e., gossip)? How often do we say something that is harsh or unkind (i.e., venting)? Mindful awareness in these moments can help us choose to say things that we know are true, that are helpful, and that are kind—or to say nothing at all.

Key Points

- Communication either fosters connection of causes disconnection
- The mindful approach to communication (Oren Jay Sofer)
    - Lead with presence
    - Come from curiosity and care. (Care can also be defined as kindness)
    - Focus on what matters (needs not strategies)
- All humans share the same needs. The needs that we are trying to meet at any given time and the strategies we engage in to meet those needs vary. Conflict arises around strategies. When we can connect on the level of needs, true dialogue is possible.
- Mindful guidelines for Communication: Truth, Intention to be helpful, Kindness. Sensitivity to Context.

References and Recommended Resources

Say What You Mean: A Mindful Approach to Non-Violent Communication by Oren Jay Sofer, 2018

Oren Jay Sofer Mindful Communication Website https://www.orenjaysofer.com/

Center for Non-Violent Communication https://www.cnvc.org/

"The Dharma of our Speech: The Lessons of Relationship and Community" in *The Dharma of Modern Mindfulness* by Beth Ann Mulligan, 2017

"Communication and Connection" in *Real Happiness at Work* by Sharon Salzberg 2013

Attending: Medicine Mindfulness and Humanity by Ronald Epstein, M.D. 2017

Care and Monitoring of the Critical Patient

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette, Indiana

Technicians will be asked at some point in their career to monitor a critical patient. These patients require a high level of care, concentration, and critical thinking skills. Technicians must be comfortable with monitoring these patients and using their brain to problem solve and alert the clinician when patient parameters change.

One of the most critical patients is a patient on the critical care ventilator. Most general practice and some specialty facilities do not have critical care ventilators, but the process of caring for a ventilator patient can be translated to any critical patient. Everything that a technician has learned in school is used on these patients. Critical patients are usually obtunded or heavily sedated. Multiple monitoring devices should be used to keep an eye on the patient's vitals, hydration status, ventilation status, and organ function. Documentation is also critical. Every change in a patient's status should be documented and vitals should be taken on the patient frequently.

The patient's airway should be assessed first. Ventilator patients should be intubated for as long as possible. Sedated patients have decreased ventilator drive and are at risk for regurgitation and aspiration. These patients are usually on multiple medications to help decrease their respiratory drive so the machine can breathe for them. Sterile technique should be used to place the endotracheal tube to make sure that there is minimal bacteria that enters the airway. One of the major complications of a mechanically ventilated patient is hospital acquired pneumonia due to bacteria being introduced by the endotracheal tube. A pulse oximeter should be placed on the patient to make sure that the patient is appropriately perfusing its organs. Capnography should also be used to make sure the patient is ventilating appropriately. A normal ETCO2 reading is 35-45 mmHg. Suctioning and humidifying of the endotracheal tube should be performed every four hours, or as needed, to prevent mucous secretions from clogging the endotracheal tube.

Hopefully, the patient had already had venous access. If not, a peripheral catheter most likely will not be enough for these patients. Triple or quadruple lumen central lines should be placed. These will help with the multiple medications that these patients are on as well as possibly be used for intravenous nutrition and blood sampling. These can be placed in the jugular veins, saphenous veins, medial veins, and ombobracheal veins. Ombobracheal veins are very hard to see, so I would suggest to go to other veins first. If placing a central line in a jugular vein, a radiograph must be performed to make sure that the catheter is placed in the cranial vena cava right above the right atrium.

An arterial catheter should be placed in these patients. These can be placed in the dorsal pedal artery, femoral artery, or coccygeal artery. Use caution in placing a catheter in the femoral artery because this can be hard to secure to the patient. An arterial blood sample can be used

to assess patient ventilation and oxygenation. Arterial catheters can also be used to more accurately assess blood pressure. Sometimes, especially in these critical patients, you are unable to place an arterial catheter because their blood pressure is so low. With these patients, you must wait until you can feel the artery until you can place a catheter.
A multiparemeter unit should be placed on the patient to assess heart rhythm, ETCO2, invasive blood pressure, oxygenation, and temperature. The ECG clips should be placed on "sticky" pads to ensure that the metal clips do not cause skin damage. There are multiple brands of these to choose from, choose what is best for your practice.

A urinary catheter should be placed in these critical patients. Technicians should use a long-term urinary catheter, such as a foley, and place with sterile technique. A collection bag should be placed on the catheter to ensure that we can measure the amount of urine that the kidneys are producing.

The patient should also be placed on a properly cushioned area. When it comes to critical patients, it is easier on everyone if the patient is placed on an elevated table. This way, you can get around the patient on all four sides. These patients are prone to pressure sores especially when they start losing muscle mass due to inactivity.

Once the patient is hooked up to all monitoring devices, there are multiple areas of the patient to consider. The patient's airway, mouth, eyes, ears, urinary system, and gastrointestinal system must be cared for. The technician must also preform physical therapy.

Airway care

If the patient has an endotracheal tube, it must be properly cared for to make sure that the patient does not acquire hospital born pneumonia. The endotracheal tube must be replaced every 24 hours with sterile technique. In a recumbent patient, you must inflate the cuff to ensure that the lungs inflate appropriately. One complication of the cuff being inflated, or over inflated, could cause tissue necrosis of the trachea. It is suggested to deflate the cuff and reposition the endotracheal tube every four hours to prevent tissue necrosis. These patients also need humidifcation of their airway. This can decrease mucous viscosity and decrease tracheal inflammation. To do this, Technicians can instill a small amount of sterile water into the endotracheal tube and then suctioning the endotracheal tube after. Suctioning of endotracheal tube is necessary to prevent mucous buildup and occlusion of the airway. This should be performed every 4 hours or on an as-needed basis.

Oral Care

Patients that are recumbent and have endotracheal tubes or tracheal tubes in, are at an increased risk of ulcers in their oral cavity. To prevent this the whole mouth must be kept moist. The technician must suction out all mucous and debris. Use a different suction catheter than the one used for the tracheal tube. This will prevent bacteria from being introduced into the

lungs. A glycerin solution can then be used to prevent drying out the tongue. The tongue can also be covered with a glycerin soaked gauze. Do not wrap the tongue as this can cause rannula's to form. The pulse oximeter probe should also be taken off the tongue and moved to a different area to prevent necrosis. This procedure should be done every four hours.

Eye Care

Critical patients that are recumbent are at an increased risk of eye ulcers because they cannot blink. Artificial tears should be placed on the eyes every two hours. Goggles can also be used to seal off the eye from the environment. Most clinics do not have goggles, so trying to keep the eyes closed with tape could also be used. Technicians should check for ulcers in the eyes at least once a day.

Urinary Care

Urinary catheters should be cleaned every 8 hours to prevent infection. This included flushing the prepuce with a dilute chlorhexidine solution and wiping the lines associated with the urinary catheter. The urinary bag should be emptied every four hours to assess kidney function. A technician should calculate how much fluid is going into the patient and how much fluid is coming out of the patient to assess kidney function and hydration status. Patients are at a higher risk of acquiring urinary catheter infections, so keeping the catheter clean will help keep the infections at bay.

Gastrointestinal/Nutritional care

Critical patients need nutrition. This can be delivered via central line by parenteral nutrition or via nasogastric or nasoesophageal tube by liquid diets. Do not place non-sterile nutrition through the central line. This can cause infections in the blood stream and create more complications for the patient. Most likely, these patients will have diarrhea. Technicians must keep the patient clean and dry. Keep a close eye on the color of the fecal material. Black or bloody stool can indicate a more serious complication and will need more attention.

Physical Therapy

Physical therapy is very important in critically ill patients. These patients lose muscle mass quickly and can acquire pressure sores and ulcers. Passive range of motion and rotating of the hips should be done every four hours. Patients can get out of the hospital quicker the sooner physical therapy is started.

Critical ventilator patients can be very time consuming and technicians will have to think and troubleshoot their way through many organ systems of these patients. There are multiple drug calculations and every part of nursing care that technicians have ever learned about. When a critical patient walks out the door, it can be a very rewarding experience for the whole team.

References:
Battaglia, Andrea M., and Andrea M. Steele. *Small Animal Emergency and Critical Care for Veterinary Technicians*. 3rd ed., Elsevier, 2016.
Silverstein, Deborah C., and Kate Hopper. *Small Animal Critical Care Medicine*. St. Louis, MO: Saunders/Elsevier, 2009

Advanced Procedures for the Veterinary Technician

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette, IN

Veterinary Technicians can do advanced procedures! There are many different technical skills that we can do without the guidance of a DVM. In this lecture we will be going over multiple ways to give intravenous fluids, how to place and maintain nasogastric and nasoesophageal tubes, how to place and maintain urinary catheters, maintain tracheostomy tubes, chest tubes, and other drainage tubes.

Alternative Routes of Peripheral Intravenous Fluid Administration

Dorsal Pedal

The dorsal pedal vein is readily accessible on most animals. It is located on the dorsal aspect of the limbs. In our ICU we typically use the dorsal pedal vein in the rear limbs rather than the forelimbs because that location seems to be more comfortable for the patient. The dorsal pedal vein is hardly ever used as a first line location to place a catheter.

Advantages

The dorsal pedal vein is hardly utilized in practice. It is usually untouched by previous catheterization attempts. This becomes important in situations when other veins have had a catheter in them. Experience in the intensive care unit has shown that use of this vein does not cause as many fluid pump problems (such as flow occlusion) as seen with use of the lateral saphenous vein.

Disadvantages

A dorsal pedal catheter requires excellent nursing care. Since the catheter is in the back limbs and close to the ground, environmental contamination is a higher risk than with a cephalic or saphenous catheter. These catheters should not be placed if the patient is incontinent or having diarrhea as the dorsal pedal region commonly becomes soiled. If the catheter becomes contaminated, it should be removed.

Intraosseous catheterization

Intraosseous (IO) catheters are an easy way to gain access to veins via the bone marrow. They are most commonly used in very small patients and exotics in which venous access is impossible, or as an alternative to a venous cut-down procedure in larger animals that anyone is unable to get venous access into. There are multiple places to place an intraosseous catheter: the trochanteric fossa of the femur, wing of the ilium, proximal humerus, and lateral aspect of the tibia are commonly cited locations in animals. Fluids and medications can be given through the catheter. The flow rate depends on the size of the bone marrow cavity and partially on the size of the needle placed. The maximum rate at which fluids can be administered through an intraosseous catheter is reported to range between 11mls/min to 24mls/min depending on size of the bone.

Procedure

Intraosseous catheters are used when a vein is not accessible. Briefly, placement requires sterile gloves, a scalpel blade, 2% lidocaine, a spinal needle or hypodermic needle (at least 22g or larger), a T-set adaptor, injection cap, bandage material, and saline. The area for catheterization is clipped and aseptically prepared. Then a small amount of 2% lidocaine is

injected under the skin prior to making a stab incision over the area that has been blocked with lidocaine. The needle is placed in the bone with a twisting motion. If you have effectively engaged at least one cortex of the bone, you should be able to move the limb itself when moving the needle.

Flush the catheter once the needle is in place; in a normal intraosseous catheter there will be some resistance. If the tissue around the catheter swells, the catheter is not placed correctly. Saline flush volumes are 5-10 mL or more to properly identify if there is saline deposition out of the marrow cavity or from a misplaced catheter tip. If placement is still questionable, two view orthogonal radiographs can easily confirm the location of the catheter. After placement, a bandage and/or tape should be placed around the catheter to keep the area clean and help prevent dislodgement.

Advantages

Intraosseous catheters can be placed if no venous access is available. Bones never collapse during shock, so intraosseous catheters are always available to administer fluids, blood products and medications. Medications and fluids take about 10 to 20 seconds to reach the heart after being administered through an IO catheter.

There is also an IO device on the market called the "EZ-IO®" system which uses a 15G needle placed into the bone via a drill-like device. The needle comes in varying lengths including 15mm, 25mm, and 45mm. This system is very easy to learn and quick to use. In one study, it took 1.2 minutes to place an IO catheter using the EZ-IO® System compared to the 10.7 minutes it took to place a central venous catheter.

Disadvantages/Complications

While many different drugs and fluids can be administered safely through an IO catheter, there are a few things which should NOT be administered IO. Administration of hypertonic saline has been reported to cause soft tissue necrosis at the insertion site and venous thrombosis. Avoid giving drugs with reported side effects of bone marrow suppression or toxicity directly into the bone marrow cavity via an IO catheter (example: chloramphenicol, sulfa drugs or chemotherapeutic agents). Total parental nutrition is not recommended to be given through an IO catheter due to its hypertonicity.

Bone fracture can occur during placement. Osteomyelitis and infection can also occur due to contamination of the insertion site or as a reaction to medications. The intraosseous catheter can become dislodged once the patient becomes more mobile. Place an IV catheter once this happens. The placement of an intraosseous catheter can also be painful. Post-placement analgesics should also be considered.

Central Venous Catheters

There are multiple different types of central venous catheters that are available for use. A few examples are the "through-the-needle" catheters and "over-the-wire" catheters. These catheters can be placed in the jugular vein, saphenous vein, medial femoral vein, and omobrachial vein. These veins allow access to the vena cava which is useful in fluid therapy, total parental nutrition, drug administration, blood product administration, blood sampling, and central venous pressure monitoring.

Procedure

The two most common techniques for placing central lines are the Seldinger technique (aka. the over-the-wire technique) and the through the needle technique. In both techniques, the patient is first clipped and aseptically prepared for catheter insertion. For the *Seldinger technique* a regular over the needle catheter (introducer catheter) is first inserted into the selected vein. A guidewire is then placed into the catheter; subsequently, the introducer catheter is taken out of the vein with the guidewire left in. Next, a dilator is placed over the wire and into the vein for a few seconds and removed to open up the vascular wall large enough to allow for catheter placement, leaving the guidewire in place. The central line is then placed over the guidewire into the vein to the desired length. To truly be a central line, the distal tip of the catheter should sit just before the right atrium.

The *through the needle technique* differs in that a large bore needle is first introduced into the vein and the catheter is then threaded through the needle until the desired length is inserted into the patient. The needle is then removed from the neck while leaving the catheter in place in the vein. The exposed needle is covered and remains outside the vein.

Advantages

There are multiple advantages to having a central venous catheter (CVC) placed into a patient. Central venous catheters are a valuable tool to use for repeat blood sampling. Central venous catheters also are used for administration of fluids, blood, total parental nutrition and medications and to measure central venous pressure. These catheters can remain in the patient for many weeks as long as no complications arise. Catheters placed via the Seldinger technique also come with multiple lumens which allows for dedicated ports for blood sampling, drug administration, fluids and nutrition.

Disadvantages/Complications

One complication is misplacement into accessory veins. Therefore, a radiograph should be used to confirm placement. Other complications include occlusion due to movement of the patient and clotting within one or more of the lumens. In one veterinary study, the most common complication was clogging of one or more of the lumens.

Peripherally Inserted Central Catheter

Peripherally Inserted Central Catheters, or PICCs, are a useful tool when a central line is not feasible. They differ from other central lines because they are placed into a peripheral catheter site such as the lateral saphenous vein and then advanced until the catheter tip is centrally located in the vena cava. As with any central line, PICCs are able to be kept in place for an extended period of time; in humans they can be used in outpatients.[15] In one human study, the average time for a PICC to stay in place was 40.5 days.[15] There are limited studies of PICC lines in veterinary patients.

Procedure

Placing a PICC is similar to placing a central venous catheter. In most cases, since the vessels are smaller than the jugular vein or other central vessels, the Seldinger technique is used. If the vessel is large enough or the patient is small enough, a longer catheter can be placed in an attempt to reach the right atrium from the peripheral site. A PICC line typically comes with a thin internal stylet to add stiffness when feeding the line; this should also be

removed once the line is in place. The PICC line is then secured. A radiograph can confirm placement.

Advantages

A PICC line can be easily placed in a general practice setting. As with any centrally placed catheter, PICCs allow easy access for blood sampling, blood/fluid/total parenteral nutrition administration, chemotherapy, and other medications.

Disadvantages

PICC lines are typically of very long length but thin diameter, there is also increased resistance to flow which can make blood sampling from these lines more difficult. Human studies report multiple other complications such as central-line associated bloodstream infection, occlusion, and dislocation. There have been minimal studies about the disadvantages of PICCs in veterinary medicine but shortcomings similar to those in humans would be expected.

Catheter modifications for neonates/pediatrics

Neonatal and pediatric emergencies are common in veterinary practice. Most neonates presenting on an emergency basis need fluids. Fluid therapy can be given in two ways: intravenously and intraosseously. It is not recommended to give neonates/pediatrics subcutaneous fluids for emergency resuscitation.

The *jugular vein* is the most common vein to use in neonates. A temporary standard over the needle catheter is recommended in this vein. The jugular catheter is placed using standard aseptic technique (as if the jugular vein was a cephalic vein) and the catheter can be secured using tape or suture. When securing the catheter with tape around the neck, be sure to keep the tape loose enough so the patient can breathe, but tight enough that the catheter will stay in place.

Intraosseous catheters can also be used in neonates. A 20 or 22 gauge needle can be placed in the proximal femur, proximal humerus, and the lateral or medial aspect of the proximal tibia as discussed previously. Intraosseous catheters should be removed once the patient starts becoming more mobile or alternative venous access is obtained.

Enteral route of fluid administration

Nasogastric/Nasoesophageal

Nasogastric and nasoesophageal, (NG/NE) tubes are another relatively non-invasive way of giving fluids to a patient. Water, electrolyte solutions, and liquid food formulations can be given through these tubes.

Procedure

Depending on the size of the patient's nostrils, pick a feeding tube ranging from 5 to 10 french. Use a topical local anesthetic such as 2% lidocaine or 0.5% proparacaine locally in the nostrils (typically 1-3 drops per nostril) prior to placing the tube. If using a feeding tube with stylet, flush the feeding tube to lubricate the stylet and later allow removal. Measure the tube to the 7^{th} to 9^{th} intercostal space for a NE tube and the 13^{th} intercostal space for a NG tube. Place the tube ventromedially within the nostril. The patient should swallow when the tube reaches the oropharynx to facilitate placement into the esophagus. Secure the feeding tube in place using sutures. A radiograph is the most accurate method to confirm placement.

Advantages

Arguably, giving fluids via the gastrointestinal tract (GIT) is more physiologic than providing intravenous fluids as long as the GIT is functioning. A variety of rates and amounts of fluids can be given through a feeding tube either as fluid/electrolyte solutions or as liquid diet such as CliniCare® (which is 81% water). In our experience, patients are less likely to develop peripheral edema when they receive fluids via the GIT. This may also be the preferred method of hydration in animals who cannot tolerate the high sodium load of intravenous fluids, such as those with severe heart disease.

Disadvantages/Complications

When placing the NE/NG tubes, there is a risk of placement into the trachea. Besides inducing coughing if this happens, aspiration pneumonia can happen. To avoid this, a lateral thoracic and/or abdominal radiograph should be taken in all patients to verify tube placement into the esophagus/stomach. It is also not recommended to place a NE/NG tube if the patient is uncontrollably vomiting as they may expel the tube. A e-collar should be placed on the patient after placement to avoid the patient pulling out the tube.

Urinary Catheter placement

Foley urinary catheter placement can be a necessary thing for patients that are unable to urinate on their own or are urinating so much that a urinary catheter can help keep them clean and dry. Urinary catheter placement can also be useful for patients in situations where the measurement of urine output is needed to monitor for hydration.

Procedure

Foley urinary catheter placement is a sterile procedure. You must ensure the utmost cleanliness when placing these catheters. Sedate the patient if necessary. Gather your supplies that include sterile gloves, sterile lubricant, a foley catheter pack that will include most of the other supplies you may need, a urinary collection bag, and 0.05% chlorhexidine scrub. Wash your hands thoroughly. Don non-sterile gloves and, for female patients, clip hair around the vulva or prepuce as needed. Flush the vulva or prepuce with 0.05% chlorhexidine solution. Insert 2% lidocaine jelly into the vulva. This will help with relaxation of the urethral opening. Once the patient is cleaned and prepped, don new sterile gloves. Remove the catheter from the sterile opening and follow the directions of the manufacturer on how to lubricate the stylet within the catheter. In male dogs, another person will extrude the penis. Test the foley balloon to make sure there are no holes within the system. Place sterile lubricant on the end of the foley catheter and insert it into the urethral opening. For female patients, this can be a little more difficult. The technician that is placing the catheter must lubricate a finger and palpate for the urethral papilla and then insert the catheter ventral and into the urethral opening. Urine should be flowing out of the catheter if the technician has placed it correctly. Fill the balloon with the correct amount of sterile water according to the package insert. Once the balloon is filled, gently pull the catheter until the balloon is seated in the bladder. Remove the stylet and attach the urinary drainage system. To maintain cleanliness of the urinary catheter, it is recommended to clean the urinary catheter site and line every 8 hours with 0.05% chlorhexidine solution. Drain the urine from the bag every four hours and record on the patients chart how much you have emptied.

Advantages

Urinary Catheters are easy to place and are a useful tool in caring for any patient with urinary issues. They are not very expensive, and most clinics can get them from their preferred vendor. As I have said before, urinary catheters can help with the measurement of urinary output which in turn can assess a patient's hydration status. It can also keep the patient clean and dry.

Disadvantages

Some disadvantages to an indwelling urinary catheter are possible urinary tract infections, cystitis, urethra inflammation, and possible self-removal, (because some patients just really don't want an indwelling urinary catheter).

Tracheostomy tube care

Tracheostomy tubes are placed by veterinarians, but that doesn't mean that they are the only ones to manage them! Once the first tracheostomy tube is placed, technicians can replace and maintain these tracheostomy tubes. As technicians, we must watch out for complications from placement like infections and tube displacement.

Maintenance includes humidification, cleaning the tube and the site, suctioning, inspection, and replacement. All of this, except replacement, should be done every two hours. The respiratory system automatically produces mucous and thick secretions to protect the airway which can clog the tracheostomy tube.

Humidification consists of instilling 0.5 to 3 mL of sterile saline into the tube. Humidification is essential because the air that the patient is breathing in is very dry. When a patient breathes normally through the mouth or nose, saliva and mucous in the upper airway humidify the air naturally.

Cleaning of the tracheostomy tube site should also be done every two hours. Whenever handling tracheostomy tubes, the technician should be wearing gloves. A 0.05% chlorhexidine solution should be placed on gauze and cotton swabs. Gentle clean around the tracheostomy tube but do not use the chlorhexidine in the wound as it can cause irritation. Change the tube tie as needed.

Suctioning of the tube can prevent accumulation of mucous within the tube. Patients should be pre-oxygenated for 5 minutes. A sterile suction catheter should be used. Quickly suction out the tracheostomy tube and remove the suction catheter and place the patient back on oxygen. A technician should suction out the tube a few times. Suctioning should be stopped if the patient is struggling to breathe.

If the tracheostomy tube has an inner lumen, the inner lumen should be removed and cleaned every 4 hours. This can be cleaned with a 0.05% chlorhexidine solution and rinsed with sterile water. If a single lumen is used, it is recommended to replace it with another tracheostomy tube while you are cleaning the first tube.

Tracheostomy tubes should be replaced with a sterile tube every 24 hours. This will prevent biofilm from forming on the tracheostomy tube. Biofilm can cause pneumonia is immunocompromised patients.

Chest Tube Maintenance

Chest Tube placement is done by a veterinarian, but the maintenance can be done by veterinary technicians. Chest tubes must be monitored 24 hours a day. Placement of a chest tube is indicated if there is a consistent pneumothorax, pleural effusion, or even after a patient has undergone thoracic surgery.

Once the chest tube is placed a three-way stopcock should be placed at the end of the tube. This will help facilitate suctioning of the tube in an efficient manner. It is very important that the veterinary technician that is taking care of this patient knows how a three-way stopcock works.

The chest tube should always be handled while wearing gloves. This will help prevent hospital born infections. The site of the chest tube should be evaluated and cleaned once a day. If there are any signs of infection, redness, or swelling, the chest tube should either be removed or replaced. The patient should always be wearing an e-collar to prevent them from pulling their own chest tube out.

To evacuate the chest tube, the veterinary technician will need a bowl, a syringe of any size larger than a 12cc, and alcohol swabs. To aspirate the chest tube, first, wipe down the three-way stop cock with alcohol. There should be a clamp on the chest tube. This should be unclamped if you are aspirating the tube. Next, attach the syringe to the three-way stop cock and figure out which way the stop cock needs to be. It should be “off” to the patient. Next, move the stopcock so it is “off” to the air. Aspirate the fluid or air off of the patient. Turn the stopcock off to the patient and inject the fluid into a bowl or evaluate how much air is in your syringe. Repeat this process until negative pressure is obtained. Remember to turn the stopcock off to the patient and clamp the chest tube once finished.

If you notice your patient in respiratory distress, the first thing you should do is aspirate that chest tube and evaluate all junctions. A pneumothorax can develop if there are any loose connections.

Air or Fluid should be added up daily to evaluate when to remove the tube. Since the tube is a foreign substance in the body, it will generate about 1 to 2 mls/kg/day of fluid because of the body’s inflammatory response system.

If a chest tube is constantly producing fluid or air and nobody can keep up with aspirating it, a continuous drainage system may have to be used. There are commercial drainage systems available, but there are also three bottle systems that can be made in house as long as you have suction available. Technicians can place a patient on continuous suction, and they can be helpful to keep a patient stable. Again, you should not remove continuous suction until the drainage is around 2mls/kg/day.

Closed Suction Drains

There are a few types of closed suction drainage systems for peritoneal drainage. One example of these drains includes Jackson Pratt drains. Jackson Pratt drainage systems create suction by the vacuum that is created when the reservoir is compressed, and all the air is removed. The fluid then flows from the peritoneum to the reservoir. The fluid should be emptied from the reservoir every 4 hours. Gloves should be worn every time the drain is touched, and alcohol swabs should be used on all ports before opening or closing the drain. The drain site should be cleaned every day with dilute chlorhexidine scrub and rebandaged. The patient should be

wearing an e-collar to prevent them from pulling out the drainage tube. Once the tube is producing little or no fluid, it can then be removed. Some complications of these drainage tubes can be infection at the site or in the peritoneal cavity, kinking of the tube itself, and clogging of the tube.

Conclusion

In conclusion, technicians can do a lot of advanced procedures that veterinarians do not have to be a part of. It is important to know the advantages, disadvantages, and maintenance of all of these advanced procedures and how we can prevent complications.

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Technician to Manager – A Hard Transition

Tami Lind, BS, RVT, VTS(ECC)
Purdue University, West Lafayette Indiana

Most technicians that have been in the field for a few years think about becoming a manager or try to step up their leadership game. The problem that most technicians face when they decide to become manager, is that they do not have the training or education to become the leaders that they would like to be. Technicians generally move up the ranks because they have been there for many years, know the "ins and outs" of the practice, or have amazing technical skills. This does not mean that they will make great managers or supervisors.

Great technician managers are ones that work on the floor as well as work hard behind the scenes on the management side. To be a great manager, you have to change your thinking. You have to think like a manager and not like an employee. Communication is key. You will have to look at your employees and watch how they communicate with everyone else around them, but you also have to look at yourself and see how you communicate with everyone around you. I suggest that your team do a personality assessment like MBTI, DISC, or Enneagram. Take that information and tailor your communication to each individual person. You cannot fix every communication problem between employees, but you can work to improve yours.

Arguments between coworkers happen everywhere, but don't get sucked into these arguments. It is not the manager's job to fix communication issues between employees. Encourage them to go and communicate with that person that they are having an argument with. We are adults and should not be hiding behind a computer or cell phone screen to communicate with another coworker. It is difficult and scary to have these conversations, but it needs to be done to fix the issues. As a manager, always document what is going on and follow up with both employees. If they do not get communication issues fixed between themselves, then it may be time for you to step in and do some mediation.

Social media, Facebook especially, can become very toxic for a manager. Managers should not get into arguments or work discussions with employees over Facebook. Social media has become a place for employees to vent to the world, and it is not a manager's position to try and fix the problem via Facebook. I would encourage every manager to not "friend" your employees on social media. It will just create more stress for the manager wondering what all the employees are saying on social media.

Corrective communication needs to happen immediately. Going to an employee a week later with a problem will not create a great work culture. Most everyone appreciates being told when they are doing something wrong right at the time that they are doing it. Also have regular meetings with them so that they don't assume that every time you meet that they are in trouble. Always document the conversations and follow up if necessary.

Managers must also learn to think globally. As an employee, one is used to thinking of their own team. Managers must be able to look at the bigger picture and see how decisions will

affect everyone. Advocate for your team, make sure they are taken care of, but also be able to see and explain how and why decisions are made. Transparency is important. Make sure your team understands how decisions are made and information is disseminated. If you are not meeting with other managers and your hospital owner or medical director, work to make that a possibility. The managers need to be viewed as their own team, communicating about their concerns, supporting decisions, and knowing what is happening with each group so they can then communicate decisions in a timely manner. Being surrounded by those in a similar position to you will help you start that global thinking and begin to see how seemingly small decisions of policy and paperwork affect everyone.

An example of thinking globally is the schedule. Scheduling is one of the hardest things for a manager. You have to make it fair to everyone and not give someone special priority, but also, you have to make a schedule that works for the hospital itself. You can allow the staff to create rotations, swaps, and have their input in the schedule as a whole. Always explain your process and give the staff explanations of the schedule and why you did it the way you did. Do not be afraid to get creative, but always remember that if that one person leaves, you're left with an empty position. Make sure that you always check with your state labor laws.

New Managers also need to make the switch to view the world as "management" would. If someone is consistently calling out sick or coming to work late, it may not seem like that big of a deal to you. However, it may be a big deal to the team and the manager has to be fair to the whole team. This thinking is hard, especially if you are a young technician that has just become a manager. This way of thinking does not come immediately, but it needs to happen to become a great manager.

Expectations are HUGE. Make sure your team knows what you expect of them. Also, make sure that they let you know what they expect of you. If they expect their manager will cover 100% of the open shifts, everyone is in for disappointment! Meeting with your team either 1:1 or as a group will give you an opportunity to lay out your expectations and ask your team how you are doing. Remember that a manager works to serve the needs of the hospital and employees, and you can only do that by communicating frequently with your team. Have a clear vision for the whole team and then everyone can be on the same page.

New managers also need to learn how to become a true leader. A lot of people can "manage" the everyday tasks of the clinic, but leaders need to also inspire and mentor their team. Most every technician in this field wants to succeed. Leaders want their people to do better. It is not a competition! Leaders do not condone bullying! In turn, if you mentor and lead your team to their goals, you will also succeed in the process. Managers should also be on the floor so that you can properly advocate for your team and you understand what they are going through. Do not ask someone to do a task that you are not willing to do yourself!

Leaders do not have to be managers either, a manager should identify those leaders on the team. Work hard to keep those employees on your team. They are the ones who are bringing people together and mentoring the new technician on the floor. If you want to become a manager in the future, being a leader is the first step to becoming a great manager.

Work friendships are hard when you become a manager. It is difficult to be friends with the employees that you manage. Managers may be accused of showing favoritism with the employees that they may "hang out with" after work. Managers, unfortunately or fortunately, need to represent the company that they work for at all times. Socializing with coworkers outside of work can become difficult, especially if everyone wants to vent about work during dinner. Managers need to be proactive about communicating issues and work to keep professional and personal relationships separate. Managers need to strive to be respected. Being "liked" is a totally different. Employees may not like every decision that you make but they need to respect your decision. Make sure you communicate often. Again, transparency is key.

Change can be difficult for any practice to implement, and some staff members may make change even more difficult. Encourage all staff to participate in changes and be sure to communicate with the team often. Even in doing all of this a practice still may encounter barriers to change. If there are some staff members actively recruiting other coworkers against the efforts to change, those people may need one on one time with a manager to work through any issues. If possible, figure out the issue with the change and act to fix that issue so everyone can move forward as a team. Change can be intimidating but each team member deserves the opportunity to voice their complaints. Implementing protocols can be a very daunting task and may feel overwhelming. I would suggest to start big and then focus down to specific procedures. Have the staff help you in researching protocols. Creating buy in will help with the staff's ownership of the practice and procedure and create a relationship of respect between you and the employees.

Managers also need to make sure that they admit their mistakes. It is ok to make a mistake! If something is not working with your management style, change it. Accept constructive criticism and make sure to get feedback often. Managers also need to make sure that they have clear decision making skills. Making a decision about what to eat for dinner is totally different than making the decision about the schedule when 2 people call out sick.

I always suggest new managers to seek out support from other managers in the field. See what works for other managers, network, and have someone to vent too. Managing is always changing. With new generations of workers, new medical protocols, and an ever-changing staff, it can be difficult to keep up.

Management is tough, but not too tough for amazing veterinary technicians who want to better their practice! Remember to always work on the hospital floor, advocate, and support your team. Always be transparent and communication changes effectively. It takes time. Don't expect to become an amazing manager overnight. Accept change and always provide top notch care to your staff, clients, and patients.

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