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INTERNAL MEDICINE

Lower Urinary Tract Diseases of the Senior Dog

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The senior dog with lower urinary tract (LUT) signs presents a unique challenge. As a dog's age increases – along with the number of comorbidities – the clinical approach must be adjusted appropriately. This article will focus on how the aging of the LUT impacts function and predisposes to disease. Additionally, it will discuss some of the more common LUT diseases seen in older dogs, and how these diseases can interact to make diagnosis and treatment challenging.

AGING OF THE URINARY TRACT

The lower urinary tract consists of three functional components that must work in coordination for appropriate micturition:

1. The detrusor muscle, consisting of smooth muscle and under parasympathetic control
2. The smooth muscle sphincter, under sympathetic control
3. The urethralis muscle, consisting of striated muscle and under somatic control

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When, Why, and How – Making the Most of the Diet Trial as a Diagnostic Test

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The classifications of adverse reactions to food can be either immunologic or non-immunologic. Food allergy is defined as any immune-mediated reaction occurring after food intake. This can be either IgE-mediated (immediate) or non-IgE mediated (delayed). Examples of non-immunologic adverse food reactions include poisoning, metabolic reactions, or anaphylactoid responses. For our purposes, we will review immunologic reactions to

food in the dog and cat and focus on making an accurate diagnosis of this condition.

PATHOGENESIS

The digestive tract is responsible for differentiating between nutrients and harmful substances each and every time a food item is consumed. The ability of the body to maintain

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In order to maintain its function, the LUT relies on a complex system for preventing infection, consisting of a combination of physical, anatomical, and cellular components. The act of voiding, presence of normal urogenital flora, high-pressure zone in the urethra, and urine composition all help to prevent bacteria from ascending and infecting the LUT. The urothelium functions as a barrier to infection but also uses pattern recognition receptors to detect bacteria and activate and recruit neutrophils and mast cells. With age, these components of normal micturition and barriers to infection can break down and increase the likelihood of lower urinary tract infection.

INCONTINENCE

While age is not a cause of incontinence, changes to the function of the urinary tract and comorbidities, especially those causing polyuria, can increase the chances of incontinence in older dogs. In senior dogs, the differentials for incontinence are shifted and it can be helpful to think of the causes of incontinence in these dogs as urethral sphincter mechanism incompetence (USMI) vs. bladder storage dysfunction. While USMI most commonly occurs in female dogs within 3 years of spaying, it is a complex and incompletely understood disease process that seems to involve hormones, changes to collagen, and patient conformation. It is not unusual for USMI to be revealed in senior dogs, either due to underlying disease or progression of previously mild signs. Bladder storage dysfunction, or detrusor instability, can be either an idiopathic condition or can be “urge incontinence,” which implies there is an underlying disease leading to the feeling of urgency.

A thorough physical exam and history will help to determine whether a dog is experiencing USMI or bladder storage dysfunction. The patient workup should include appropriate lab testing to elucidate any comorbidities that are contributing to the incontinence. These cases of incontinence should always have a urine culture and sensitivity performed, as bacterial infection can contribute to incontinence and can be found more commonly in incontinent dogs.

Treatment of incontinent senior dogs should include treating contributing comorbidities and treating the incontinence. Dogs with USMI may benefit from phenylpropanolamine or estrogen compounds, while those with detrusor instability should be treated with drugs that provide smooth muscle relaxation of the detrusor muscle, such as oxybutynin. Refractory cases of USMI may require further intervention, such as hydraulic urethral occluding devices or urethral bulking agents.

URINARY TRACT INFECTIONS

Bacterial urinary tract infections (UTI) are a common finding in senior dogs. Finding bacteria in the urine of a senior dog can be due to subclinical bacteriuria, uncomplicated UTI, or complicated UTI.

Subclinical bacteriuria (SCB) in humans is defined as the presence of bacteria, with or without pyuria, and lacking clinical signs associated with a UTI. In veterinary medicine, the recommendation is not to treat SCB, but the definition is not as distinct. Given the potential for owners to miss clinical signs of a UTI and the concern that some patients may not have the appropriate neurologic or orthopedic function to display signs, clinical judgment must be made on a case-by-case basis. Some recommend the presence of pyuria as reason to treat a UTI in veterinary medicine for these reasons. While there are currently no perfect guidelines, it is important to note that there is no evidence that dogs with untreated bacteriuria and pyuria are likely to have worse outcomes even if they have comorbidities present.

Uncomplicated UTIs are those that occur in an otherwise healthy patient with <3 occurrences per year, no recent antibiotic use, and no underlying abnormalities of the lower urinary tract. These should be cultured and treated with amoxicillin or trimethoprim sulfa for 7 days.

Complicated UTIs are those that occur in a patient with an anatomic or functional problem with the lower urinary tract or a comorbidity that increases the risk of a UTI. A cystocentesis urine sample should be obtained, and treatment should consist of 4 weeks of an appropriate antibiotic based on a urine culture and sensitivity panel. The urine should ideally be recultured one week after starting the antibiotic and one week after finishing.

Recurrent UTIs are three or more UTIs in a 12-month period and can be either due to relapse or reinfection. Relapse is infection with the same organism, as determined on a culture and sensitivity panel, within 6 months. Reinfection is recurrence of a UTI of a different organism. In senior dogs with recurrent UTIs, any predisposing factors should be identified and treated. Therapies to prevent recurrent UTIs are not proven, but include prophylactic antibiotics, cranberry extract, mannose, and probiotics.

LOWER URINARY NEOPLASIA

Tumors of the bladder and urethra account for 0.5-1.0% of canine neoplasia. The majority of these tumors are transitional cell carcinoma (TCC). The median age at diagnosis for TCC is 12 years. The bladder tumor antigen test, and more recently

BRAF testing, can be helpful screening tools for TCC, but definitive diagnosis through a combination of imaging, cytology, and cystoscopy is recommended. Staging of TCC can provide valuable information on expected prognosis. The second most commonly diagnosed LUT tumor in senior dogs is prostatic carcinoma. The median age at diagnosis is 10 years, and it occurs more commonly in neutered males compared to intact males. Prostatic carcinoma can often be palpated on rectal exam. Diagnosis should be through a combination of radiographs, ultrasound, and traumatic catheterization for cytology. Polypoid cystitis is a proliferative inflammatory disease, typically associated with chronic UTIs. While rare, polypoid cystitis should remain a differential in cases of suspected neoplasia, as it can be mistaken for TCC.

A COX-2 inhibitor NSAID is the mainstay of treatment for both TCC and prostatic carcinoma, and has been shown to significantly increase survival times for both tumors, even when used as the only therapy. Consultation with a veterinary oncologist is recommended after diagnosis, as there are multiple chemotherapy protocols available for both tumors, and stereotactic radiation therapy has shown promise with prostatic carcinoma. In advanced cases, palliative therapy with tumor laser ablation or urethral stenting may offer relief of clinical signs and improved survival times.

CONCLUSION

Lower urinary tract disease in senior dogs is common, but because of the presence of comorbidities, diagnosis and treatment can be a challenge. The approach to these cases requires not only diagnosis and treatment of the primary cause of the LUT signs, but also attention to the underlying diseases that may have predisposed the patient.

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homeostasis in the face of exposure is called tolerance. In order for tolerance to be achieved, the gut must maintain an intact mucosal barrier, regulate the immune response, eliminate antigens appropriately, and generate a system of tolerance for antigens. The Gut Associated Lymphoid Tissue (GALT) is composed of four distinct compartments of lymphocytes. Each provide the surveillance and response capacity necessary to operate a fully functional gastrointestinal barrier between what we consume and what we absorb.

The rate of intact protein absorption through the mucosal barrier of the gastrointestinal tract (GIT) will depend on many factors. First, enterocytes must have normal morphology and functionality. If the cells of the barrier are not normal or not functioning appropriately, the barrier is defunct. Second, IgA must be present in order to appropriately bind and remove antigens. IgA serves as a marker for antigens and helps aid in their homeostatic elimination. Third, digestion needs to be complete to aid in the processing of food into smaller, less antigenic molecules. Complications after surgery or comorbidities that impact digestion could play a role in the barrier function of the GIT. Similarly, food composition plays a role. If food is easily digestible, there will be less risk of intact, large protein absorption. And, finally, the presence of inflammation will impact the stability of the barrier. Inflammation begets inflammation.

If an antigen is something considered as foreign to the body that induces an immune response, what exactly is a “food antigen”? Technically, all food is potentially antigenic since it is foreign and has potential to initiate an immune response. Food is made up of complex matrices of natural constituents such as proteins, fats, and carbohydrates. As the body digests food, by-products of the original food structure are created. Fats are metabolized into fatty acids. Carbohydrates are broken down into sugar molecules. Proteins are digested into amino acids and small peptides.

Most antigens are proteins. They are composed of amino acid chains that have numerous unique structures called epitopes. These regions are the exact recognition sites that a specific immunoglobulin molecule would bind to. The larger the protein, the more epitopes. The less digestible the protein, the larger it will be. The more epitopes and the less digestible, the more antigenic.

When you combine poor barrier function, altered self-regulation, reduced elimination of antigens, and a lack of tolerance, you are left with the potential for an allergic cascade. Notice that this classification could be considered for any barrier – skin or gastrointestinal.

The allergic cascade is perpetuated by a shift from a tolerant phenotype to an intolerant one. As tolerance is lost, the predominant immunoglobulin shifts from IgA to IgE. Type 1 hypersensitivity reactions are driven by antigen binding, which leads to IgE cross-linking and histamine release from mast cells. The result: inflammation. This translates clinically into vomiting, diarrhea, and other clinical signs. While we do not believe that all food allergy in dogs and cats is mediated by Type 1 hypersensitivity reactions, we do believe that IgE sensitization plays an important role. Type I, Type III, and Type IV hypersensitivity reactions are likely part of the immunologic basis of food allergy in the dog.

Inflammation driven by IgE and histamine release further weakens the mucosal barrier's integrity. This leads to more intact protein molecules being absorbed and additional sensitization of new proteins. Furthermore, if the gut is broken down, and antigens travel through the bloodstream, mast cells bearing IgE in the skin or sensitized basophils in the blood can be activated. This can lead to cutaneous clinical symptoms and is the presumed pathomechanism of cutaneous adverse food reaction in the dog.

FOOD ALLERGY STATS – DOGS AND CATS

There is only one test to diagnose adverse food reaction in the dog (or cat), and that is an elimination diet trial with a prescription or home-cooked diet that is made from a novel protein and carbohydrate. Serum tests for food-specific IgE and IgG, intradermal testing with food antigens, lymphocyte proliferation tests, fecal food-specific IgE, patch, gastroscopic, and colonoscopic testing have been evaluated. In general, these tests have low repeatability and accuracy. The only test recommended for the diagnosis of food allergy in the dog is the elimination diet trial.

The ideal composition of a diet used to diagnose food allergy in the dog or cat has many components. Simply, the diet should be one that the patient was not previously exposed to, has a limited number of new, highly digestible proteins OR is a hydrolyzed protein, contains a lower protein content and additives, and is nutritionally adequate for the life stage of the patient. Choices include home-cooked diets that contain a novel protein and carbohydrate, commercial novel protein diets, and commercial hydrolyzed protein diets.

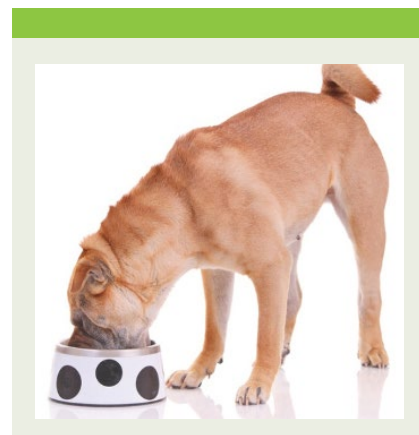
At this time, there are concerns regarding the use of home-cooked diets and commercially prepared novel protein diets for the purposes of diagnosing food allergy in the dog or cat. Home-cooked diets are poorly balanced and can be quite costly to

prepare. While a commercial novel protein diet is balanced, they are not currently recommended for use for diagnosing food allergy due to their variable efficacy and concerns with cross-reactivity.

Cross-reactivity occurs when proteins in one substance are like proteins in another. The immune response that is generated is the same or similar when exposure of one or the other occurs. This can happen between foods, between foods and environmental triggers, or between environmental triggers. An example includes cross-reactivity between chicken and crocodile protein leading to anaphylaxis in a chicken-allergic child. We have limited understanding of this in our veterinary patients. However, enough is known in human medicine to generate pause when using these diets for diagnostic purposes.

Commercial hydrolyzed protein diets subvert concerns of cross-reactivity via hydrolysis. This process reduces whole proteins to smaller peptides and amino acids. Molecular weight is reduced, and epitopes are disturbed. As a result, IgE cross-linking is prevented and allergic response is much less likely. These diets are also nutritionally complete for various life stages. My current recommendation for a diagnostic diet for food allergy is an extensively hydrolyzed, commercially prepared food that is nutritionally balanced for the patient's life stage.

The diet trial should last 8 weeks. This has been shown to be an adequate amount of time to identify over 90% of food-allergic patients. Interestingly, gastrointestinal signs tend to clear up within 2-3 weeks of starting an exclusively fed commercial hydrolyzed protein diet. This is often a first marker for me to determine if the diet will be useful from a diagnostic perspective. If GI signs have not resolved within the first 2 weeks, consider a different diet for your diet trial.



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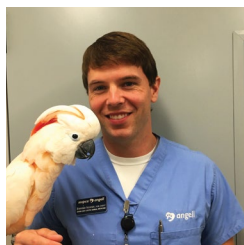
Interpreting the diet trial involves demonstrating a reduction in pruritus and a resolution in other clinical signs. This can be complicated if there are treatments on board that could mask symptoms, infection present that has not been adequately treated, or partial responses due to the ebb and flow of environmental allergy. In the end, it is important to remember that a diet is a benign, conservative strategy that can drastically improve the health and life quality of an allergic patient. Continuing the diet past the 8-week period to sort these potential complicating factors out is usually well worth it. However, this should always be done deliberately to avoid having your patient on a diet that is medically unnecessary.

Confirming your diagnosis of food allergy involves an oral food challenge. There are many ways to perform dietary challenges. My recommendation is to tailor all “life after the diet trial” steps to your individual patient and client. Nevertheless, it is important to know that it can take up to 14 days for a dog to show clinical flare after an oral food challenge; cats can take up to 7 days. Therefore, all challenges should be spaced out accordingly.

Communication about the diet trial should be direct and informative. In order to improve adherence to diet trial recommendations, veterinarians should improve knowledge, reduce perception of barriers, and heighten self-efficacy (owner confidence). This is one of the few diagnostic tests that is left in the hands of a pet owner. We need to improve our communication about this test in order to improve our diagnostic outcomes. Improving health outcomes through communication is a goal that we all strive for, and that is especially the case when it comes to the diet trial and food allergy.

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Dental Anatomy and Disease of the Domestic Rabbit (*Oryctolagus Cuniculus*)

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Domestication of *O. cuniculus* took place between the 5th and 10th centuries, and rabbits have since distributed to every continent save Antarctica. The presence of sharp rostral incisors resulted in their classification alongside rodents. However, their incisor anatomy is also what sets them apart, given that a second pair of tiny upper incisors is present behind the major set. Despite strong similarities between rodents and rabbits, it was determined in the 20th century that the dentition represented an example of divergent evolution that separated rabbits into a separate mammalian order, Lagomorpha. A species that was once raised for meat and fur has since transformed into more than 60 recognized fancy breeds ranging in size, shape and coat. They have since found their way into our homes as pets, but regardless of size or breed, all pet rabbits are susceptible to dental disease. Whether acquired or congenital, their unique anatomy predisposes them to dental disease unlike that seen in dogs and cats

Rabbit teeth are classified as elodont (for their continuous growth with no anatomic root) and hypsodont (for having a long crown). The dental formula of the rabbit is $2(I \frac{2}{1}, C \frac{0}{0}, PM \frac{3}{2}, M \frac{3}{3}) = 28$. The lack of canine teeth creates an elongated diastema between the incisors and premolars. Rabbit mouths exhibit anisognathism, which means that their lower jaw is narrow when compared to the upper. Rabbits also have a maximum gape of about 20-25 degrees, which makes evaluation of the teeth and dental procedures more difficult. The portion of the tooth that can be visualized above the gum line is called the clinical crown, while the portion that remains below the surface is the reserve crown. Since the teeth are continuously growing, they also do not have a true root, but instead have an apex from which new growth emerges.

The upper incisors consist of hard enamel on the rostral surface and softer dentin along the lingual surface. The differing densities of these surfaces creates a sharp edge as it wears. The lower incisors are covered in enamel on the labial and lingual surfaces, which better suits their normal occlusal placement between the major maxillary incisors and the smaller

secondary set of upper incisors, better known as the peg teeth. At rest, the opposing incisors and cheek teeth (premolars and molars) remain in contact. When eating, rabbits will use the incisors to cut the food into manageable lengths using a vertical, chopping motion. Once the food is passed to the cheek teeth, a rotating horizontal stroke is used to grind the food prior to ingestion. This horizontal motion is a large contributor to proper dental wear, which is only achieved when coarse roughage such as hay is consumed. During this process, every lower cheek tooth occludes with two upper cheek teeth with the exception of the first mandibular premolar (PM 3) and the last mandibular molar (M 3). This dual occlusion allows for normal wear even in the absence of one opposing cheek tooth.

Any process that impedes the normal eruption and wear of elodont teeth has the potential for causing dental disease, which is divided into four main classes: congenital, traumatic, metabolic bone disease, and abnormal wear. Rabbits born with prognathism or brachygnathism are likely to develop overgrown incisors and potentially abnormal wear of their cheek teeth due to an abnormal pairing of the upper and lower jaw. This is commonly seen in dwarf and lop rabbits. Any traumatic event that alters the jaw or fractures teeth into the alveolar cavity can offset wear or result in infection. Metabolic bone disease is relatively rare, but has been confirmed in several studies where parathyroid hormone was elevated and calcium levels were low. Demineralization of the skull and loosening of the teeth within the alveolar bone causes changes in the occlusal surface, while periosteal deformation predisposes to infection.

The most important class of dental disease is acquired through improper wear. Wild rabbits select foods high in fiber and silicates, which require exaggerated horizontal movement and appropriate wear of the clinical crown. In captivity, a diet high in grass hay and fresh greens is the best substitute for selections of their wild counterpart but cannot exclude the potential for acquired disease. However,



rabbits that lack fiber in their diet are far more likely to end up developing dental disease over time. Without fiber and normal masticatory movements, the elodont teeth continue to grow and create pressure on the apex. The apex will be pushed further into the socket, elongating the reserve crown and bending the apex, which will change the direction of growth in the clinical crown. Over time, lingual points will develop along the mandibular cheek teeth and buccal points will be created on the maxillary cheek teeth as the normal chewing motions cannot wear the exaggerated points. The horizontal chewing motion required to grind hay will aggravate lesions in the mouth and rabbits will begin to prefer pellets that can be crushed using vertical masticatory motions, further exacerbating the problem.

As dental disease begins to affect the patient, owners may notice weight loss, dysphagia, drooling, anorexia, change in fecal size/quantity, excessive salivation, or change in food preferences. While onset of gastrointestinal stasis can have many etiologies, deciding if dental disease is playing a role can typically be determined during the physical exam. Inspection of the incisors can be achieved by gently pulling the upper lips caudally from both sides, with special care

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taken not to occlude the nostrils by pulling the lips dorsally. This will allow you to inspect the occlusion of the incisors from the front and sides, identifying if the lower incisors fit in the notch between the two sets of upper incisors. The limited range of motion of the jaw and narrow gape of the patient makes cheek teeth more difficult to examine. The diastema provides a gap for insertion of appropriate instruments to visualize the cheek teeth. A plastic otoscope cone is a safe tool to use, as iatrogenic tooth damage is unlikely if the patient were to chew it. Metal nasal speculums have the benefit of visualizing the lingual and buccal aspects of the teeth simultaneously but should be used by a more experienced handler to avoid a fracture. Subtle abnormalities are likely to be overlooked in a conscious exam, but subtle cues like excessive saliva or bubbles can give confidence that a sedated exam should be recommended.

Evaluation of whether the patient can undergo a sedated exam is an important first step. Some animals may benefit from supportive care to ensure they are a better anesthetic candidate. Certain cases also benefit from advanced imaging prior to a sedated dental procedure to help plan the approach. Rabbits with jaw abscesses, exophthalmia, dacryocystitis, or persistent purulent nasal discharge could all have an elongated reserve crown that has dragged bacteria from the mouth into the soft tissues, which sets up infection. Four-view skull radiographs are one way to try and elucidate which teeth are contributing to the presenting clinical signs. Computed tomography is the preferred diagnostic modality given the significantly improved information you can gain, which helps with treatment planning.

Incisor malocclusion can be treated by either routine trimming of the affected teeth or by extraction. Deciding which plan to offer is based on signalment, history, and diagnostic workup. Young rabbits with congenital incisor malocclusion are better candidates for extraction, as the frequency and cost of trimming can become overwhelming every 4-6 weeks for the next 7-10 years. Extraction is the only option for rabbits presenting with tooth root infection. Trimming can be performed awake in calm rabbits who allow for proper manual restraint. Sedation or gas anesthesia will be necessary for some rabbits to prevent iatrogenic injury to the surrounding soft tissues during a trim. A Dremel tool with a diamond wheel is the preferred instrument to reduce the likelihood of longitudinal fractures.

A tongue depressor is used on the mesial surface to protect the soft tissues once the disc has transected the incisors. Incisor trimming with nail trimmers, wire cutters, or rongeurs should all be done with caution. Crushing the teeth with these tools can lead to longitudinal fractures, which will cause introduction of bacteria to the reserve crown and surrounding soft tissues. Incisor extraction is always performed under anesthesia. The use of a Crossley Incisor Luxator as well as flattened and curved large-gauge hypodermic needles are required. Ensuring that all the dental pulp is removed is important to reduce the incidence of the incisors growing back in. Using properly curved needles to curettage the alveoli after extraction can reduce the incidence of regrowth.

Molar malocclusion is especially common in patients with reduced hay intake. Molar points can be identified on routine exam in a healthy animal or during examination of an animal with a reduced appetite. Rarely are molar points able to be effectively trimmed in the awake patient. Inhalant anesthesia is required to allow for a patient to be properly placed on a dental board, where full examination of the cheek teeth can be performed. A low-speed drill is the ideal tool to quickly and effectively reduce molar spurs or malocclusion. Hand tools, such as rongeurs and dental rasps, can be useful in conjunction with a low-speed drill but would prolong a dental procedure if used alone. Finding loose or diseased teeth may indicate the need for extraction. Intraoral extraction can prove difficult due to the limited gape of the rabbit mouth (20-25 degrees) and the elongated

diastema, which places the cheek teeth farther back in the mouth. If indicated, a Crossley Molar Extractor and extraction forceps will be necessary. If only one molar is removed, then removal of the opposing tooth is not necessary. Each cheek tooth engages with two opposing teeth and will allow for normal wear.

Dental abscesses are a common sequelae of chronic dental disease. If the teeth are not wearing properly, the pressure from the elongated teeth opposing one another will push the reserve crown toward the apex. This will allow for oral bacteria to be pulled beneath the gum line and create a periapical infection. Dental abscesses can be found along the mandible, inter-mandibular space, cheeks, maxilla, or retrobulbar space. A CT scan is always recommended to determine which teeth are involved so they can be removed during debridement. Without removing these teeth, recurrent abscesses are more likely. Culture and sensitivity of the abscess capsule should be acquired during surgical excision and debridement of the abscess. Marsupialization of the site and flushing daily with dilute povidone iodine allows for healing by second intention. Wound packing with antibiotic-soaked umbilical tape or antibiotic-impregnated beads has also been described.

Understanding the anatomy and physiology of rabbit dentition can help impress upon clients the important role a diet high in fiber can have. Many of the common dental problems we encounter can be avoided through proper diet alone. Prevention is key since management of improper dental wear requires frequent follow-up for the life of the patient.

FIGURE 1

Figure 1 Examining incisors: smooth whiskers back, ensure nares open, part lips, and examine from front and side.



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Patent Ductus Arteriosus and Other Causes of Continuous Murmurs

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Patent ductus arteriosus (PDA) is the most common congenital heart defect in dogs. Retrospective studies suggest that PDAs account for ~ 11-32% of congenital heart disease in dogs; PDAs are also fairly common in cats, accounting for ~ 11% of all feline congenital heart diseases. This disease can affect dogs of all sizes, but some breeds are more predisposed, especially Maltese, toy poodle, miniature poodle, and German shepherd dogs. A genetic predisposition has been proven in the poodle and is suspected in other breeds. There is a female predisposition, as well.

The ductus arteriosus originates from the 6th aortic arch and connects the pulmonary artery to the aorta. During fetal development the pulmonary vasculature is constricted, and the ductus allows oxygenated blood to shunt from the pulmonary artery (arrow) aorta (arrow) body. At birth, lung expansion occurs and oxygen tension in the systemic vasculature increases. This leads to pulmonary vessel dilation and significantly decreased pulmonary vascular resistance. Within minutes to hours of birth, there should be contraction and physiologic closure of the ductus. A PDA results due to failure of closure of the ductus arteriosus after birth.

As a result of having a PDA, blood shunts continuously from “left to right” (aorta to pulmonary artery) due to higher aortic blood flow compared to pulmonary blood flow, resulting in continuous flow and a continuous murmur. The amount of left-to-right shunting depends on the size of the PDA and amount of blood allowed across the defect. The continuous left-to-right shunting causes the following cycle of blood flow through the heart and lungs:

Aorta → PDA → Main pulmonary artery → Lungs → Left atrium → Left ventricle → Aorta → PDA → etc.

Ultimately, if the PDA remains patent, this will lead to increased pulmonary flow → increased venous return to the left atrium (LA) and left ventricle (LV) → left-sided volume overload → LA dilation, LV dilation → left-sided congestive heart failure.

FIGURE 1

Amplatzer Canine Duct Occluder (ACDO): A self-expanding nitinol mesh device, composed of a short waist that separates a flat distal disc from a cupped proximal disc. The device is designed specifically to conform to the shape of the canine ductus.



Unfortunately, if the left-to-right shunting PDA is not closed, this can lead to congestive heart failure by the age of 2 years old in most dogs. Additionally, there is the possibility of the PDA flow reversing; right-to-left shunting can occur due to increased pulmonary vasculature resistance, increased pulmonary blood flow, and pulmonary hypertension. Treatment of right-to-left shunting PDAs is very different compared to left-to-right shunting PDA and will not be addressed in this article.

CLINICAL ASSESSMENT

Diagnosis of a PDA can be strongly suspected based on signalment, history, and physical exam findings. Patients with PDAs are often asymptomatic; patients with severe volume overload may show signs related to CHF (dyspnea/tachypnea, coughing, exercise intolerance, syncope). The murmur of a PDA is classically heard as a continuous “washing machine” murmur at the dorsal heart base. It is often heard cranially, and we recommend auscultating further up into the armpit in all dogs, especially puppies!

Definitive diagnosis of a PDA is made via echocardiography, though thoracic radiographs are also helpful in the initial workup. Thoracic radiographs may reveal left heart enlargement, pulmonary overcirculation, and often a “triple bump” appearance (due to pulmonary artery and aorta dilation plus a ductal aneurysm); finally, there may be evidence of congestive heart failure. Echocardiography of PDA will allow determination of heart enlargement and pumping function and visualization of the PDA morphology and size.

CARDIOLOGY

Patent Ductus Arteriosus and Other Causes of Continuous Murmurs

CONTINUED FROM PAGE 8

TREATMENT OPTIONS

Left-to-right shunting PDAs are one of the few cardiac diseases in dogs and cats in which treatment can essentially be considered curative. Surgical treatment requires a lateral thoracotomy to directly visualize, isolate, and ligate the PDA. This is commonly performed in very small dogs (e.g., dogs < 3 kg), cats, or in some larger breed dogs (especially German shepherd dogs!) with specific anatomical features of their PDA that are only amenable to surgical ligation. A less invasive option commonly performed at Angell is PDA occlusion using an Amplatz Canine Duct Occluder (ACDO). This minimally invasive procedure involves placing a catheter within the femoral artery, performing an angiogram (contrast study) of the PDA, then placing an ACDO within the PDA. This has a high success rate and overall low complication rate. Many dogs' hearts can return to normal size, sometimes as soon as the next day!

OTHER CAUSES OF CONTINUOUS MURMURS

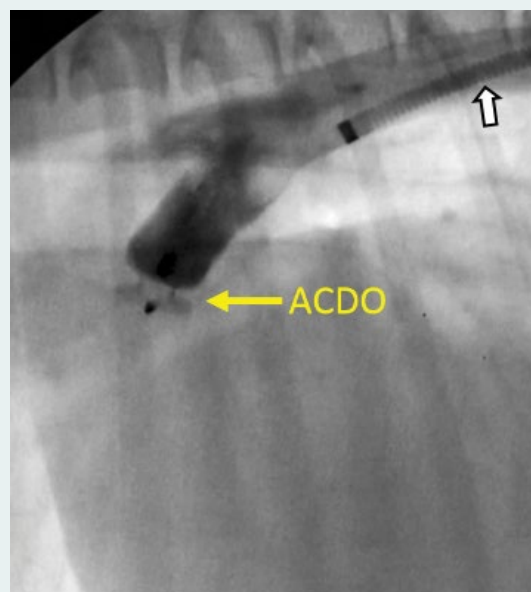
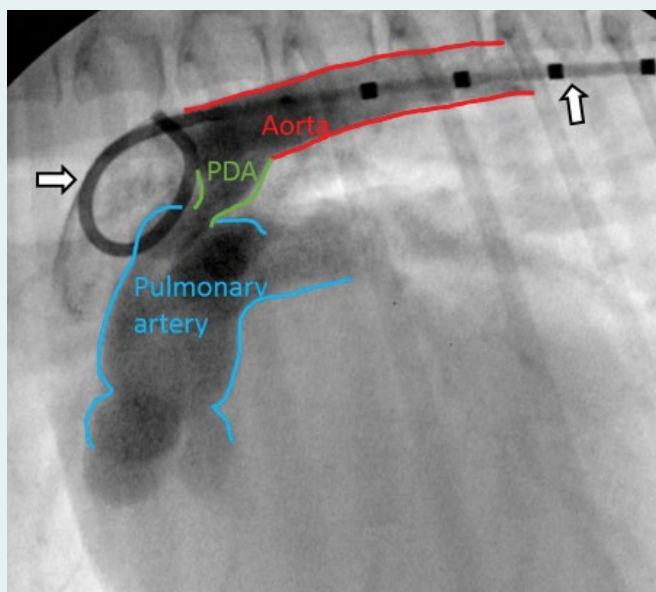
Though far less common, there are a few other conditions to be aware of that can also cause a continuous heart murmur. These conditions are typically a bit more challenging to diagnose and treat. Some examples include aorticopulmonary window (a "window" between the major vessels, as opposed to a ductus or vessel, which can be due to a congenital abnormality or trauma); truncus arteriosus (in which the aortic and pulmonary trunks fail to divide – quite rare), ruptured sinus of Valsalva (a.k.a. ruptured aortic sinus), and fistulae (e.g., systemic or pulmonary arteriovenous fistulae/shunts or coronary artery fistula). Either way, if a continuous murmur is auscultated, we recommend referral to a local cardiologist for further evaluation!

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FIGURES 2

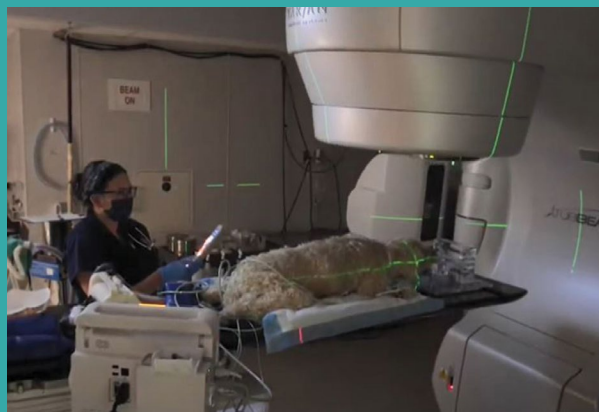
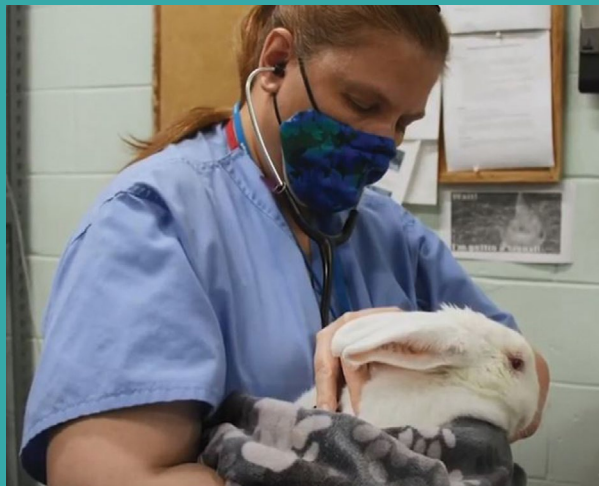
↘ "Before" and "after" angiograms of a patient with successful PDA occlusion via ACDO. The image on the left shows blood flow from aorta → PDA → pulmonary artery. The image on the right reveals successful occlusion of the PDA (lack of flow from the aorta into the pulmonary artery)! (White arrows with black outlines identify the catheters in each image; the catheter in the "before"/left image is a marker pigtail catheter. The catheter in the "after"/right image is a Check-Flo catheter.)



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- Massage
- Consultation and fitting of assistive devices
- Chiropractic



Land-based Exercise Area



Hydrotherapy – AquaPaws Water Treadmills



Hydrotherapy – Indoor Pool



The Dangers of Rodenticides to Pets

Amanda Lohin, DVM

angell.org/emergency | 781-902-8400 | MSPCA-Angell West

Toxicities are an all too common presentation for the many species we see through the emergency service. One of the more common toxicities comes from ingestion of rat bait (a.k.a. rodenticides). Rodenticides come in many different forms, with different mechanisms of action to ultimately lead to death. Some rodenticide toxicities are very treatable, while others are not. The most treatable rodenticides are the anticoagulant rodenticides, while others, including Bromethalin (neurogenic), Cholecalciferol (leading to severely elevated calcium levels) and zinc phosphide (causing poison gas production) tend to be less treatable, although all toxicities are dose dependent. Below is a quick overview of each type of rodenticide, their mechanism of action, and treatment options. If ever you suspect your pet has ingested any type of rodenticide, it is highly recommended to have them evaluated by a veterinarian and to consult an animal poison control center such as ASPCA Poison Control (1-888-426-4435).

ANTICOAGULANT RODENTICIDES

Anticoagulant rodenticides are probably the most commonly used and the most frequent rodenticide toxicities seen through the veterinary emergency room. There are many different formulations in two categories: first-generation and second-generation. First-generation anticoagulant rodenticides (derivatives of “warfarin,” which is a commonly used anticoagulant in human medicine) last for approximately 7 days, but second-generation anticoagulant rodenticides generally last for 4 weeks. Anticoagulant rodenticides work by inhibiting the use of vitamin K in the body, and therefore they prevent the production of clotting factors dependent on vitamin K, leading to a coagulopathy (i.e., internal bleeding). Previously healthy animals should have adequate stores of these clotting factors, so clinical signs of bleeding after ingestion are usually not seen until these stores have been depleted, somewhere between 3-7 days.

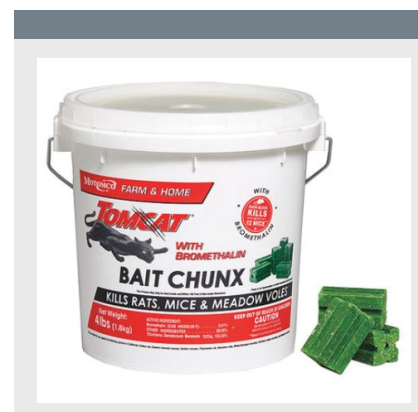
If ingestion of these baits is known to be recent (within hours), standard decontamination protocols (inducing vomiting, use of activated

charcoal) are warranted, followed by treatment with vitamin K therapy for anywhere from 7-30 days depending on the type of bait ingested (first- or second-generation). If ingestion of bait is suspected and the time frame is unknown or there are clinical signs of bleeding already present, clotting times should be checked to confirm a coagulopathy, followed by treatment to stop bleeding and replace blood loss (packed red blood cell and plasma transfusion) to stabilize, followed by treatment with vitamin K as described. Clotting times should be checked between 48-72 hours after finishing the prescribed course of vitamin K to ensure that further therapy is not warranted.

BROMETHALIN

Bromethalin is a neurotoxic rodenticide that, unfortunately, has recently become more commonly used. This particular rodenticide works on the cellular level to cause swelling of cells within the central nervous system, which leads to swelling of the brain and increased pressure within the head (intracranial pressure). Bromethalin is fairly rapidly absorbed in the gastrointestinal tract. Clinical signs of toxicity can occur anywhere from hours to days following ingestion, depending on the dose. The signs that occur when a high dose is ingested typically include hyperexcitability, hyperthermia (increased body temperature), muscle tremors, and seizures, and they occur within 2-12 hours of ingestion. When a lower dose is ingested, clinical signs are often delayed (1-4 days), and usually consist more of progressive ataxia (unsteady gait), decreased use of the hind end, decreased spinal reflexes, muscle tremors, decreased/dull mentation, GI signs (vomiting, anorexia, diarrhea), and abnormal eye motion (nystagmus, abnormal pupil size). Depending on the dose, these signs can progress over weeks to include paralysis, seizures, coma, and death.

Unfortunately, there is no antidote for bromethalin toxicity. Treatment options for bromethalin ingestion/toxicity depend on timing following ingestion. If it has been <4 hours since ingestion, standard decontamination protocols (inducing



vomiting and treatment with multiple doses of activated charcoal) are indicated to minimize absorption of the toxin, followed by supportive care for any clinical signs that may develop. If it has been >4 hours since ingestion, treatment is usually aimed more toward supportive care for clinical signs that develop (increased intracranial pressure and muscle tremors), as decontamination protocols will be less effective given the rapid absorption of the toxin. For animals who ingest a large dose of bromethalin and do not receive immediate intervention, the prognosis is poor. When it is not fatal, recovery may take days to weeks for the neurological signs to fully resolve, and some animals can experience prolonged anorexia, requiring temporary feeding tube placement. At any point during toxicity, development of severe neurologic signs (i.e., seizures, coma, or paralysis) carries a very poor to grave prognosis.

CHOLECALCIPHEROL (VITAMIN D3)

Cholecalciferol rodenticides cause increased absorption of calcium from the GI tract and decreased excretion of calcium through the kidneys, leading to increased levels of calcium in the blood (hypercalcemia) as well as phosphorus (hyperphosphatemia). When calcium and phosphorus reach a certain level in the blood and remain elevated for a prolonged period of time,

EMERGENCY AND CRITICAL CARE

Canine and Feline Extractions and Dealing with Common Complications

CONTINUED FROM PAGE 12

this can lead to mineralization of the soft tissue of the body (kidneys, GI tract, liver, muscles including the heart, etc.). A diagnosis of cholecalciferol toxicity is made based on history of exposure, clinical signs, and consistent findings on blood work. Clinical signs can occur within 12-72 hours from exposure. Clinical signs include anorexia, lethargy, vomiting, diarrhea or constipation, GI bleeding, nausea, decreased motor function, and elevated body temperature.

Much like other rodenticides, immediate treatment includes standard decontamination protocols (inducing vomiting, activated charcoal, and cholestyramine) to minimize the amount of toxin absorbed. For animals who are already clinical or have an elevated calcium noted on their blood work, treatment with aggressive diuresis with fluid therapy and diuretic medication to increase excretion of calcium through the kidneys, steroids, and calcium binders can all be used to try and decrease calcium levels in the blood. If the phosphorus levels are also significantly elevated, phosphorus binders can be used. Follow-up blood work on a daily and then weekly basis is often recommended for up to 4 weeks.

Again, if at any point you suspect your pet may have ingested rodenticide, it is recommended to have them evaluated by a veterinarian immediately. Knowing the type of rodenticide, approximate amount ingested, and approximate date/time of ingestion is very important information to help your veterinarian and animal poison control center to know better what your pet needs for decontamination protocols and for treatment and prognosis.

ZINC PHOSPHIDE

Zinc Phosphide is one of the less commonly seen rodenticides, although it is rapidly fatal. This type of bait is commonly used for control of rats, mice, voles, gophers, and prairie dogs. Zinc phosphide baits work by releasing phosphine gas in acidic environments (i.e., the stomach), which leads to respiratory distress, asphyxiation, gastric distention, and acute abdominal pain, as well as obstructive shock. Once the phosphine gas has been produced, it causes massive cellular death. The onset of clinical signs can be rapid, and even more so on a full stomach as this will decrease the gastric pH (make the stomach more acidic). Clinical signs can be seen within minutes to hours of ingestion and can include lethargy, vomiting (with/without blood), wheezy/rapid breathing, unsteadiness, decreased mental awareness, and seizures. When large doses are ingested, death can occur within 3-5 hours. Unfortunately, there is no antidote to zinc phosphide toxicity. The goals of treatment are focused on decontamination and decreasing the presence of gastric acidity to reduce toxicity below fatal levels. Decontamination includes inducing vomiting (in a well-ventilated area, as the phosphine gas is also toxic to humans), gastric lavage (pumping the stomach), and treatment with activated charcoal and antacids, as well as basic supportive care (fluid support and treatment of shock, liver support).



NSAIDs in Geriatric Patients

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Non-steroidal anti-inflammatory drugs (NSAIDs) are at the forefront of pain management in human and veterinary species. Their benefits include an extended duration of action and analgesic efficacy. Other advantages include multiple formulations (injectable and oral), a large selection of veterinary-labeled products, non-controlled, and non-sedating. However, as with anything, there are disadvantages ranging from mild to severe in dogs and cats. In order to safely administer NSAIDs to geriatric patients, veterinarians need to understand the mechanism of action, potential adverse side effects, and dosing strategies. Most importantly, veterinarians need to educate clients about the importance of pain management in their geriatric pet, but make them very aware of the risks associated with NSAID administration.

MECHANISM OF ACTION

Traditional NSAIDs are inhibitors of cyclooxygenase (COX), working to limit the production of prostanoids – prostaglandins, thromboxanes, and prostacyclins – the products of arachidonic acid breakdown. Prostanoids are important in both health (e.g., gastroprotection, clotting, renal blood flow) and disease (e.g., pain, inflammation, cancer progression). Two COX isoforms, COX-1 and COX-2, are both constitutively expressed and induced. COX-1 expression occurs across almost all tissues and aids in preserving homeostasis. COX-2 is more inducible, increasing in production in tissues where injury occurs. COX-1 has some part in amplifying nociception, but COX-2 has a larger role in pain pathophysiology.

Individual NSAIDs inhibit COX enzymes differently. Since COX-1 is more of a housekeeping enzyme, its inhibition is thought to cause the most NSAID-induced side effects. In an attempt to avoid this, NSAIDs with a greater propensity for COX-2 suppression have been developed and termed “COX-2 selective” or “COX-2 preferential,” mainly meaning drugs that inhibit COX-2 primarily and have less affinity for COX-1. COX-2 selective or preferential NSAIDs are not without risk though

– acute kidney injury (AKI), thromboembolic disease, and gastric ulceration are still possible, which is consistent with the physiological role of COX-2 in a number of tissues.

ADVERSE EFFECTS

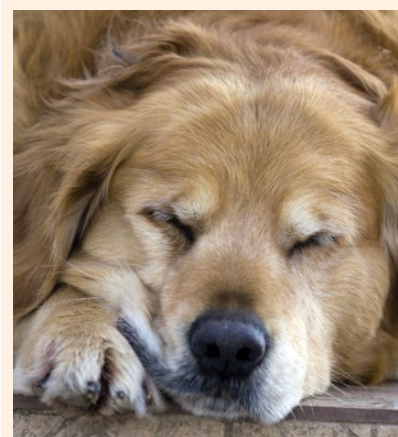
Adverse effects most commonly reported relate to the gastrointestinal (GI), renal, and hepatic systems. Most of the appreciated adverse effects are better documented and researched in dogs. Information is still quite lacking in cats. Dogs are extremely sensitive to the GI effects of NSAIDs. Any patient, dog or cat, displaying inappetence, vomiting, diarrhea, melena, or lethargy could have gastric irritation or ulceration and medication should be stopped. NSAIDs decrease renal autoregulation, impacting the amount of blood flow the kidney receives. NSAIDs should not be used in patients that are dehydrated, hypotensive, or hypovolemic. Given the large percentage of geriatric cats with some degree of chronic kidney disease, NSAID use should be conservative long term and appropriate monitoring is advisable. Hepatotoxicity is a rare side effect of NSAIDs. More importantly, an adequately functioning liver is necessary for drug metabolism to avoid toxicity. Lastly, NSAIDs should be avoided in patients that are hypoproteinemic or thrombocytopenic/coagulopathic (naturally or drug-induced) as risk of toxicity or bleeding increases, respectively, with these disturbances and concurrent NSAID administration.

ADMINISTRATION AND PAIN MANAGEMENT

NSAIDs work peripherally and centrally to elicit their analgesic effects. They can be used to treat or prevent pain in the acute or chronic settings, following injury, and in cancer patients. NSAIDs work synergistically with other analgesics, allowing for dose reduction. Oral bioavailability is excellent following administration and with most NSAIDs, it is recommended to give with food (read prescribing information for drugs periodically to ensure safest administration practices). With chronic NSAID use in geriatric patients, doses should never be exceeded and can

often be reduced to the minimum effective dose that produces a desirable effect. Dogs in particular may not have a desirable effect on one NSAID, in which case it is often useful to try a different NSAID given the appropriate washout period (~3 days). This does NOT hold true for adverse effects – attempting to switch to an alternative NSAID in patients that had undesirable side effects on one NSAID should be done cautiously, if at all.

Geriatric pets deserve adequate comfort during their later years and safe administration of NSAIDs is possible – the main goal is finding the minimum dose needed to keep the pet's quality of life as optimal at home as possible while not causing any major systemic adverse effects. To accomplish this (likely the most important step), client education is essential. Thorough history taking is a must, as many pet owners may not realize their pet is already on an NSAID (e.g., aspirin) or on a medication that should not be given concurrently with an NSAID (e.g., steroid). Adverse drug events more commonly follow improper use of NSAIDs, and this can be lethal. Clients need to be aware of risks associated with both short- and long-term NSAID therapy. It's recommended to have take-home guidelines describing the type of medication, administration instructions, and any clinical signs to watch out for at home that would prompt immediate cessation of the drug and veterinary examination.



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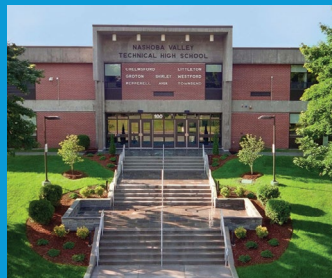
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angell at nashoba

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Left to Right: Dr. Laurence Sawyer. Angell at Nashoba on campus at Nashoba Valley Technical High School (Westford, MA).



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Left to Right: Dr. Erin Turowski. Angell at Essex on campus at Essex North Shore Agricultural and Technical School (Danvers, MA).



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- Pectus Excavatum – Treating a Congenital Anomaly of the Chest Wall – Sue Casale, DVM, DACVS
- Common Pediatric Treatment Myths: Busted – Virginia Sinnott - Stutzman, DVM, DACVECC
 - A look at commonly held beliefs that are either incorrect or outdated about treating various pediatric emergencies with a focus on treatment
- It's Cute When They're Little: Normal vs. Not in Puppies and Kittens – Terri Bright, Ph.D., BCBA-D, CAAB
- Pediatric Endocrine Diseases – Evan Mariotti, DVM, DACVIM

How to Handle Vestibular Disease & Intervertebral Disc Disease

Wednesday, April 7, 2021

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SPEAKERS:

- Rob Daniel, DVM, DACVIM (Neurology)
- Jennifer Michaels, DVM, DACVIM (Neurology)



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*Boston-based pathologists and radiologists serve both Boston and Waltham locations **Available only in Waltham

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