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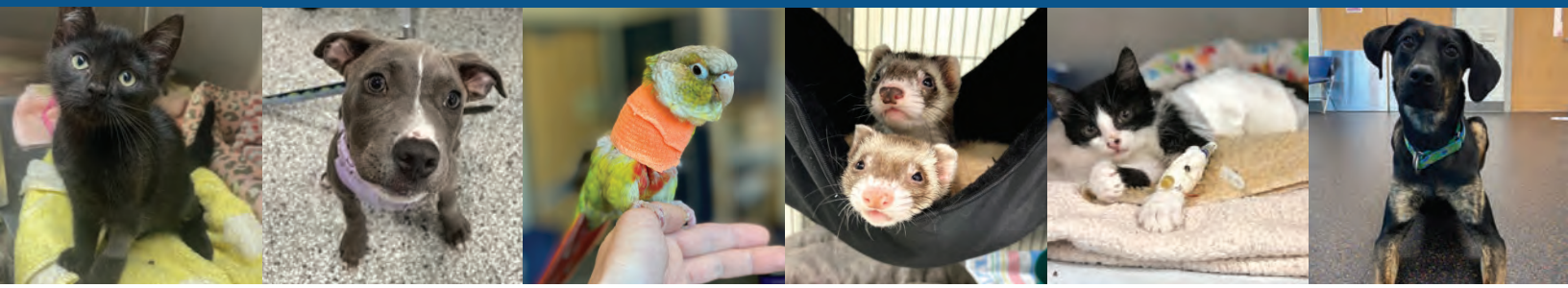

OPHTHALMOLOGY

Golden Retriever Pigmentary Uveitis (GRPU): A Primer for Pet Owners and Their Veterinarians

Martin Coster, DVM, MS, Diplomate of the American College of Veterinary Ophthalmologists
angell.org/eyes | ophthalmology@angell.org | 617-541-5095

Introduction and Terminology

Golden Retriever Pigmentary Uveitis (GRPU) is presumed to be a hereditary eye disease afflicting Golden Retriever dogs, possibly as early as 1.75[1] to 3 years of age,[2] but generally considered to develop between 4.5 years and 14.5 years.[3] Up to 23.9% of Golden Retrievers may be affected after 8 years of age.[4] Most dogs are affected in both eyes, although the onset can be asymmetric.

Due to historical descriptions reported as the disease was emerging, it is sometimes interchangeably referred to as Golden Retriever Uveitis (GRU),[3] Pigmentary Uveitis (PU),[3, 5] and Pigmentary/Cystic Glaucoma (PCG).[1, 6] As of the time of this writing, GRPU seems to be the most accurate descriptive name,[4, 7] as the disease manifests with dispersion of pigment within the eye and symptoms of internal ocular inflammation (uveitis). Dogs often become blind and experience pain from glaucoma (high pressure), which is considered to be from the effects of inflammation and accumulation of pigment and other

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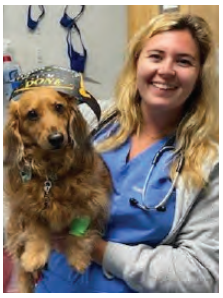

ONCOLOGY

Mast Cell Tumors in General Practice: The Highs and Lows of Canine Cutaneous Mast Cell Tumors

Gretchen McLinden, DVM, DACVIM (Medical Oncology)
angell.org/oncology | oncology@angell.org | 617-541-5136

Mast cell tumors (MCT) are the most common cutaneous tumor in dogs. A primary care veterinarian often diagnoses MCT, and many can have excellent outcomes even without referral to a medical oncologist. However, there is a large spectrum of behaviors for these tumors. This includes the small MCT that will be cured with a straightforward surgery, all the way to the larger or more aggressive MCT that lead to life-limiting disease in just a few months. It is important to understand how to approach these tumors so that clients can be well-informed as to how best to move forward once a diagnosis of MCT is made.

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Golden Retriever Pigmentary Uveitis (GRPU): A Primer for Pet Owners and Their Veterinarians

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substances within the eye. However, even though these eyes appear outwardly inflamed, when histopathology is performed on them, the inflammatory component is typically quite minor.[2, 5] It is unclear whether this is due to the effects of treatment or a feature of the disease. It is also essential to distinguish GRPU from other causes of uveitis, such as neoplasia (e.g., lymphoma) and infectious disease (e.g., *Ehrlichia canis*), beyond the scope of this review.[8]

Diagnosing GRPU

A progressive, insidious disease, GRPU often causes blindness of both eyes due to the effects of glaucoma at its end-stage. Earlier diagnosis requires careful ophthalmologic examination. The Golden Retriever Club of America's (GRCA) Code of Ethics requests that all Golden Retrievers that have been bred have annual eye examinations by a board-certified veterinary ophthalmologist for their entire lifetime, from two to three years of age.[9] It is also recommended that all Golden Retrievers be examined by an ophthalmologist annually due to the prevalence of GRPU.

The mode of inheritance of GRPU has been narrowed down by pedigree analyses to either autosomal dominant with incomplete penetrance or polygenic inheritance.[1] In other words, the condition is considered hereditary. It can be passed on in the genetic code as a dominant gene or multiple genes, but the disease may skip generations. The incidence of GRPU has likely been increasing over the past few decades. Since affected dogs do not develop the disease until after breeding has typically occurred, it has been very difficult to select against GRPU in breeding programs.

No genetic test is currently available for GRPU, but research is ongoing. Blood samples for DNA analysis from any affected dogs, and posthumous donations of eyes (along with a DNA sample) from normal-eyed Golden Retrievers 12+ years old, can be submitted to Dr Townsend at Purdue University.[10] To help support ongoing research efforts, any owner can also choose to have their dogs' DNA banked at the OFA's Canine Health Information Center (CHIC) DNA Repository (blood or cheek swabs) for a minimal fee.[11]

Key features of GRPU include uveal cysts (not necessarily diagnostic for GRPU on their own), radial lens pigmentation, fibrinous debris in the anterior chamber, iris-lens adhesions (posterior synechia), cataract formation, glaucoma, and eventual blindness.

FIGURE 1

› Solitary thin-walled cyst in the left eye of a Golden Retriever. Note the cyst is transilluminated, with the green tapetal reflection shining through. There is also an incidental wedge-shaped cataract at the medial aspect of the lens. This eye was NOT diagnosed with Golden Retriever Pigmentary Uveitis.

FIGURE 2

› Numerous thick-walled uveal cysts in the anterior chamber of the left eye of a Labrador Retriever. This is in contrast to the thin-walled cyst in Figure 1, which is more commonly seen in Golden Retrievers.

Cysts

Uveal (or iris) cysts may be present as an incidental, insignificant finding in Golden Retrievers (Figure 1). Cysts must be distinguished from other pigmentary changes such as pigmentary neoplasia (e.g., melanocytoma or melanoma). *Just because a Golden Retriever has cysts does not mean it has GRPU.* However, the presence of any cystic change within Golden Retriever eyes should warrant more cautious monitoring, perhaps more frequently than annual eye exams. One study showed 61.5% of cases with thin-walled cysts progressed to GRPU within one year.1 It has been postulated that dogs with thin-walled cysts are more likely to develop GRPU than those with thick-walled cysts (Figure 2).

Cysts may be present behind (posterior to) the iris (Figure 3) and may or may not be observable even with dilation. They may be attached to the ciliary body behind the iris or may be found free-floating. Cysts can also be reabsorbed or can rupture, dispersing pigment onto the lens capsule or corneal endothelium

FIGURE 3

› Numerous thin-walled uveal cysts posterior to the iris of the left eye of a Golden Retriever. This eye was NOT diagnosed with Golden Retriever Pigmentary Uveitis, but cautious monitoring was recommended.

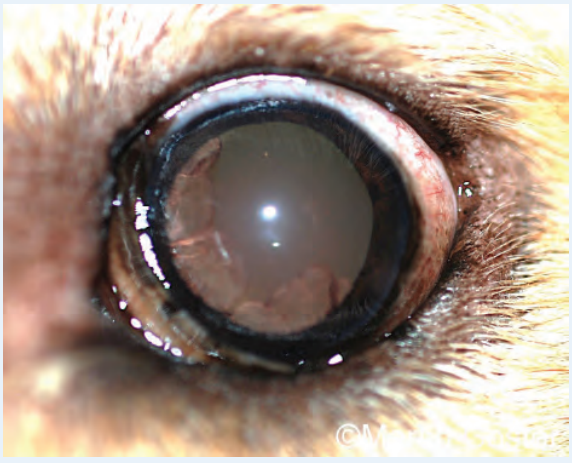

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FIGURE 4

› Thin-walled uveal cyst in the left eye of a Golden Retriever. There is also pigment dispersion on the lens capsule adjacent to the cyst, which is suspected to be from other collapsed cysts. So far, this eye lacks radial pigment, so it is considered at high risk for Golden Retriever Pigmentary Uveitis.

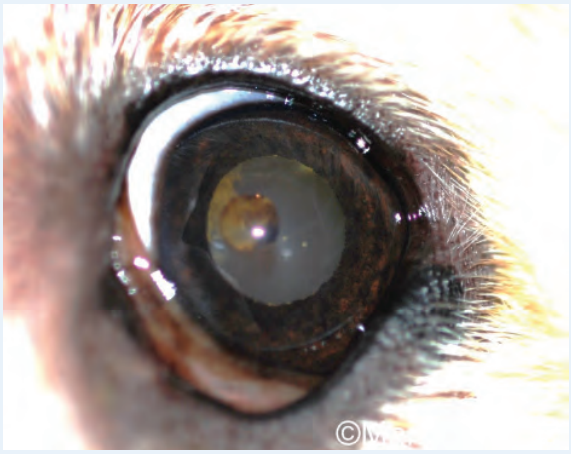

FIGURE 5

› Cysts, pigment, and a collapsed blood-containing cyst (hematocyst, the red area out of focus) in the right eye of a Golden Retriever diagnosed with Golden Retriever Pigmentary Uveitis, but lacking radial pigment dispersion.

(Figure 4). Rarely, cysts may contain blood (hematocysts, Figure 5) or may be associated with intraocular bleeding (Figure 6). Cysts may only be observed using high-frequency ultrasound, but this test is not routinely performed nor recommended for monitoring, as the prognosis for these cysts is unknown.

The uveal cysts in GRPU are thought to contain hyaluronic acid,[2] secreted from the ciliary body epithelium where the cysts arise. This thick substance might be an instigating factor for low-grade inflammation and glaucoma, or may occlude the drainage angle to exacerbate glaucoma. Alternatively, the cysts themselves may mechanically displace the iris forward in some cases, closing the drainage angle to cause glaucoma.

Pigment Deposition

A key diagnostic feature of GRPU is that pigment is deposited onto the front surface of the lens (anterior capsule), in a radial fashion (like the spokes on a bike tire, Figures 6-10). This finding in a Golden Retriever is essentially pathognomonic for GRPU.[3, 4] It is possible that the pigment is deposited onto the lens capsule from the iris during normal pupillary excursions, perhaps exacerbated by inflammation or the presence of thicker fibrinous material within the aqueous humor. However, other non-radially-oriented pigmentation can be present. This deposition can occur from collapsed/deflated uveal cysts (and thus, on its own, this pigment would not be diagnostic for GRPU), and adhesions of the iris to the lens (posterior synechia due to inflammation, which may or may not be from GRPU). Pigment can also disperse onto the inner endothelial lining of the cornea, from collapsed cysts either incidentally or from GRPU (Figure 10).

Fibrinous Debris in the Anterior Chamber

The presence of "cobweb"-like cloudy material throughout the anterior chamber is another diagnostic feature of GRPU (Figures 10, 11). Typically, this is found in combination with other signs such as uveal cysts and pigment dispersion. The material could be fibrin, a byproduct of inflammation, or

FIGURE 6

› Radial pigmentation ("spoke-like" deposition) and blood clot on the anterior lens capsule of the right eye of a Golden Retriever with Golden Retriever Pigmentary Uveitis. Cataract is also present throughout the lens. Bleeding is an uncommon feature of GRPU.

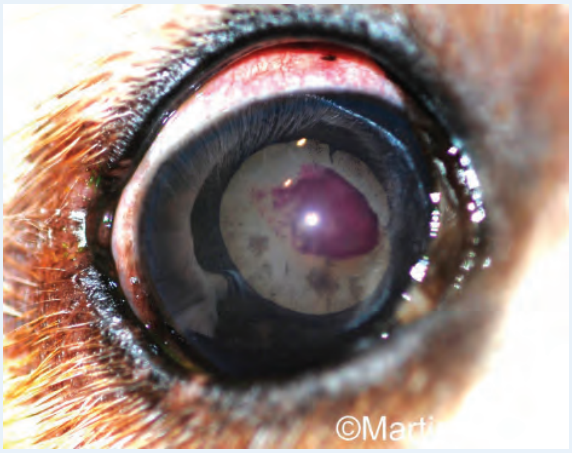

FIGURE 7

› Radial "spoke-like" pigmentation on the anterior lens capsule of the left eye of a Golden Retriever Pigmentary Uveitis patient. There are also larger areas of pigment deposition. Age-related nuclear sclerosis can be seen in the lens. The contralateral eye is shown in Figure 8.

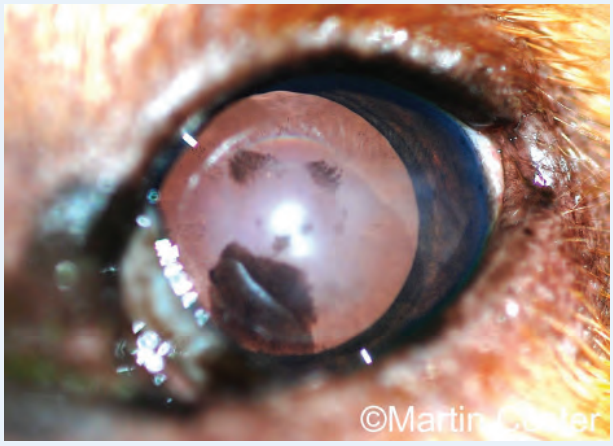

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FIGURE 8

› Radial "spoke-like" pigmentation on the anterior lens capsule of the right eye of a Golden Retriever Pigmentary Uveitis patient. Age-related nuclear sclerosis can be seen in the lens. The contralateral eye is shown in Figure 7.

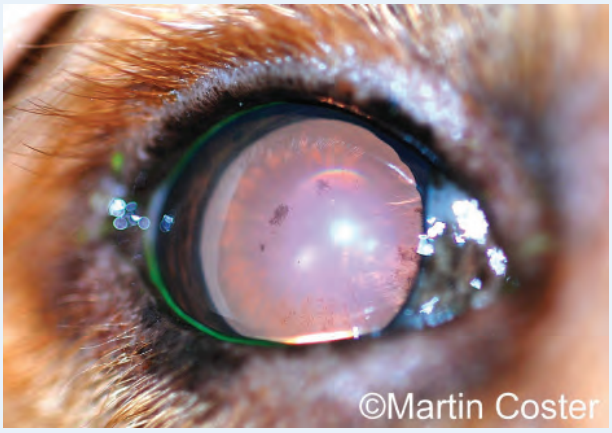
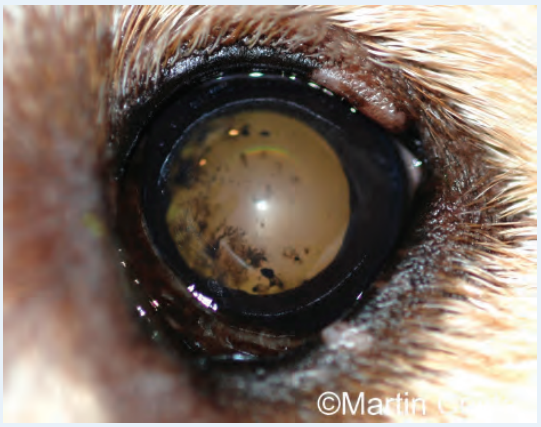


FIGURE 9

› Dense pigmentation across the anterior lens capsule of the left eye of a Golden Retriever Pigmentary Uveitis patient. Some of the classic radially-oriented pigment is present, along with more prominent, larger pigment deposits. This is likely from a combination of pigment exfoliation from the iris and collapsed cysts. Mild age-related nuclear sclerosis is present in the lens.



perhaps a protein-rich fluid released by collapsing cysts or even produced by the ciliary epithelium itself. The evidence that this is fibrin comes from the observation that intraocular injections of tissue plasminogen activator (tPA) may dissolve the material as it would do to fibrin,[3] although other anecdotal reports have suggested no effect.[4] Although only three cases of tPA injection were reported (in which one developed glaucoma), the present author has found the same clinical effect. As reported, though, some patients will develop glaucoma following tPA injection. For this reason, the present author reserves tPA injections for fibrinous eyes that are already succumbing to glaucoma (and thus, perhaps have little left to lose).

Cataract Formation

Golden Retrievers can have congenital cataracts, a hereditary tendency to cataracts, and/or develop age-related cataracts along with lens haze (nuclear sclerosis) later in life. These conditions are distinct from, and not necessarily related to, the cataracts associated with GRPU. Cataracts in GRPU cases typically start anteriorly, where pigment dispersion and cysts are present, and

FIGURE 10

› Fibrinous strands in the anterior chamber of the right eye of a Golden Retriever Pigmentary Uveitis patient. A deposit of pigment on the inner endothelial lining of the cornea is present, to which the fibrinous material attaches; the pigment is likely a collapsed cyst. Internally, less in focus, is radial pigmentation on the lens capsule. Superiorly, partially obscured by the upper eyelid, is a corneal opacity consistent with degenerative keratopathy (perhaps from the inflammatory disease itself or topical steroid treatment).

progression is highly variable. In this author's experience, GRPU-related cataracts are rarely the sole cause of vision loss (which is due to glaucoma instead). Cataract surgery in GRPU-affected patients has not been reported. It is not recommended by the present author, due to a distinct possibility of surgical failure from inflammation and glaucoma, and the overall poor prognosis of affected cases.

Glaucoma

Ultimately, blindness in GRPU patients occurs from intractable glaucoma.[7] In an early report, 50% of cases were blinded within one year,[3] but a more recent study showed 13.7% were blind with a duration of glaucoma of 3.8 years.[7] This difference is likely due to earlier diagnoses from better monitoring and enhanced understanding of the disease, along with earlier therapeutic intervention.

Fibrinous debris and inflammatory scarring (posterior synechia, where the iris adheres to the lens capsule) are risk factors for the development of glaucoma. High ocular pressure damages the retina and optic nerve, and is usually quite painful, even if stoic Golden Retrievers do not always show it. Squinting, tearing, rubbing at the eye, and appetite and behavior changes should be monitored for. Veterinarians should monitor intraocular pressures (IOPs) routinely in GRPU patients. When detected early, glaucoma may be managed with standard glaucoma drops (dorzolamide and/or timolol). Latanoprost is typically avoided due to its potential exacerbation of inflammation, along with the miotic effects on the pupil — constriction of the pupil may cause pupillary block if the pupil entraps thick fibrinous debris and/or cysts. However, this author will cautiously use latanoprost, with informed client consent, if other treatment modalities are failing.

Treatment of GRPU

It is truly unknown whether therapeutic intervention necessarily controls or slows GRPU. However, given that the disease clinically presents with inflammation, topical anti-inflammatories have been the mainstay of therapy. When GRPU-affected eyes are enucleated and examined histologically, the inflammatory component is minimal, but interpretation is limited since most have already been treated.[2] Topical steroids such as prednisolone acetate and dexamethasone (given in the formulation of Neomycin-Polymyxin-Dexamethasone to ensure penetration into the eye) are typically recommended. Non-steroidal anti-inflammatory drugs (NSAIDs) have been postulated to

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FIGURE 11

› Large fibrinous web-like material throughout the anterior chamber of the right eye of a Golden Retriever Pigmentary Uveitis patient. Haze from generalized corneal edema is present secondary to glaucoma.

potentially increase eye pressures, and so are typically avoided.[3] However, this author will use topical NSAIDs (diclofenac or flurbiprofen) when the response to steroids alone is not enough (with cautious IOP monitoring) or if corneal ulceration/infection precludes using a topical steroid. No difference in GRPU progression was noted in a small sample size of cases on a steroid compared to NSAIDs.[7]

Management of glaucoma, as previously described, is paramount once IOP elevates above normal values. Dietary supplementation of an oral antioxidant formulation (e.g., Ocu0GLOTM, Animal Necessity) has been reported, but the effects are unknown.[7]

As already discussed, intraocular (intracameral) injections of tPA may dissolve the fibrinous material, but it often re-forms within three to six months, and sometimes the injection is associated with a profound exacerbation of glaucoma.

Surgical interventions for GRPU are limited. In painful, blinded, glaucomatous eyes, enucleation is warranted and considered the standard of care. The present author has also had success with intraocular gentamycin injections to cease intraocular fluid production and hence control glaucoma, but this procedure is not usually used in other inflammatory glaucoma patients due to the potential for ongoing painful inflammation.

Conclusion

Golden Retriever Pigmentary Uveitis is a devastating disease causing glaucoma and blindness in Golden Retrievers, especially in advanced age. Many will have to be enucleated to control their pain. However, with better understanding and monitoring for the disease, along with early interventional treatments, the prognosis may be improving. There remains a great deal we do not yet know about GRPU. The disease does not yet have a cure, and its best hope is the development of a genetic test. With genetic testing, more selective breeding can occur to hopefully eliminate the disease from the Golden Retriever population.

This article is dedicated to Jack, Oliver, Remington, and all other dogs battling GRPU.

Disclaimers: This article is an original summary of peer-reviewed, published data, written through the lens of the author's 20 years' experience in clinical veterinary ophthalmology practice. No Artificial Intelligence was used in the preparation of this manuscript or images. Images have been cropped and enhanced with simple brightness, contrast, and sharpness adjustments to better show pertinent details, and copyright watermarked, but no other edits have been made. All rights are reserved; permission to harvest the text or images in this article for AI training or other uses is NOT granted.

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↘ Spotlight on Cardiology; Interventional Procedures at Angell

We are excited to welcome Elizabeth Malcolm, DVM, DACVIM (Cardiology) to Angell. Dr. Malcolm joins Katie Hogan, DVM, DACVIM (Cardiology) and Terry Huh, DVM, DACVIM (Cardiology) on the Cardiology team. The team has extensive experience in advanced interventional cardiology and a passion for performing these procedures, including patent ductus arteriosus (PDA) closures, balloon valvuloplasty for pulmonic stenosis, and pacemaker implantation.

With the addition of Dr. Malcolm, we look forward to expanding appointment availability for routine cardiology consultations and reducing the multi-month wait for those appointments. Meanwhile, interventional cases are prioritized and typically scheduled within 1-2 weeks, or sooner. We've also built flexibility into our appointment schedules to ensure timely access for urgent or congenital cases.

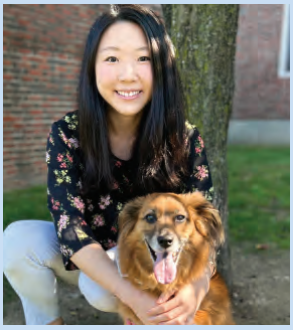
Angell remains a trusted referral destination for interventional cardiology thanks to our valued referring partners, collaborative team, and established infrastructure to ensure excellence. If you have a patient who needs one of one of these procedures, we're here to help quickly and expertly.

angell.org/cardiology

Meet the Cardiology Team

Dr. Katie Hogan

Dr. Katie Hogan is a board-certified cardiologist who completed her cardiology residency at Angell in 2017. She has a strong interest in interventional cardiology and continues to pursue further training in advanced methods of diagnosing and managing congenital heart disease. She performs various interventional catheterization procedures, including PDA repair, balloon valvuloplasty, and pacemaker implantation. She works closely with other specialists to further advance the interventional radiology program at Angell. Her clinical interests include feline heart disease, especially the prevention of thromboembolic diseases, exotic animal cardiology, and arrhythmia management.



Dr. Terry Huh

Dr. Terry Huh is a board-certified cardiologist. She earned her veterinary degree at the Cummings School of Veterinary Medicine at Tufts University, where she first developed an interest in cardiology. Following graduation, she completed a rotating internship at the Animal Medical Center in NYC and a cardiology residency at the University of Pennsylvania. Her cardiology interests include advanced echocardiography, interventional cardiology, and chronic heart failure management.

Dr. Elizabeth Malcolm

Dr. Elizabeth Malcolm is a board-certified veterinary cardiologist who finished her residency at Texas A&M University in July 2024. She is originally from northern California and attended veterinary school at the University of California, Davis. Her cardiology interests include medical management of advanced cardiac disease and interventional cardiology.

ONCOLOGY

Mast Cell Tumors in General Practice: The Highs and Lows of Canine Cutaneous Mast Cell Tumors

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Overview of Canine Cutaneous Mast Cell Tumors

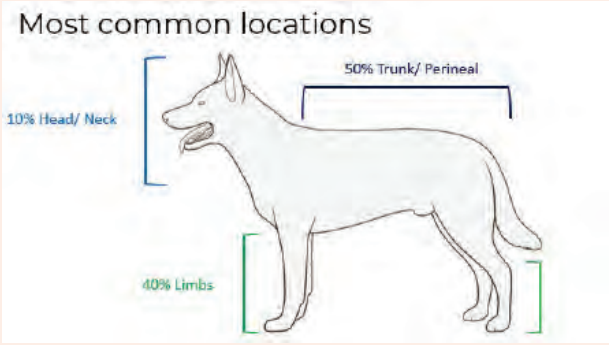
Mast cell tumors are primarily found in older dogs but can be seen at any age. They can take on any appearance. However, many will be small, hairless cutaneous masses on the trunk (50%), limbs (40%), and head or neck (10%). The most important prognostic indicator for MCT is the grade, which requires a biopsy to obtain. Fortunately, low-grade MCT is most common, making up 59% to 81% of all cases. As the work-up, surgical approach, and prognosis change with grade, it can be difficult to figure out how best to navigate MCT once a diagnosis is made via fine needle aspirate. The problem most clinicians run into is that a biopsy is required to determine grade; however, if we were to know the grade in advance, we may approach these cases differently.

Low-Grade vs High-Grade Characteristics

Certain characteristics are used to predict grade in MCT. Small, slowly growing, non-ulcerated, cutaneous tumors are often low grade. Large, rapidly growing, ulcerated tumors found at mucocutaneous junctions (including head/ neck) are typically high grade. Dogs with no systemic signs at the time of MCT diagnosis are more likely to have low-grade tumors, while dogs who feel sick at the time of diagnosis (lethargy, weight loss, stomach upset) are more likely to have high-grade tumors. Brachycephalic breeds typically develop low-grade MCT, while Shar-Peis almost always develop high-grade MCT. Multiple cutaneous tumors are not considered a high-grade characteristic, as each of these tumors should be considered its own individual tumor.

Staging

When there is suspicion for a tumor being high grade based on characteristics, staging is recommended, given the high rate of metastasis for high-grade tumors (up to 95%). Staging is not typically recommended



for low-grade MCT, as the risk of metastasis is much lower (2% to 16 %). The most common area for metastasis for MCT is the regional lymph node (RLN). Therefore, it is important to sample the RLN if it is accessible, especially if it is abnormal. Knowing if there is spread to an RLN helps with prognosis and treatment recommendations. Dogs with MCT that have spread to RLN tend to do better if that lymph node is removed at the time of surgery than those who have metastatic lymph nodes left untreated. The second most common location that MCT spread to are the spleen and/or liver. It is important to note that ultrasound is neither sensitive nor specific at determining if metastasis to the spleen and/or liver is present. Therefore, sampling (fine needle aspirate) is required to determine whether metastasis is present. MCT very rarely spread to the lungs or other regions within the chest. Therefore, chest x-rays are not considered standard-of-care for staging for MCT unless there is a specific concern for a particular patient.

Treatment of Low-Grade MCT

Surgery should be done to remove presumed low-grade MCT, and the mass should be submitted for histopathology to confirm the grade. As mentioned, any abnormal or confirmed metastatic RLN should also be removed during surgery. For tumors that are not amenable to surgery, or for situations where a dog has had multiple cutaneous tumors and the client no longer wishes to pursue surgery, steroids (either systemic/ prednisone or local/ triamcinolone) can be considered. While some tumors will go into a complete remission from steroid therapy, others may only partially respond or remain stable in size. Steroids can also be used to help decrease the size of tumors prior to surgical excision.

Treatment of High-Grade MCT

Given the high rate of recurrence (50%) or metastasis (55% to 95%), the majority of high-grade MCT will lead to life-limiting complications within one year of diagnosis. Therefore, consultation with a medical oncologist to discuss a dog's particular prognosis and chemotherapy is recommended. Before consultation, supportive medications can be used. These medications include H1 antagonists (ex. cetirizine), H2 antagonists (ex. famotidine), PPIs (ex. omeprazole), and prednisone (cytotoxic to mast cells).

Conclusion

Given the broad spectrum of behavior of canine cutaneous mast cell tumors, diagnosing them can be a stressful experience for both the clinician and the client. By being as prepared as possible and understanding what features to look out for and when to recommend further diagnostics or referral, these tumors can become more approachable within the scope of general practice.

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↘ MSPCA-Angell West, Waltham

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The Emergency & Critical Care service at the MSPCA-Angell West (Waltham, MA) is available 24/7 for clients whose pets need immediate medical care for life-threatening trauma or disease.

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TSH Monitoring in I-131 Treated Hyperthyroid Cats

David Carabetta, DVM, DACVECC
angell.org/i131 | i131@angell.org | 617-522-7282

Navigating Post-Radioiodine Hypothyroidism in Feline Hyperthyroidism: The Role of TSH Monitoring

Feline hyperthyroidism, a common endocrinopathy in older cats, is frequently managed with radioactive iodine (RAI) therapy. While RAI is highly effective in resolving hyperthyroidism, it carries the risk of inducing hypothyroidism, a complication that can significantly impact feline health. Therefore, meticulous monitoring is essential, and thyroid-stimulating hormone (TSH) testing plays a pivotal role in detecting and managing this post-treatment complication.

Understanding the Post-RAI Thyroid Landscape

RAI works by selectively destroying hyperfunctioning thyroid follicular cells. While this effectively normalizes thyroid hormone levels, it can inadvertently ablate excessive thyroid tissue, leading to iatrogenic hypothyroidism. This consequence can manifest in various forms:

- Overt Hypothyroidism: Characterized by clinical signs like lethargy, weight gain, decreased appetite, and dermatological changes, along with a total thyroxine (tT4) below the reference range and elevated TSH.
- Subclinical Hypothyroidism: Defined by a tT4 that is within the lower end of the reference range, and TSH that is mildly elevated, suggesting a subtle decline in thyroid reserve.

The Clinical Utility of TSH Testing

TSH, secreted by the pituitary gland, acts as a sensitive indicator of thyroid function. In cats, its concentration is inversely related to tT4 levels. When tT4 decreases, TSH increases to stimulate thyroid hormone production. This feedback loop makes TSH a valuable tool for:

- Early Detection of Hypothyroidism: TSH elevation can precede a decrease in tT4, allowing for the identification of subclinical hypothyroidism before significant clinical signs emerge.
- Distinguishing Between Hypothyroidism and Euthyroid Sick Syndrome: In cats with non-thyroidal illnesses, tT4 may be suppressed, mimicking hypothyroidism. TSH measurement can help differentiate between true hypothyroidism, where TSH is elevated, and euthyroid sick syndrome, where TSH remains within the reference range.

Pre-RAI TSH Considerations

While tT4 and free T4 (fT4) are the primary diagnostic tools for hyperthyroidism, pre-RAI TSH is often suppressed in hyperthyroid cats and can be used to support an otherwise equivocal diagnosis of hyperthyroidism. Detectable levels of TSH at the time of treatment are associated with an increased risk of hypothyroidism post-treatment. They may be a consideration in delaying RAI treatment for cats with mild hyperthyroidism.

Post-RAI Monitoring Protocol

A standardized post-RAI monitoring protocol is crucial for early detection and management of hypothyroidism. A suggested protocol includes:

- Initial Check (1 Month Post-RAI): Measure tT4, TSH, and renal values. This initial check helps confirm the response to RAI treatment and identify cats with overt or subclinical hypothyroidism.
- Intermediate Check (3 Months Post-RAI): Repeat T4, TSH, and renal values. Some cats that initially appear euthyroid could progress to hypothyroid. And some cats initially classified as hypothyroid will show a rebound in thyroid function.

Interpreting TSH Results and Clinical Implications

- Elevated TSH with Low tT4: Indicates overt hypothyroidism. Levothyroxine supplementation is warranted. If this is the initial 1-month post-RAI recheck and no clinical signs or azotemia are present, then delaying treatment and re-evaluation can be considered.
- Elevated TSH with Normal tT4: Suggests subclinical hypothyroidism. Close monitoring and potential levothyroxine supplementation may be considered, especially if clinical signs or new azotemia have developed.
- Normal TSH with Normal tT4: Indicates euthyroidism. Continued monitoring is recommended.
- Suppressed TSH with elevated tT4: Indicates persistent hyperthyroidism. Further treatment may be required.

Detecting Occult and Subclinical Hypothyroidism

The sensitivity of TSH in detecting subtle changes in thyroid function makes it a powerful tool for identifying occult and subclinical hypothyroidism. By monitoring TSH levels, veterinarians can identify cats with early thyroid dysfunction before they exhibit overt clinical signs. This proactive approach

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allows for timely intervention and prevents the development of more severe hypothyroidism. Supplementation of levothyroxine can provide a survival benefit in some iatrogenic hypothyroid cats.

Benefits of Early Detection and Intervention

Early detection and intervention of post-RAI hypothyroidism offer several benefits:

- Improved Quality of Life: Early intervention enhances the cat's overall well-being by preventing or minimizing clinical signs.
- Reduced Morbidity: Hypothyroidism can lead to various complications, including cardiac dysfunction and neurological abnormalities. Early treatment minimizes these risks.
- Enhanced Client Satisfaction: Proactive monitoring and management demonstrate a commitment to optimal patient care, fostering client trust.
- Preventative care: Catching issues before they become clinical allows for more effective treatment and less suffering for the cat.

Conclusion

TSH testing is an indispensable tool in the post-RAI management of feline hyperthyroidism. Its sensitivity and specificity make it highly effective in detecting overt and subclinical hypothyroidism. By incorporating TSH monitoring into a standardized post-RAI protocol, veterinarians can provide optimal care for their feline patients, ensuring their long-term health and well-being. Proactive monitoring, coupled with prompt intervention, allows for the successful management of RAI-induced hypothyroidism, ultimately improving the quality of life for cats with hyperthyroidism.

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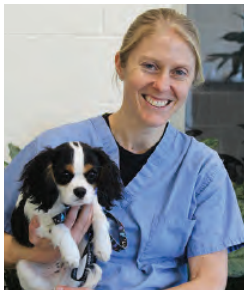

Treating Anesthetic Hypotension: Beyond the Fluid Bolus

Becca Reader, DVM, DACVAA
angell.org/anesthesia | anesthesia@angell.org | 617-541-5048

Introduction

Many of the commonly used anesthetic agents produce dose-dependent vasodilation, and hypotension is frequently observed in patients placed under general anesthesia. Left untreated, anesthesia-related hypotension can have significant consequences. As a result, it is important to recognize and treat hypotension when it occurs. One of the most commonly utilized treatments for hypotension under anesthesia is the administration of an intravenous fluid bolus. However, several other factors may be contributing to low blood pressure. This article will discuss additional contributors to anesthesia-related hypotension and provide a visual algorithm to help determine which factors are likely contributing to low blood pressure and how to treat them.

› *Photo courtesy Phil Zeltzman*

Hypotension: Definitions

Traditionally, hypotension under anesthesia has been defined as a mean arterial blood pressure (MAP) below 60 mmHg.[1] This is considered the minimum pressure required to provide adequate blood flow to the kidneys. In addition to maintaining a MAP > 60 mmHg under anesthesia, one should also aim to maintain a systolic blood pressure (SAP) > 90 mmHg and a diastolic blood pressure (DAP) > 40 mmHg. At pressures lower than this, there is a risk of causing acute kidney injury secondary to hypoperfusion.

Many would argue that the lower limit for an acceptable MAP should actually be slightly higher than 60 mmHg. Recall that autoregulatory mechanisms allow for maintaining constant blood flow between MAPs of 60 to 160 mmHg. However, autoregulatory mechanisms rely on input from the sympathetic nervous system, which is depressed under general anesthesia. Additionally, if a MAP of 60 mmHg is considered the minimal acceptable blood pressure, patients may not be assessed as hypotensive, and treatments may not be initiated until the patient is outside the zone of autoregulation.[2] As a result, it is likely more appropriate to set a MAP of 70 mmHg as the lower limit for acceptable blood pressure under anesthesia.

Mean arterial blood pressure is determined by the product of the patient's cardiac output (CO) and systemic vascular resistance (SVR). This can be represented by the equation MAP = CO x SVR.[2] Cardiac output can be further broken down into the product of heart rate (HR) and stroke volume (SV). Rewritten with the expanded definitions, MAP is determined by the product of HR x SV x SVR. Derangements in any one of these components may result in a low MAP.

Anesthesia-Associated Hypotension: Rationale Behind the Fluid Bolus

As mentioned above, many of the commonly used anesthetic drugs produce dose-dependent vasodilation. It is also important to note that of these drugs, inhalant anesthetics (e.g., isoflurane) cause vasodilation at clinically relevant concentrations.[3] In addition to causing arterial vasodilation, anesthetic drugs also cause venodilation. This venodilation effectively increases venous capacitance, making it appear that the venous circulation can hold a larger blood volume.[3] Because the animal's blood volume has not actually changed, this results in a "relative" hypovolemia manifesting clinically as hypotension.[3] Since the underlying cause of this hypotension is dose-dependent vasodilation secondary to anesthetic agents, the first action steps should be to decrease the concentration of inhalant being delivered to the patient, if feasible.

An intravenous fluid bolus is also considered an appropriate intervention for treating anesthesia-associated hypotension. However, the fluid bolus is not directly intended to correct the "relative" hypovolemia. Instead, the administration of an IV fluid bolus takes advantage of the impact that preload has on the strength of cardiac contraction (or stroke volume). Up to a certain point, the strength of ventricular contraction is proportional to the amount of blood returning to the right side of the heart. An IV fluid bolus will increase the amount of blood returning to the heart, resulting in an increased "stretch" of cardiac myocytes, increasing stroke volume and improving cardiac output.[3] Recall the equation, MAP = HR x SV x SVR; in this situation, an IV fluid bolus increases the cardiac stroke volume to improve MAP.

Other Contributors to Hypotension

Looking closely at the components of MAP (HR x SV x SVR), it is clear that several factors may contribute to hypotension under anesthesia, and intravenous fluids will only address a few of these factors. Derangements in heart rate, particularly bradycardia, may result in decreased cardiac output and contribute to hypotension under anesthesia. In this circumstance, an IV fluid bolus would be unlikely to improve the patient's blood pressure, and interventions should instead be aimed at increasing the patient's heart rate. Likewise, many anesthetic agents - and sometimes underlying disease - can decrease a patient's cardiac contractility. In this circumstance, efforts should be made to reduce the concentration of anesthetic agent delivered to the patient or improve cardiac contractility using a positive inotrope. Finally, as discussed previously, most anesthetic drugs reduce systemic vascular resistance through dose-dependent vasodilation. Every effort should be made to decrease the concentration of these agents administered

Treating Anesthetic Hypotension: Beyond the Fluid Bolus

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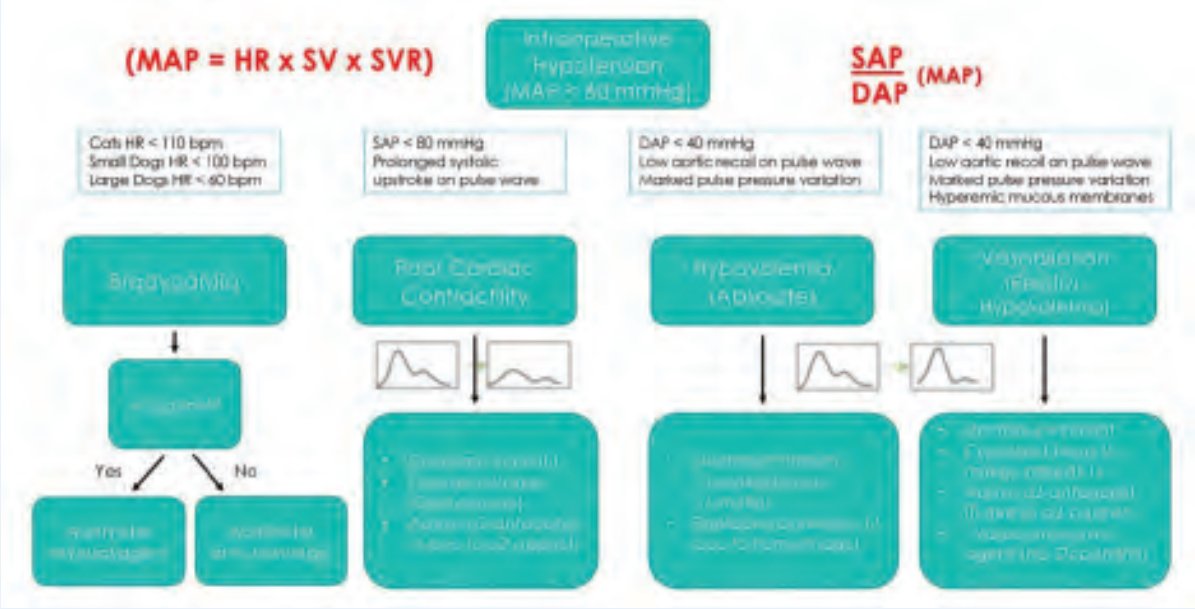
› A visual algorithm illustrating the various potential contributors to hypotension under anesthesia. Mean arterial pressure (MAP) is determined by the product of heart rate x stroke volume x systemic vascular resistance. Derangements in any one of these components may result in a low MAP, and treatments should be aimed at the most likely contributing factor to the patient's low blood pressure.



to the patient, and under specific circumstances, an IV fluid bolus may be an appropriate intervention. However, if an IV fluid bolus fails to improve the patient's blood pressure, subsequent interventions should improve vascular tone using vasopressors.

Inotropes and Vasopressors

Inotropes (e.g., dobutamine) increase myocardial contractility, whereas the administration of vasopressors (e.g., norepinephrine, phenylephrine, vasopressin) causes vasoconstriction and increased systemic vascular resistance. Dopamine has dose-dependent effects and doesn't tend to fit nicely into one category or another (see below for more detail). The decision of which inotrope or vasopressor to use and when requires a fundamental understanding of the mechanism of action of each agent, as well as a clinical interpretation of the pathophysiology behind the low blood pressure.

As mentioned above, dopamine is an endogenous catecholamine with dose-dependent effects. The typical dose rates used for dopamine are 1 to 15 mcg/kg/min.[1,2] At low doses (1–5 mcg/kg/min), dopamine's main sites of action are at the dopamine 1 and 2 (DA1 and DA2) receptors, resulting in renal vasodilation and increased blood flow to the kidneys.[2] Mid- to high-range doses (5–10 mcg/kg/min) activate beta-1 receptors, increasing heart rate and contractility.[2] Doses greater than 10 mcg/kg/min activate alpha-1 receptors, resulting in peripheral vasoconstriction.[2] The recommended dose for dopamine for improvement of cardiac output is 7 mcg/kg/min, but ultimately, the CRI should be dosed to effect based on response (or lack thereof). This author typically uses dopamine as a first-line agent to treat anesthesia-associated hypotension refractory to the IV fluid bolus.

Norepinephrine is an endogenous catecholamine with primary effects at both alpha-1 and beta-adrenergic receptors, with effects at the alpha-1 receptors predominating.[2] The typical dose rates used for norepinephrine are 0.05 to 2.0 mcg/kg/min.[1,2] At low to mid-range doses, norepinephrine activates primarily alpha-1 receptors, resulting in peripheral vasoconstriction and increased systemic vascular resistance.[2] At high doses, norepinephrine will activate beta-1 receptors, increasing heart rate and contractility.[2] Norepinephrine is best used to treat septic shock or other disease states that result in systemic vasodilation.

Dobutamine is a synthetic catecholamine with primary effects at beta-1 and beta-2 receptors.[2] The typical dose rates used for dobutamine are 1 to 10 mcg/kg/min.[1,2] At lower dose ranges (1–5 mcg/kg/min), dobutamine activates primarily beta-1 receptors, increasing cardiac contractility and heart rate.[2] At higher doses, dobutamine may activate beta-2 receptors and will induce a decrease in systemic vascular resistance due to beta-2-mediated vasodilation.[2] Dobutamine is typically used in patients with known cardiac disease that is associated with decreased contractility or in clinical situations where either drugs or disease are known to impact cardiac contractility negatively.

Conclusion

Intravenous fluids are important in treating anesthesia-associated hypotension, but an IV fluid bolus may not always be the most appropriate intervention. It is important to evaluate all contributors to MAP (MAP = HR x SV x SVR) and target interventions at the component of the MAP equation most likely to be causing the patient's low blood pressure.

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Canine Testicular Neoplasia

Pamela Mouser, DVM, MS, DACVP
angell.org/lab | pathology@angell.org | 617-541-5014

Bob Barker, the 35-year host of "The Price is Right" and a strong animal rights advocate, would prompt viewers to spay and neuter their pets at the end of each television broadcast. This mantra was subsequently passed to the current host of the game show, Drew Carey. The shift in veterinary medicine to promote pet sterilization, which occurred in the 1970s, is touted as a major factor in reducing the number of animals presented to—and euthanized by—shelters in the decades that followed.[1] This widespread practice of sterilizing animals at an early age, which is now commonplace in the United States, likely reduced the number of testes submissions to diagnostic pathologists over time, as gonads were routinely removed prior to developing pathology. While not necessarily the basis of the 1970s sterilization movement, early castration certainly prevents the development of testicular tumors. This article is designed to refresh your memory of testicular tumor types, summarize testicular tumor diagnoses made over a ten-year period at Angell Pathology, and compare Angell tumor types/numbers to published retrospective studies from around the world.

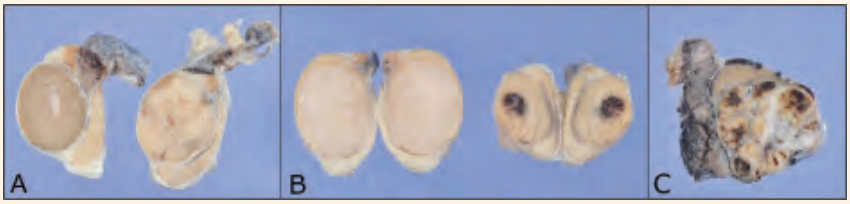
Canine testicular tumors arise from one of two—sometimes both—basic components in the testis: sex cord stromal elements or germ cells.[2] Sex cord stromal tumors include Sertoli cell tumors and interstitial (aka Leydig) cell tumors. Seminoma is the predominant germ cell tumor type. In some instances, a single tumor may have both germ cell and sex cord stromal elements, earning the classification of mixed germ cell-sex cord stromal tumor (MGC-SCST). In short, and as a potential spoiler alert for all the upcoming data in this article, dogs with testicular tumors will overwhelmingly have ONE OF THREE TUMOR TYPES: Sertoli cell tumor, interstitial cell tumor, or seminoma. Individual tumors with mixed components (MGC-SCST) or testes containing more than one of these tumor types are not uncommon. Any other tumors, such as the ever-exciting teratoma containing teeth or hair, are remarkably rare.

Canine Testicular Tumors: Pathology

Sertoli cell tumors, which arise from the supporting cells lining the seminiferous tubules, typically appear as nodular to multinodular, white to grey (less commonly tan to yellow), very firm masses. Expansile growth, causing testicular enlargement or distortion, may occur with Sertoli cell tumors (Figure 1C). While malignancy is uncommon, larger tumors may have an increased risk of metastasis. Histologically, neoplastic cells resemble their normal counterparts with elongate columnar to fusiform cell shape and small nuclei, and demonstrate either intratubular or diffuse growth supported by dense fibrous stroma. The fibrous stroma contributes to the firm nature of Sertoli cell tumors grossly, and can be a helpful distinguishing factor for this tumor type. Sertoli cell tumors may get more attention than the other two testicular tumor types as they are the most likely to be hormonally productive, causing a paraneoplastic syndrome of apparent hyperestrogenism. Visually observable clinical signs may include enlarged nipples or gynecomastia, pendulous prepuce, altered behavior around male dogs, contralateral testicular atrophy, and bilaterally symmetric alopecia.[2]

FIGURE 1

Formalin-fixed testes from three dogs. A: Half of each bisected testis with attached epididymis from a 15-year-old golden retriever. The testis to the left is relatively normal to serve as a comparison. The testis on the right is diffusely expanded by a homogenous tan bulging mass, confirmed microscopically to be a seminoma. B: Both bisected testes from an adult Chihuahua. The homogenous tan mass expanding the testis on the left is a seminoma. The discrete nodular yellow mass with focal hemorrhage in the testis on the right is an interstitial cell tumor. C: Half of the bisected left inguinal (cryptorchid) testis from a 5-year-old husky presenting with alopecia and mammary enlargement. The expansile mass distorting the testis is multinodular, tan to yellow with multifocal hemorrhage, and has abundant dense white fibrous stroma. The mass was confirmed microscopically to be a Sertoli cell tumor.



Interstitial cell tumors, arising from the androgen-producing cells between seminiferous tubules, appear grossly as discrete, yellow, soft nodules with frequent hemorrhage (Figure 1B). These tumors are rarely malignant. Like Sertoli cell tumors, interstitial cells forming masses resemble their normal counterparts with polygonal cell shape, abundant eosinophilic cytoplasm, fine lipid-type vacuolation, and small nuclei. Stroma separating cells into nests and lobules is fine, unlike the dense fibrous stroma seen with Sertoli cell tumors. Hormonal production and paraneoplastic syndromes are considered rare with this tumor type.[2]

Seminomas are derived from germinal epithelium (spermatogonia) within seminiferous tubules. Grossly, these tumors are homogenous pale tan, grey, or white, bulge from the cut section, and range from soft to moderately firm (Figure 1A, B). The color and consistency bear some resemblance to lymphoma. These masses can become quite expansive, resulting in enlargement or deformation of the affected testis. Similar to Sertoli cell tumors, seminomas can have a tubular or diffuse growth pattern microscopically. Neoplastic germ cells are large, round to polyhedral, and have large nuclei with prominent nucleoli. Variation in nuclear size (anisokaryosis) and multinucleated cells are common, as are mitotic figures, giving all seminomas a malignant histologic appearance despite most tumors having benign behavior.[2]

Canine Testicular Tumors: Comparison

A search of biopsies submitted to Angell Pathology from the past ten years resulted in 156 testes from 107 dogs. Of these submissions, 92 testes (59%) had tumors identified; 46 (29%) had non-neoplastic lesions, including processes such as inflammation, vascular compromise, or atrophy; 13 (8%) had no significant microscopic abnormalities; and 5 (3%) did not include identifiable testicular tissue. Dogs with tumors ranged from 4 to 17 years in age.

PATHOLOGY

Canine Testicular Neoplasia

CONTINUED FROM PAGE 14

TABLE 1

› Canine testicular tumors from Angell Pathology compared with published retrospective studies.

Study	Sertoli Cell Tumor	Interstitial Cell Tumor	Seminoma	Mixed Germ Cell-Sex Cord Stromal Tumor (MGC-SCST)	Multiple Tumors	Other Tumors
Angell 2014-2025*	19/92 (20.6%)	30/92 (32.6%)	25/92 (27.2%)	2/92 (2.2%)	16/92 (17.4%)	
N. America 1976[4]**	137/410 (33.4%)	96/410 (23.4%)	127/410 (31.0%)		46/410 (11.2%)	4/410 (1.0%)
Poland 2007[5]***	7/55 (12.7%)	9/55 (16.4%)	34/55 (61.8%)		2/55 (3.6%)	3/55 (5.5%)
Italy 2008[6]	9/110 (8.2%)	55/110 (50%)	46/110 (41.8%)			
Taiwan 2009[4]	16/96 (16.7%)	25/96 (26%)	33/96 (34.4%)	22/96 (22.9%)		
Italy 2012[7]	37/183 (20.2%)	18/183 (9.8%)	108/183 (59%)	10/183 (5.5%)		10/183 (5.5%)
Slovenia 2014[8]	59/301 (19.6%)	86/301 (28.6%)	144/301 (47.8%)	12/301 (4%)		
Brazil 2020[9]	61/220 (27.7%)	64/220 (29.1%)	88/220 (40%)	7/220 (3.2%)		
Russia 2022[10]	80/447 (50.8%)	227/447 (50.8%)	107/447 (23.9%)	33/447 (7.4%)		

Highlighted cells denote the most common tumor type in each study.
All studies reported data on the total number of TUMORS with the exception of:
**Data reported out of a total number of TESTES.*
***Data reported out of a total number of DOGS.*

Table 1 lists the number and percentages represented by each tumor type out of the total number of testes evaluated. Note that all testicular tumors evaluated at Angell during the selected timeframe are one of the three types described above, with two mixed tumors containing a combination of germ cells and sex cord stromal elements (MGC-SCST). More than 15% of testes had multiple tumors observed within the same testis. Table 1 also compares Angell's data with several published retrospective studies from multiple countries worldwide. Note that most studies reported data from a total number of tumors, not the number of testes or dogs, which removes the data point for multiple tumors. Highlighted cells denote the tumor type diagnosed most frequently in each study. The "other" tumors identified in three of the studies included five hemangiosarcomas, four hemangiomas, and one each of granulosa cell tumor, sarcoma, carcinoma, fibrosarcoma, gonadoblastoma, adenomatoid tumor, mesothelioma, lipoma, and metastatic mast cell tumor from scrotal skin.

Canine Testicular Tumors: Take-Home Points

- Canine testicular tumors are overwhelmingly one of three types: Sertoli cell tumor, interstitial (Leydig) cell tumor, or seminoma.
- Paraneoplastic signs of apparent hyperestrogenism (feminization, symmetric alopecia, contralateral testicular atrophy) are most often associated with Sertoli cell tumors.
- While Sertoli cell tumors and seminomas may infrequently be malignant, the vast majority of canine testicular tumors are benign and cured by castration.
- While not explicitly discussed in this article, cryptorchidism increases the risk of testicular tumor development.

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Canine Parvovirus and the Canine Parvovirus Monoclonal Antibody

Catherine Sumner, DVM, DACVECC
angell.org/emergency | emergency@angell.org | 781-902-8400

Canine parvovirus (CPV) is a highly contagious disease first recognized in the 1970s. Despite broadly available and effective vaccines, it remains dangerous for dogs today. Young puppies and dogs that have not been vaccinated are the most likely to contract CPV, as well as those with a weakened immune system. Transmission of CPV occurs via direct contact (dog to dog) or, commonly, through indirect exposure to environments, objects, etc., that have been contaminated with infected feces. Parvovirus is a very hardy virus, able to survive for months outside of an animal in the environment. The virus is resistant to extremes of temperature and humidity, and many common household cleaning products do not destroy it. Because a dog infected with CPV sheds a huge number of virus particles into the environment, another dog can be unknowingly exposed by visiting an area after an infected dog. Additionally, an infected dog can continue to shed viral particles for two weeks after the resolution of their clinical signs.

When a vulnerable dog is initially infected with CPV by ingesting the virus, there is an incubation period of ~3 days to 1 week before clinical signs are noted. During this time, the virus replicates in oropharyngeal and mesenteric lymph nodes, invading lymphocytes and spreading CPV elsewhere in the animal through the bloodstream. Then, CPV infects rapidly dividing cells from the bloodstream, notably those of the small intestinal epithelium and the bone marrow. The virus binds to the transferrin protein receptor on the cell's surface to enter cells.

The main clinical signs of CPV result from the injury to these rapidly dividing cells that the virus targets. Damage to the small intestinal epithelium results in a breakdown of the blood-intestinal barrier, an essential barrier preventing fluid loss through the GI tract and bacterial translocation from the GI tract into the bloodstream. It also prevents the absorption of nutrients and results in gastrointestinal bleeding. The small intestinal epithelium in a healthy dog is continuously being replaced with new cells made in the crypts of Lieberkühn, but CPV prevents these new cells from being made. Meanwhile, CPV destroys the developing immune cells in the bone marrow, eventually resulting in decreased white blood cell counts. Clinical signs include anorexia, lethargy, profound vomiting and diarrhea, gastrointestinal bleeding, dehydration, and progressing to hypovolemic shock. They can progress to include infection and sepsis secondary to bacteria crossing the blood-intestinal barrier into the bloodstream.

Historically, treatment of CPV has been essentially supportive care and symptomatic treatment, with many patients developing clinical signs to the point that they require hospitalization. For dogs that are hospitalized, many will need more than three days in the hospital, which is a significant financial burden for the patient's family. The mainstays of treatment have been aggressive IV fluids, anti-emetics, analgesics, antibiotics to address secondary infections, and nutritional support. Ultimately, treatment has essentially supported the patient while the virus "runs its course."

In 2023, a targeted canine parvovirus monoclonal antibody (CPMA) became commercially available through USDA conditional approval. The CPMA is a chimeric antibody, meaning it has portions from two species: a dog constant region and a rat variable region. The rat variable component is the part of the CPMA that interacts with CPV; the CPMA rat variable portion binds to a location on CPV that would typically bind to the transferrin receptor on a host cell. With this CPV location blocked, the CPV cannot bind to the transferrin receptor and thus cannot enter the host cell. If CPV cannot enter cells, those cells will not be damaged, preventing adverse clinical sequelae.

EMERGENCY & CRITICAL CARE

Canine Parvovirus and the Canine Parvovirus Monoclonal Antibody

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Studies performed on CPMA have demonstrated that patients receiving CPMA had a quicker resolution of clinical signs, which can translate into a shorter hospitalization and prevent complications. In an efficacy study on puppies testing positive for CPV, those treated with CPMA versus a control group had a faster resolution of severe diarrhea, vomiting, fever, inappetence, and lymphopenia. Additionally, there was 0% mortality in the CPMA group versus 57% mortality in the control group. It also appears that dogs testing positive for CPV treated with CPMA shed less parvovirus in their feces within days of treatment, which could decrease the amount of CPV in the environment, which is of note given how resilient CPV is. In a study of 147 healthy dogs administered CPMA, it was well tolerated, with no anaphylactic reactions reported. 4% of these healthy dogs developed injection site reactions, which was the most common adverse reaction recorded.

CPMA is labeled for use in dogs at least eight weeks or older. It is dosed at 0.2 mL/kg and administered intravenously. CPMA is delivered frozen and must be kept frozen until use (stored at less than or equal to 5° F). When ready to be used, it should be thawed at room temperature and then administered immediately. CPMA is only administered once to a patient, and it should be administered as early as possible in an affected patient, as blocking CPV from entering host cells will slow the effects of the virus. In a patient severely affected by CPV, administering CPMA may not help significantly, as CPV has already entered cells and caused the damage, resulting in observable clinical signs. It is also important to note that patients treated with CPMA should continue to be treated with all the other supportive measures used historically to treat CPV patients. CPMA is an addition to our treatment options but does not replace the existing mainstays of treatment.

Canine parvovirus is a disease that we treat with regularity, and it is exciting to have a targeted treatment for it beyond symptomatic and supportive care. Since CPMA is a newly available treatment option, there is little clinical experience. It has the potential to decrease the significant morbidity, mortality, financial burden, and emotional burden of treating dogs with CPV.

↘ New Boarding and Grooming Services at Angell

The MSPCA-Angell is excited to introduce in-house, healthy boarding and grooming for Angell clients. The new space—built alongside our expanded Critical Care Unit on the second floor—offers pets a safe, comfortable stay and owners peace of mind.

Opened at the end of August, the service is still in its launch phase as we build a program that matches the exceptional care Angell is known for. Having a veterinary hospital on-site sets us apart, and while the hospital is separate from the boarding and grooming areas, medical support is available if needed. Boarding guests will enjoy enrichment and personalized attention, while grooming will focus on wellness and creating a positive experience for pets and owners.

Clients who have visited non-emergency/non-urgent Angell services in the past year are eligible to book. Reservations will be available online or by phone for convenience and personal service.

Brielle Friedman, Manager of Boarding and Grooming, with Nova (left) and Vina (right).

Managing Rabbit Dental Disease and Owner Expectations for the Referring Veterinarian

Lauren Gawel, DVM
mspca.org/waltham_services/avian-and-exotic-medicine/ | AngellWestAE@angell.org | 617-989-1561

Introduction

Rabbits are the third most popular companion mammal in America, behind dogs and cats, with an estimated 1.5 million households owning them, according to the AVMA.[15] Dental disease is one of the most common disorders of rabbits, affecting 10% or more of rabbits presenting to referral hospitals and up to 40% of rabbits considered healthy by owners.[9,6] Dental disease tends to be overwhelmingly due to incorrect diet, and severe disease can occur as early as 4 months of age.[6] It is important to continually assess dentition in any rabbit presenting either for wellness or emergency medical care and to have husbandry discussions with owners right away to reduce the risk of serious progressive disease.

Dental Anatomy and Causes of Disease

Rabbit dental anatomy is highly unique, and understanding it is integral to diagnosing, assessing, and treating dental disease. It is highly recommended to review the information found here: Dentition and Disease of the Domestic Rabbit (Oryctolagus cuniculus) by MSPCA Angell's Brendan Noonan, DVM, DABVP.[8]

Diagnosis and Clinical Signs

Rabbits often present emergently with vague clinical signs of reduced appetite and defecation, summarily labeled "GI stasis". However, "GI stasis" is not a diagnosis; rather, it is a collection of clinical signs, including hyporexia to anorexia and reduced to absent fecal production. GI stasis can be caused by anything from stress to organ failure and everything in between, most certainly including dental disease. Other clinical signs that can indicate underlying dental disease include ptyalism, moist dermatitis of the chin and/or neck, change in feed preferences, quidding (spitting out partially chewed feed), bruxism, facial masses, exophthalmia, ocular discharge, and respiratory disorders.[13,6]

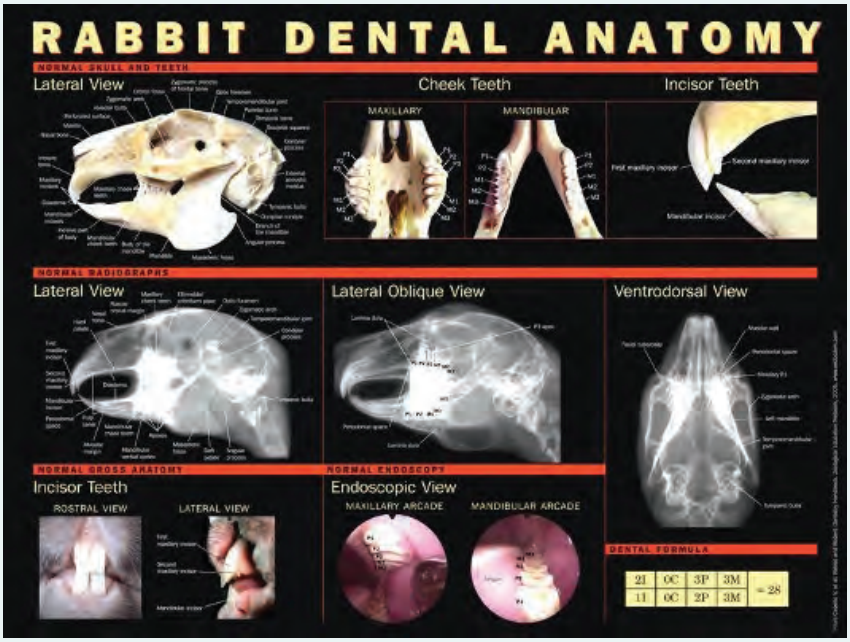
A combination of physical exam and imaging should make the diagnosis. The physical exam should involve an extraoral assessment with palpation along the maxilla and mandible to assess for swellings, bony irregularities, lack of symmetry, or moisture. Additionally, an intraoral exam should be performed by direct visualization of the incisors and with an otoscope featuring a wide-diameter cone, or, if available and with some training, a bivalve nasal speculum, to view the cheek teeth. The cheek teeth arcades should be assessed for wavemouth or stepping of the arcade (some teeth in the arcade being taller than others), points (also observe and note any suspected gingival proliferation of the maxillary gums, as this can hide the severity of buccal points), and ulcerations of the tongue or cheeks. In cases of more severe infection, you may see or smell purulent material and/or brown, misshapen, or missing teeth.

Intraoral exam, while necessary, is inherently limited by the small gape of a rabbit and only reveals about 20% of potential disease.1 Skull radiographs or CT can be utilized for imaging, though CT is largely preferable. If taking skull radiographs, deep sedation is needed for exact positioning for truly diagnostic images. Essential views of the head include laterolateral (LL), right-to-left and left-to-right latero-oblique (LO), and dorsoventral (DV) or ventrodorsal (VD).[5] Rostrocaudal and intraoral views assess other specific areas.[5]

CT also requires sedation, although a CT scan can often be achieved with lighter sedation in a faster timeframe, as positioning is simpler. Furthermore, with a CT assessment, superimposition is inherently avoided, image quality is improved, and 3D visualization enables superior assessment, resulting in an 80% increase in diagnosis accuracy and over a 56% increase in guiding extraoral treatment plans.[2]

FIGURE 1

› Rabbit dental anatomy chart.3



Managing Rabbit Dental Disease and Owner Expectations for the Referring Veterinarian

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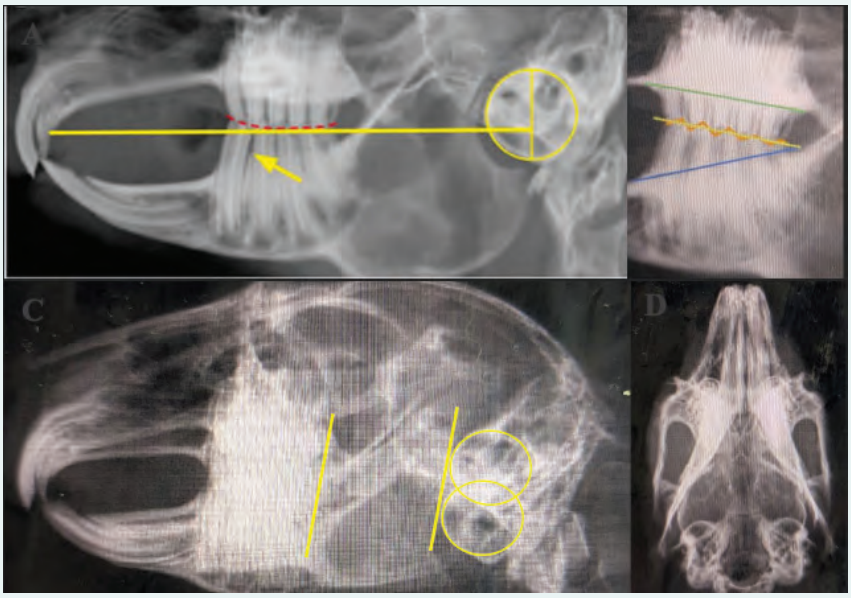
FIGURE 2

A, LL, applied Böhmer and Crossley anatomical reference line shows elongation of the first mandibular premolar causing bending, a widening adjacent space (arrow), and subsequent caudal slant to the maxillary occlusal surface (red dashed line).[1] B, LL, normal cheek teeth with lines showing occlusal surface (orange), common axis (yellow), hard palate (green), and mandible dorsal margin (blue).[5] C, LO, proper alignment of a 10-20 degree rotation showing tympanic bullae overlap (yellow circles) and parallel angle of arcades and bullae (yellow lines), both R and L should be taken.[5,14] D, VD, DV is also acceptable.[3]



Treatment, Prognosis & Setting Expectations

Animals that present in unstable or weakened states should be stabilized and have supportive care prior to addressing their dental disease in more depth. This often will involve subcutaneous fluids (60 to 100mL/kg/day split over BID to TID administration, depending on potential heart disease and level of dehydration), analgesia (buprenorphine or hydromorphone SQ in hospital, meloxicam 1mg/kg q24, gabapentin 5-15mg/kg PO q8-12h, tramadol 10mg/kg PO q8-12h if needed at home), and syringe feeding with a critical care herbivore diet at 50mL/kg/day split over TID administration. It is always preferred to have a CBC and Chemistry Panel performed prior to sedation, anesthesia, and NSAID use to assess systemic health. Owners should be informed that anesthesia can carry a 1.39% to 4.8% chance of anesthetic death if systemically healthy vs. sick, respectively, compared to 0.1% to 0.2% in dogs/cats, but is necessary for chronic management of dental disease.[10]

Management, Not a Cure

It is imperative to provide clear, compassionate, and practical information that emphasizes the chronic nature of the condition and the necessity of ongoing care with frequent follow-up. Owners must understand that once disease is present, medical care is entirely about management, not a cure. There are several ways to manage this process, depending on the severity, extent, location of the disease, and owner factors, including finances, willingness to accept risk, and compliance with home care.

Explaining the dental anatomy of rabbits and how the continuous growth of their teeth can initiate a progressive cycle of malocclusion helps enable owners to understand why ongoing management is so important. It must be discussed that often numerous arcades are affected, malocclusion is progressive, and the start or relapse of infection is not uncommon. Note that extraction and debridement may be required to treat infection, but can result in chronic malocclusion, which needs to be assessed with regular dental trimmings. Rechecks with an awake intraoral exam at minimum, and imaging ideally, are recommended every four to six months for life. Management is a lifelong process that requires regular rechecks, good compliance with at-home care and monitoring, and often a significant financial investment, regardless of the chosen management method. Potential exceptions may be purely palliative care or management of acute, mild, and quickly treated cases of dental disease.

Proper Diet

A proper diet and continuous chewing are essential for maintaining rabbit teeth; disease will progress if the diet is not suitable. Hay should always be available and continuously eaten. Timothy hay is the ideal roughage; however, some rabbits will not eat this, especially if they are already experiencing dental pain. Softer hays, such as orchard and botanical, can also be offered. Alfalfa hay should never be offered; it is high in protein and calcium and can lead to obesity and calciuria or urinary stones. Pellets should be limited to 1/8 cup once or twice daily and consist of Timothy hay with no added seeds or fruits. Rabbits with ad lib pellets often underconsume hay, leading to dental overgrowth due to a lack of wear. Toys and rabbit-safe sticks are good forms of enrichment, but they will not help manage dental disease.

Dental Trimming

It is important to address the need for deep sedation or anesthesia for trims, as well as owner concerns about cost, time, and safety. It is essential to note that rabbits have a narrow mouth opening and are often resistant to oral examinations. With an unsedated exam, it is impossible to assess dental disease fully, let alone properly treat it. The jaw must be fully relaxed to determine disease severity, ensure proper occlusion after correction, and decrease the risk of severe trauma and stress. Furthermore, incorrect trimming of teeth can cause worsened malocclusion, and an occlusal angle of 10 degrees must be maintained to prevent further progression of the disease. Finally, improper tools risk fractured teeth and gum trauma. Incisor overgrowth or malocclusion can often be more easily corrected with the use of a Dremel diamond wheel and a tongue depressor used as a tissue guard, either while awake or under light sedation. However, cheek tooth elongation, points, and malocclusion require general anesthesia and special equipment for proper grinding and safety.

Odontogenic Abscesses

Inform the owner that there are several options for treating abscesses, and that these cases can have very challenging long-term management needs, often requiring frequent procedures and imaging. The importance of proper antibiotic use, pain control, imaging, and nutritional support should be emphasized. It is also important to discuss with owners the unique caseous nature of rabbit pus and that lancing and draining are not an effective or cost-saving treatment. Instead, anesthetized procedures to fully debride abscesses and perform antibiotic packing, as well as marsupialization with or without flushing of the site at home, may be necessary to resolve abscesses. Additionally, potential tooth extraction and orofacial surgery may be required to resolve the abscesses.[14, 7] Rabbits treated solely with systemic antibiotic therapy have about a 25% to 31% abscess resolution rate, depending on whether antibiotic selection is empiric or guided by culture, respectively, while

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surgical intervention can result in a 90% to 93% rate of resolution.[7] However, owners must be informed that resolution relies heavily on owner compliance with medications, wound care, and diet at home, as well as regular follow-up exams, imaging, and potentially procedures. The rate of recurrence for odontogenic abscesses is 8% to 16%, and the disease-free time after resolution ranges from four to sixty months, with a mean of 29 months.[6]

When facing the need for antibiotic use, remember that penicillins, licosamides, macrolides, and cephalosporins are toxic when given orally to rabbits. Culture is always recommended. The best samples to submit include a piece of an abscess capsule or infected tooth or bone; thus, obtaining a sample may not even be possible until a procedure is performed. When empiric antibiotic use is necessary prior to debridement, or when palliative care only is elected and culture is declined or sampling is not possible, consider antibiotic stewardship, bone penetration, and common bacteria associated with these abscesses. Odontogenic abscess flora is commonly a mixture of gram-positive and gram-negative aerobes and anaerobes.[7] Trimethoprim sulfa 25-30mg/kg PO q12h is a recommended first-line broad-spectrum antibiotic to prescribe prior to the return of culture results, which has been shown to be effective against many common bacterial isolates from these abscesses.14 Fluoroquinolones, azithromycin, and beta-lactams have good bone penetration; however, fluoroquinolones are second-tier drugs and should be avoided when possible for empirical use. Azithromycin is slightly more likely to cause gastrointestinal upset and is less broad-spectrum; beta-lactams cannot be given orally. Penicillin Procaine G at a dose of ~65,000 IU/kg SQ q24-48h is a suitable first-line antibiotic with bone penetration, effective against many common odontogenic bacteria. However, this treatment requires consistent administration by the owners and requires careful cleaning of any spilled drug immediately to prevent accidental ingestion. Chloramphenicol is an effective second-line oral alternative, but the risk of aplastic anemia to the owner must be discussed. Metronidazole 20mg/kg PO q12h is an appropriate and effective choice for coverage of anaerobes.

Take Home Points

When preparing to refer a rabbit for dental disease management, it is essential to clarify that dental trims are not a one-time fix. Proper diet and compliance with recommended home care are crucial, and treatment plans can change drastically based on the severity and progression of the disease, so regular follow-up is required. The frequency of trims, medications, and potential other interventions depends on the specific nature of the dental disease and the rechecks, which may require imaging, sedation, or anesthesia, as well as revision procedures. Before treating or referring, it is essential to set clear expectations about what managing dental disease entails, including the time, financial investment, and prognosis, based on the individual patient's case as well as a comprehensive understanding of rabbit dental disease. Reinforce that early intervention can dramatically improve quality of life and overall prognosis.

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↘ Angell Fall 2025 Continuing Education

Register at angell.org/ce

Sunday, October 5, 2025
8:15am – 2:45pm
Live Webinar
5 CE Credits *(RACE approved)*

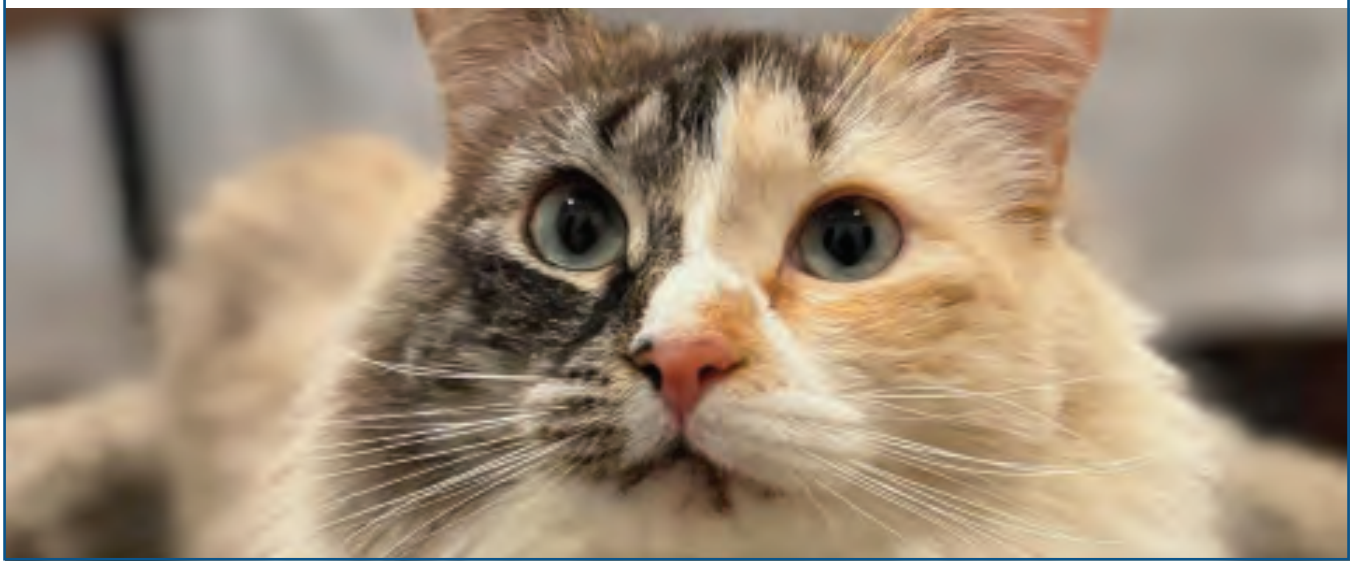
Speakers:

- **Top 5 Retinal Conditions Seen at Angell in Practice and How They Were Addressed –** *Daniel Biros, DVM, DACVO*
- **How & When Veterinarians Should Refer to Behavior vs Trainer –** *Terri Bright, Ph.D., BCBA-D, CAAB*
- **Using Radiation to Mend a Broken Heart –** *Kristine Burgess, DVM, MLA, DACVIM (Medical Oncology)*
- **Management of Degenerative Valve Disease in the General Practice –** *Terry Huh, DVM, DACVIM (Cardiology)*
- **Acute GI Imaging, with a Focus on Radiographs –** *Alexander Quilty, DVM, DACVR*

Wednesday, October 15, 2025
6:15pm – 8:45pm
Live Webinar
2 CE Credits *(RACE approved)*

Speakers:

- **Feline Chronic Gingivostomatitis –** *Shawna Han, DVM (Practice Limited to Dentistry)*
- **A Review of Canine Oral Masses from Angell –** *Pamela Mouser, DVM, MS, DACVP*



STAFF DOCTORS AND RESIDENTS

■ We encourage you to contact Angell's specialists with questions.

Main Phone: 617-522-7282 (Boston) | Main Phone: 781-902-8400 (Waltham) | Veterinary Referrals: 617-522-5011
Angell at Essex: 978-304-4648

CHIEF OF STAFF

Ann Marie Greenleaf, DVM, DACVECC
agreenleaf@angell.org

24-HOUR EMERGENCY & CRITICAL CARE MEDICINE, BOSTON

Alison Allukian, DVM
aallukian@angell.org

Matthew Araujo, DVM
maraujo@angell.org

Kiko Bracker, DVM, DACVECC
Service Director
kbracker@angell.org

David Carabetta, DVM, DACVECC
dcarabetta@angell.org

Zachary Carlson-Sypek, DVM
zcarlsonsypek@angell.org

Callie Cazlan (Verschoor), DVM
ccazlan@angell.org

Elton Chan, DVM
echan@angell.org

Nicole D'Addezio, VMD
ndaddezio@angell.org

Michael Gay, DVM
mgay@angell.org

Ashley Goldacker, DVM
agoldacker@angell.org

Molly Graham, DVM, DACVECC
mgraham@angell.org

Alanna Horton, DVM
ahorton@angell.org

Audrey Koid, DVM, DACVECC
akoid@angell.org

Katherine McKean, DMV
kmckean@angell.org

William Orrico, DVM
worrico@angell.org

Nathalie Suciu, VMD
nsuciu@angell.org

Megan Steinhilber, DVM
msteinhilber@angell.org

Sam Vitali, DVM
svitali@angell.org

Megan Whelan, DVM, DACVECC, CVA
Chief Medical Officer
mwhelan@angell.org

24-HOUR EMERGENCY & CRITICAL CARE MEDICINE, WALTHAM

Jordana Fetto, DVM
jfetto@angell.org

Hilary Kinney, DVM
hkinney@angell.org

Ashley Lockwood, DVM, DACVECC
alockwood@angell.org

Aiden Masri, DVM
amasri@angell.org

Thamires Nunes, DVM
tnunes@angell.org

Courtney Peck, DVM, DACVECC
Chief Medical Officer, Waltham
cpeck@angell.org

Abbey Petronzio, DVM
apetronzio@angell.org

Jessica Seid, DVM
jseid@angell.org

Catherine Sumner, DVM, DACVECC
Chief of Staff, Waltham
csumner@angell.org

Elvira Zaripova, DVM
ezaripova@angell.org

ANESTHESIOLOGY

Stephanie Krein, DVM, DACVAA
skrein@angell.org

Becca Reader, BA, DVM, DACVAA
rreader@angell.org

Declan Ryan, DVM
dryan@angell.org

Kelly Sullivan, PhD, DVM, DACVAA
ksullivan@angell.org

AVIAN & EXOTIC MEDICINE (W/B)

Lauren Gawel, DVM
(Waltham)
lgawel@angell.org

Brendan Noonan, DVM, DABVP (Avian Practice)
bnoonan@angell.org

Anne Staudenmaier, VMD, DABVP (Avian Practice)
(Waltham)
astaudenmaier@angell.org

Patrick Sullivan, DVM, DABVP (Avian Practice)
psullivan@angell.org

BEHAVIOR (W/B)

Terri Bright, PhD, BCBA-D, CAAB
tbright@angell.org

Allyson Salzer, PhD, BCBA-D, CAAB
asalzer@angell.org

Bridgette Wilson, DVM
bwilson@angell.org

CARDIOLOGY

Alice Chirn, MA, BVetMed
achirn@angell.org

Katie Hogan, DVM, DACVIM (Cardiology)
(Boston and Waltham)
khogan@angell.org

Terry Huh, DVM, DACVIM (Cardiology)
thuh@angell.org

Barbara Linnehan, DVM, DACZM
blinnehan@angell.org

Elizabeth Malcolm, DVM, DACVIM (Cardiology)
emalcolm@angell.org

Sarah Rogg, DVM
srogg@angell.org

DENTISTRY

Shawna Han, DVM
shan@angell.org

Jessica Riehl, DVM, DAVDC
jriehl@angell.org

Joyce Tai, DVM/MS, DAVDC
jtai@angell.org

DERMATOLOGY (W/B)

Meagan Painter, DVM, DACVD
(Waltham)
mpainter@angell.org

Brooke Simon, DVM
bsimon@angell.org

DIAGNOSTIC IMAGING (W/B)

Naomi Ford, DVM, DACVR
nford@angell.org

Alex Quilty, DVM, DACVR
aquilty@angell.org

Steven Tsai, DVM, DACVR
stsai@angell.org

Sarah Slaughter, DVM
slaughter@angell.org

Ruth Van Hatten, DVM, DACVR
rvanhatten@angell.org

INTERNAL MEDICINE (W/B)

Nyla Bent, DVM
nbent@angell.org

Douglas Brum, DVM
dbrum@angell.org

Maureen Carroll, DVM, DACVIM
mccarroll@angell.org

Anita Fothergill, VMD
afothergill@angell.org

Lisa Gorman, DVM, DACVIM
(Waltham)
lgorman@angell.org

Antonia Ioannou, BVMS, DACVIM
aioannou@angell.org

Shawn Kearns, DVM, DACVIM
skearns@angell.org

Evan Mariotti, DVM, DACVIM
emariotti@angell.org

Susan O'Bell, DVM, MPH, DACVIM (Internal Medicine)
Service Director
sobell@angell.org

Juliana Picard, DVM
jpicard@angell.org

Ursula Ramalho, DVM
uramalho@angell.org

Vanessa Rozo, DVM
vrozo@angell.org

Annie Sheu-Lee, DVM, DACVIM
asheulee@angell.org

Lorena Sistig, DVM
lsistig@angell.org

STAFF DOCTORS AND RESIDENTS

CONTINUED FROM PAGE 22

Daniela Vrabelova Ackley DVM, MS, DACVIM
(Waltham)
dvrabelova@angell.org

Yvonne Wei, DVM
ywei@angell.org

NEUROLOGY

Rob Daniel, DVM, DACVIM (Neurology)
rdaniel@angell.org

Michele James, DVM, DACVIM (Neurology)
Service Director
mjames@angell.org

Jennifer Michaels, DVM, DACVIM (Neurology)
jmichaels@angell.org

ONCOLOGY

Sarah Adrianowycz, DVM, DACVIM (Medical Oncology)
sadrianowicz@angell.org

Kristine Burgess, DVM, MLA, DACVIM (Medical Oncology)
kburgess@angell.org

Megan Cray, VMD, DACVS (Surgical Oncology)
mcray@angell.org

Gretchen McLinden, DVM, DACVIM (Medical Oncology)
gmclinden@angell.org

Jillian Walz, DVM, DACVIM (Medical Oncology) , DACVR (Radiation Oncology)
jwalz@angell.org

OPHTHALMOLOGY

Daniel Biros, DVM, DACVO
dbiros@angell.org

Martin Coster, DVM, MS, DACVO
mcoster@angell.org

PATHOLOGY (CLINICAL & ANATOMIC)*

Patty Ewing, DVM, MS, DACVP
pewing@angell.org

Pamela Mouser, DVM, MS, DACVP
pmouser@angell.org

SURGERY (W/B)

Kristen Behrens, DVM
kbehrens@angell.org

Matthew Boules, DVM
mboules@angell.org

Sue Casale, DVM, DACVS
scasale@angell.org

Hadley Gleason, VMD, MS, DACVS
(Waltham)
hgleason@angell.org

Dilraj Goraya, DVM
dgoraya@angell.org

Michael Pavletic, DVM, DACVS
Service Director
mpavletic@angell.org

Jennifer Peterson-Levitt DVM, MS, DACVS-SA
(Boston & Waltham)
jpetersonlevitt@angell.org

Sophia Topulos, DVM, DACVS
(Waltham)
stopulos@angell.org

Nicholas Trout MA, VET MB, DACVS, ECVS
ntrout@angell.org

URGENT CARE BY APPOINTMENT ONLY, WALTHAM

Tamara Kremer Mecabell, DVM
tmecabell@angell.org

Natasha Pakravan, DVM
npakravan@angell.org

Julie Shields, DVM
jshields@angell.org

ANGELL AT ESSEX

Heidi Broadley, DVM
hbroadley@angell.org

Sara Gardiner, DVM
sgardiner@angell.org

(W/B) Services available at both our Waltham and Boston locations
*Boston-based pathologists and radiologists serve both Boston and Waltham locations

↘ Courtesy Shuttle for Patients Needing Further Specialized Care

Angell Animal Medical Center offers the convenience of our MSPCA-Angell West facility in Waltham, MA. The Waltham facility offers Urgent Care and specialized service appointments. If needed, an oxygen-equipped courtesy shuttle can transport animals to Boston for further specialized care and then return them to Waltham. Whether in Boston or in Waltham, our specialists regularly collaborate and plan treatments tailored to our patients' emergency, surgical, and specialty needs.

WE OFFER A BROAD RANGE OF EXPERTISE AND DELIVER THIS CARE WITH THE ONE-ON-ONE COMPASSION THAT OUR CLIENTS AND PATIENTS DESERVE.

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Our Service Locations

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I-131 Therapy
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617-541-5014

*Available only in Waltham

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