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Hemangiosarcoma and Heart Base Tumors

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Introduction

Canine cardiac tumors are most often divided into two broad categories: right atrial hemangiosarcoma (HSA) and heart base tumors (HBTs), the latter of which are most commonly chemodectomas. Both are clinically significant, but they differ markedly in their biology, clinical presentation, and patient outcomes. In both settings, radiation therapy is becoming an increasingly valuable tool, not only for cytoreduction, but also for its hemostatic properties.

Right Atrial Hemangiosarcoma

Right atrial hemangiosarcoma is the most common primary cardiac tumor in dogs. It typically arises in the right atrium or auricular appendage, and it may appear either as a discrete nodule or a more infiltrative lesion. Clinical signs vary depending on stage. Early in the course, dogs may only show vague lethargy, weakness, or coughing. As the tumor progresses, however, more dramatic presentations are common, including collapse, dyspnea, and acute hemorrhagic pericardial effusion.

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Neurology and Pharmaceuticals: Debunking Common Misconceptions

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There are many common misconceptions regarding the use of certain pharmaceuticals in neurological patients, which result in the use of some medications that should not be used or the refusal to use medications that are actually safe. This could potentially be detrimental to patients. This article aims to highlight some of these common misconceptions, review where the misinformation

originated from, and clarify what the recent data actually report.

Ketamine and Intracranial Pressure

Myth: Ketamine causes an increase in intracranial pressure (ICP) and, therefore, should not be used in patients with intracranial disease.

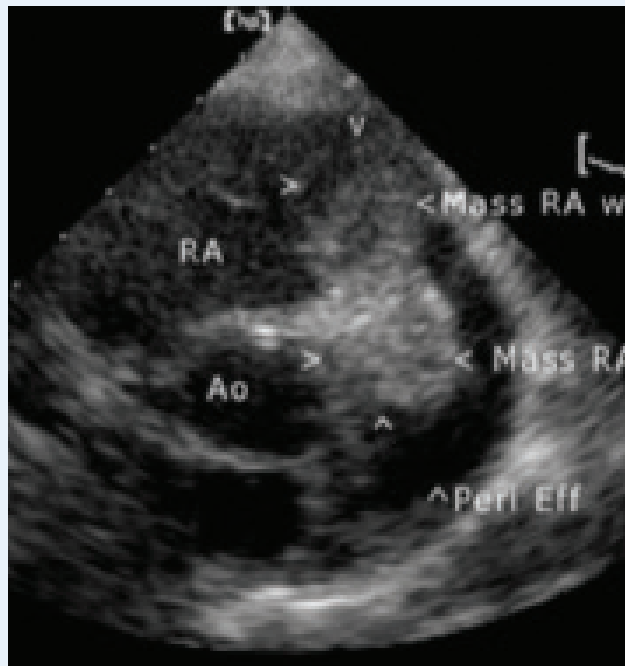
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› Echocardiogram of patient with hemangiosarcoma



In many cases, tamponade and cardiogenic shock are the events that bring patients to veterinary attention.

Diagnosis relies heavily on echocardiography, which is both sensitive and specific for identifying these lesions. Pericardial fluid cytology is rarely diagnostic, with the exception of cases of cardiac lymphoma (extremely rare).

Unfortunately, the prognosis with traditional therapies is very poor. Without intervention, survival may be measured in days. Pericardiectomy can extend survival to two to three months, and the combination of surgery with chemotherapy offers a median survival of four to six months. Chemotherapy alone after resection may increase survival to approximately five or six months, but in all cases, metastatic spread and recurrent bleeding remain major obstacles.

Radiation therapy is being used more frequently to address these limitations. The rationale is twofold: to reduce tumor bulk and stabilize fragile vasculature to decrease bleeding. A pilot study using a single 12-Gy fraction demonstrated significant reduction in the need for pericardiocentesis, with a median survival of about 79 days. More advanced protocols, such as hypofractionated IMRT in combination with vinblastine and propranolol, achieved resolution of pericardial effusions in all treated dogs, with responses ranging from complete remission to stable disease and a median survival of 326 days. Larger retrospective series show similar findings, with pericardiocentesis frequency dropping by over 80% and a median survival of 137 days. Importantly, when CT guidance is used, adverse events are less frequent. Taken together, these studies suggest that radiation, especially when combined with systemic therapy, can extend survival into the many-month to nearly-one-year range, while improving patient stability and comfort.

› Radiograph of patient with heart base tumor



Heart Base Tumors (HBTs)

While hemangiosarcoma is aggressive, fast-growing, and highly metastatic, HBTs behave very differently. Chemodectomas, the most common HBTs, tend to be slow-growing, more indolent, and often discovered incidentally. The clinical presentations also differ: Hemangiosarcoma usually presents with acute collapse and tamponade, whereas HBTs are more likely to cause chronic signs from mechanical compression, such as coughing, dyspnea, exercise intolerance, or syncope. These biological differences also shift the therapeutic goals. Hemangiosarcoma demands crisis stabilization and palliation of bleeding, while HBTs are better suited to long-term local control strategies.

Chemodectoma

HBTs are typically located at the root of the aorta and pulmonary arteries, near the carotid bifurcation and jugular veins. They are usually encapsulated, homogeneous, space-occupying masses. Clinical signs reflect compression of adjacent structures and include coughing, respiratory distress, or exercise intolerance. Metastasis is reported but occurs in only about 20% of cases.

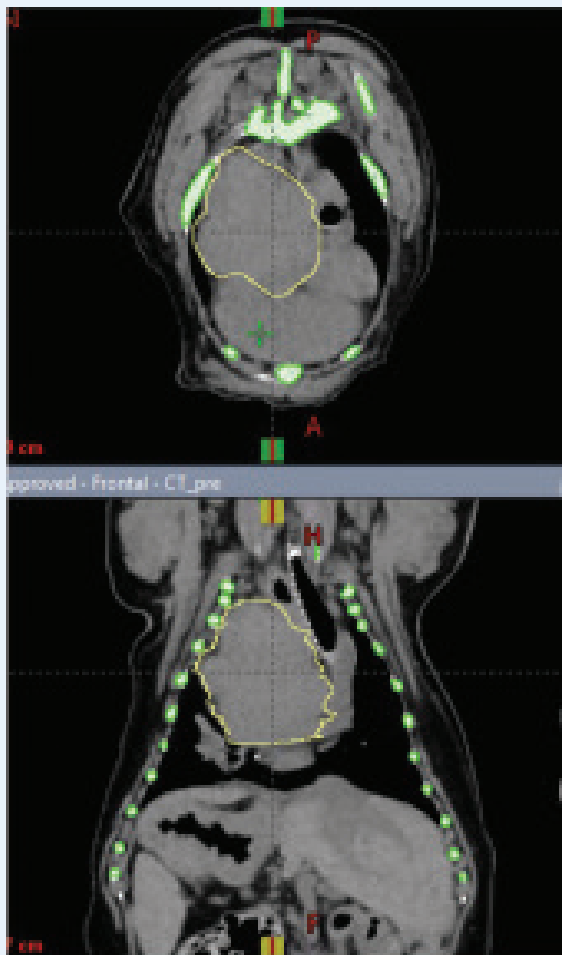
Surgical removal is often not feasible due to the tumor's location, so radiation therapy is the treatment of choice. Both stereotactic body radiation therapy (SBRT) and conventional fractionated radiation therapy (CFRT) have shown excellent results. In a study using SBRT, the median survival was 404 days, with most dogs achieving either partial response or stable disease. In another series comparing SBRT and CFRT, overall median survival was 785 days. Dogs treated with CFRT lived a median of 817 days, while those receiving SBRT had a shorter but still meaningful median survival of about 414 days. Side effects are generally mild, though arrhythmias, congestive heart failure, or sudden death have been reported in rare cases. Overall, radiation is well tolerated and provides durable tumor control in this population.

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× CT scans of patient with chemodectoma



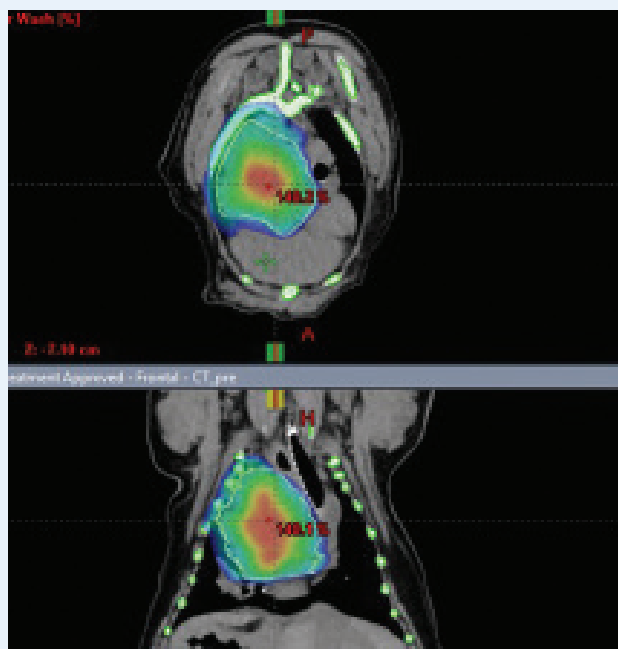
Hemostatic Role of Radiation

Beyond tumor shrinkage, radiation has important hemostatic effects. By inducing fibrosis, promoting platelet adhesion, and creating pro-thrombotic changes in tumor vasculature, it can reduce or even stop hemorrhage. In human oncology, palliative radiation is widely used to manage bleeding in gastrointestinal and other cancers. This same principle is increasingly recognized in veterinary oncology, especially in the management of cardiac hemangiosarcoma, where stabilization of vascular integrity is just as important as tumor cytorreduction.

Conclusion

Right atrial hemangiosarcoma remains one of the most aggressive cardiac tumors, with a guarded to poor prognosis. However, radiation therapy, particularly when combined with chemotherapy, can extend survival significantly and improve quality of life by reducing bleeding and stabilizing patients. Heart base tumors, in contrast, are much more indolent, and radiation therapy provides excellent long-term local control, often extending survival beyond a year. In both contexts, radiation plays a dual role — tumor cytorreduction and hemostasis — that is central to management.

× CT scans revealing tumor regression in patient with chemodectoma, six months after completing a course of stereotactic radiation therapy



Optimal outcomes require a multidisciplinary approach, bringing together cardiology, oncology, and advanced imaging to deliver tailored, evidence-based care for these challenging but increasingly manageable diseases.

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Ophthalmology	3 weeks	N/A
Surgery	6 days: TPLO consult 3 weeks: Consult for soft tissue surgery	3 weeks: Orthopedic consults (including TPLO) 2 1/2 weeks: Consult for soft tissue surgery
Urgent Care (Mon.-Fri.)	N/A	Same-day appointments

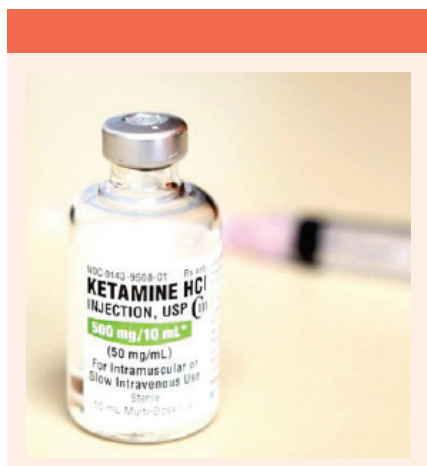


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Background: Ketamine is an NMDA receptor (NMDAR) antagonist. The NMDAR are the most ubiquitous excitatory receptors in the central nervous system (CNS). When glutamate, the NMDAR ligand, is released from the pre-synaptic nerve terminal, it binds to the NMDAR, resulting in an influx of calcium ions into the post-synaptic neuron, thus triggering an action potential and many other intracellular processes. Traditional uses of ketamine include sedation, anesthesia, and pain management. The benefit of ketamine over other sedatives/anesthetic drugs is its relative lack of hemodynamic and respiratory depression. For pain management, the beneficial effects of ketamine far outlast the actual drug levels in the system.

Origin of the Myth: A collection of approximately six case reports and case cohorts from the 1970s. These publications described increasing ICP in patients with intracranial disease receiving ketamine.

Problems with the original data: The reports were all low-patient volume, non-controlled, descriptive papers, including patients with abnormal CSF pathways (e.g., patients with hydrocephalus). In addition, given the time at which these papers were published, the ICP monitoring techniques are very outdated, and there was no assessment of the clinical impact of the documented ICP changes.

New Data: A plethora of more recent data, including controlled studies, systematic reviews, and meta-analyses, document the safety of ketamine in patients with intracranial disease.

- Most of these studies document no impact on ICP
- A few studies even show a decrease in ICP
- The few studies that show an increase in ICP associated with ketamine administration fail to demonstrate any negative clinical impact

In addition, many of these studies have also documented potentially beneficial effects of ketamine, including an increase in cerebral perfusion pressure and mean arterial pressure, which are essential for maintaining cerebral blood flow. The use of ketamine in patients with intracranial disease also allowed for a reduction in the requirement of other medications, including other sedatives, vasopressors, and pain medications such as opioids.

Not only is this a misconception, but withholding ketamine in this subset of patients potentially results in missing out on some particular benefits of ketamine that could be useful for patients with intracranial disease. Modern uses of ketamine, including its use for the management of chronic pain, depression, seizures, and migraines, are important to acknowledge. There is also promising research regarding the neuro-protective effect of ketamine in post-trauma or post-stroke patients, given its action against NMDAR, the activation of which can lead to neuronal excitotoxicity and neuronal necrosis.

Conclusion: Ketamine is overall safe to use in patients with neurological disease without fear of significantly increasing ICP. In addition, there are many mechanisms and secondary uses for ketamine that may make it beneficial in neurological patients.

Acepromazine and the Seizure Patient

Myth: Acepromazine causes seizures and, therefore, should not be given to any patient with a history of seizures or one who is at risk for seizures.

Background: Acepromazine is one of the phenothiazine medications, a group which also includes chlorpromazine. Phenothiazines are neuroleptic drugs that suppress spontaneous movements and complex behavior while leaving spinal reflexes and unconditioned behaviors intact. Acepromazine is commonly used to reduce anxiety and for sedation/anesthesia.

Origin of the Myth: The potential risk of seizures with phenothiazines was originally documented in human medicine in the 1950s. The first veterinary paper documenting a connection between phenothiazines and seizures was published in the 1970s. The purpose of this study was to describe clinical and EEG observations in dogs with seizures and to relate these findings to human epileptic conditions. The researchers rapidly administered high doses of chlorpromazine and monitored EEG. One-half of dogs had EEG changes consistent with epileptiform activity, and 5% had clinical signs



consistent with a seizure. This study formed the basis of the concern surrounding acepromazine and seizures.

Problems with the original data: The study used chlorpromazine, which is known to be more epileptogenic than acepromazine in people. The dogs were also administered 5 to 50 times the recommended dose of chlorpromazine; the risk of seizures with phenothiazines in people is dose-dependent. Lastly, this effect was seen in an experimental setting where the authors intentionally tried to elicit seizure activity.

New Data: In the last 20 years, three studies have been published documenting the safety of acepromazine in seizure patients.

1. Two retrospective studies evaluated the use of acepromazine for sedation or as part of an anesthesia protocol in patients with a history of seizures. There was no observed correlation between ACE administration in dogs with a seizure history and the recurrence of seizure activity during hospitalization.
2. A retrospective study comparing the incidence of seizures in patients undergoing myelography with one group receiving acepromazine as part of the anesthetic protocol and one group not receiving acepromazine. There was no reported difference in seizure incidence between groups.

Conclusion: The lack of correlation between the original data regarding chlorpromazine and seizures and the clinical use of acepromazine, as well as the newer data documenting no increased incidence of seizures in at-risk patients receiving acepromazine, suggest that acepromazine is safe to use in patients with a history of seizures.

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Potassium Bromide (KBr) and Pancreatitis

Myth: KBr causes pancreatitis. If a patient on KBr develops pancreatitis, the medication must be discontinued.

Background: KBr is an anti-seizure medication primarily used as an adjunct therapy in refractory patients. A halide salt increases negative ion transmission across the neuronal cell membranes, resulting in hyperpolarization and reduced membrane excitability. This results in an increase in the seizure threshold and stabilization of the neurons against excitatory inputs.

Origin of the Myth: The first mention of a connection between bromide and pancreatitis was in the early 1990s in two papers evaluating the use of KBr in epileptic dogs. These studies mentioned a few dogs that developed pancreatitis after initiating KBr therapy. Of note, all of the affected dogs were also receiving phenobarbital, and two had a history of dietary indiscretion. A subsequent letter to the editor published in 2000 described a subpopulation of six dogs included in a study evaluating the efficacy of phenobarbital, half of which developed pancreatitis when KBr was added to their treatment protocols.

As a result of these reported cases, several additional studies in the 2000s to 2010s evaluated various laboratory values, including cPLI, C-reactive protein (CRP), amylase, lipase, lipids, and liver values in patients receiving KBr. While bromide-treated dogs did have relatively higher liver values, amylase and lipase values, and triglycerides, only one study showed a difference in cPLI values, and there was no difference in CRP.

Problems with the original data: All prior studies utilized data collected from diagnostic lab submissions to reference laboratories. As such, there was limited to no correlation to clinical disease or clinical status of the patients. In addition, by including only those patients with diagnostic lab submissions, there is an inherent bias toward sick patients. Second, there was no assessment of any lab values or clinical history before initiating KBr therapy. In addition, a vast majority of the patients with abnormal laboratory values were not receiving KBr alone; rather, they were receiving polytherapy, including phenobarbital. Lastly, there was no assessment of other factors that could potentially predispose patients to pancreatitis, including diet, dietary indiscretion, breed predispositions, or comorbidities.

New Data: In 2012, a systematic review evaluating the safety of KBr in dogs reported that the association between GI side effects, including pancreatitis, and using KBr was one of the least well-supported. The review concluded that there was “not enough evidence to determine whether dogs receiving KBr were at a higher risk of developing pancreatitis.”

Conclusion: Given the paucity of data to support an association between KBr and pancreatitis, it is important not to withhold this highly effective anti-seizure drug from patients who may benefit from its use nor to discontinue this treatment in patients who are currently receiving it. This is especially true given the use of KBr primarily as an adjunct therapy for refractory patients, as they are unlikely to have many other treatment options available for seizure control.

Acute medical management, along with long-term lifestyle and diet changes that would be recommended for any patient with pancreatitis, have been equally effective for patients on KBr without necessitating discontinuation of this therapy.

Psychotropic Medications and the Seizure Patient

Myth: Psychotropic medications cause seizures and, therefore, should not be used in patients with a history of seizures or at risk of seizures.

Background: Psychotropic medications are medications used for the treatment and management of depression and anxiety. In general, these medications work by increasing certain neurotransmitters, including serotonin, norepinephrine, and dopamine. Commonly used psychotropic medications include:

- Selective Serotonin Reuptake Inhibitors (fluoxetine, sertraline)
- Tricyclic Antidepressants (amitriptyline, clomipramine, imipramine)
- Serotonin-Norepinephrine Reuptake Inhibitors (venlafaxine, duloxetine)
- Serotonin Antagonist and Reuptake Inhibitors (trazodone)

Behavioral comorbidities are common in people with epilepsy (PWE). About 1/3 of PWE develop mood or anxiety disorders (MAD), with depression and anxiety being the most common. Cognitive impairment and ADHD are also described. Interestingly, there is a well-documented bidirectional effect between MAD and epilepsy in both adult and pediatric patients. Not only are PWE more likely to develop MAD, but people with MAD are more likely to develop epilepsy. If one condition is poorly controlled, the presence or severity of the other may increase, suggesting a potential common etiology. In addition, anti-seizure drugs can impact behavior with polytherapy linked to more severe MAD.



Behavioral comorbidities are also documented in dogs with epilepsy, with about two-thirds of epileptic dogs showing behavioral changes, including fear, anxiety, cognitive impairment, and ADHD-like behaviors. While medications can impact the severity of behavioral comorbidities, they have been documented in drug-naïve epileptic dogs, thus proving a direct connection between the two conditions. In addition, these comorbidities are more severe in refractory epileptics, suggesting a possible similar connection between epilepsy and behavioral disorders as that which is described in people.

The identification and management of behavioral comorbidities in epileptic patients is critical due to the impact on quality of life. In PWE, psychological comorbidities are significantly associated with poorer health-related quality of life. In fact, the negative impact of MAD on health-related quality of life is often found to be greater than the impact of epilepsy-related factors,

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including seizure frequency and seizure severity. In veterinary medicine, behavioral comorbidities, including reduced trainability, altered reactions to daily situations, and fear-related behavior, have been documented to affect daily life and quality of life for both patients and owners.

Origin of the Myth and Problems with the Data: While both human and veterinary papers report seizures in patients administered various psychotropic medications, many of these in people and all in veterinary medicine are in the setting of misuse, overdose, or toxicity. In addition, clinical trials performed in humans do document increased seizures in people receiving psychotropic medications compared to the general population but fail to show a significant difference compared to people receiving placebos. Therefore, it is likely that the increased seizures noted in this population of patients are more a result of the established connection between MAD and epilepsy rather than an effect of the medications themselves.

New Data: There are numerous studies, systematic reviews, and meta-analyses evaluating the risk of psychotropic medications in seizure patients, the majority of which show no negative effect. In fact, a small number report an improvement in seizure control in patients receiving psychotropic medications.

Although a majority of the commonly used psychotropic medications have been determined to be safe for seizure patients, four medications have been associated with an increased risk of seizures: bupropion, clomipramine, amoxapine, and maprotiline. Only clomipramine is used in veterinary medicine.

Conclusion: In summary, all commonly used psychotropic medications in veterinary medicine, with the exception of clomipramine, are safe to use at therapeutic doses in pets with seizures. In addition, evidence shows that managing primary behavioral disorders or behavioral comorbidities in epileptic patients is important for improvement in the quality of life of both pets and their owners.

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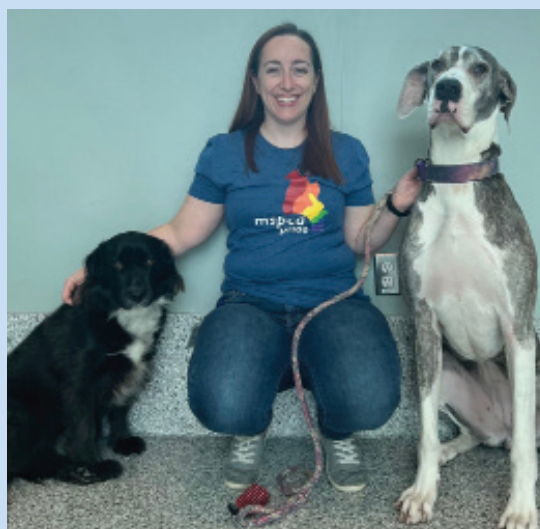
» Grooming and Healthy Boarding Services at Angell

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

These services provide the exceptional care for which Angell is known. Having a veterinary hospital on-site sets us apart and, while the hospital is separate from these new areas, medical support is available if needed. Healthy Boarding is committed to providing canine and feline guests with enrichment, including play group, and personalized attention, while grooming is focused on creating a positive wellness experience for pets and their owners.

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



TLC for AKI and IMHA: A Brief Review of HD, TPE, and HP

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Introduction

Over the past ten years, many advances have been made in our ability to diagnose and treat severely life-threatening illnesses, including acute kidney injury (AKI), immune-mediated hemolytic anemia (IMHA), and toxin exposure. The use of extracorporeal therapies has become a significant player in our ability to successfully treat these illnesses through early intervention and the ability to gain rapid control of ongoing disease processes. While the pathophysiology of AKI and immune-mediated diseases is beyond this article's scope, extracorporeal therapies are becoming more common and accessible for veterinary patients. Clients may ask their family veterinarian whether these modalities could help their pets. This article aims to provide basic information about the modalities of hemodialysis, therapeutic plasma exchange, and hemoperfusion so that you are well-prepared to have these conversations with your clients and feel confident when considering referral of your patients.

Basic Overview of Modalities

The broad mechanism of each of these modalities focuses on “treating” or processing a patient's blood outside the patient's body. Blood is removed from the patient using a large bore jugular catheter, pumped through a filter (or centrifuge) to remove molecules from the blood, and then returned to the patient. We select a process to do this based on the treatment goals. Understanding the basic principles of each process helps decide which modality is ideal for a given patient.

Hemodialysis (HD)

Hemodialysis uses a semipermeable membrane to selectively remove unwanted substances from a patient's blood. In renal failure, the goal is to remove uremic toxins, including creatinine, blood urea nitrogen (BUN), and potassium, which lead to the clinical signs of renal failure. We use hemodialysis to take over this role of the kidneys when they are too injured to do it successfully, in an attempt to reduce the adverse consequences of severe azotemia. In renal failure, many uremic toxins accumulate. However, we tend to use BUN and creatinine as representatives because they are easily measured and monitored. Hopefully, the kidneys will recover from the insult and regain their normal function with time. We can also use dialysis to remove select unwanted toxins, such as ethylene glycol, to limit their detrimental effects on the kidneys.

Hemodialysis uses two main mechanisms to remove these unwanted substances and toxins from the blood: diffusion and convection. Diffusion is the movement of solutes (molecules, toxins) across a semipermeable membrane along a concentration gradient. Blood flows along one side of the semipermeable membrane, while dialysate (which contains no uremic toxins) flows along the other side. Diffusion occurs due to the random movements of small molecules coming into contact with the semipermeable membrane (located inside the dialysis filter or dialyzer) and then moving across the membrane into dialysate. This occurs because, in renal failure, there is a high concentration of uremic toxins in the patient's blood. When we expose



the blood to dialysate, the uremic toxins move down the concentration gradient — out of the blood and into the dialysate, which is then discarded. The blood and fresh dialysate are circulated through the filter multiple times during the treatment, gradually decreasing the level of uremic toxins in the blood. Solute concentration is the main determinant of diffusion.

Convection uses a different method for solute removal. In convection, a positive pressure is exerted across the semipermeable membrane, pushing water and larger solutes into the dialysate, which are then removed. This process can also be used to only remove water, such as in cases where patients are fluid-overloaded (this is called ultrafiltration).

Convection does not rely on a concentration gradient to move solutes across the membrane, so it is more effective in removing larger solutes.

There are two main types of platforms for hemodialysis, based on which process (diffusion or convection) you want to use. Historically, the platforms are intermittent hemodialysis (IHD) and continuous renal replacement therapy (CRRT). IHD primarily uses diffusion, whereas CRRT mainly uses convection (although this platform can also perform some diffusion). No data (human or veterinary) supports that one modality is more beneficial in treating renal failure. A lot of the time, whichever platform is used is determined by whichever machine is available (if you aren't lucky enough to have both types). As the name suggests, IHD is usually a shorter, more aggressive treatment — it allows for more rapid shifts in solutes and fluid, which may be risky in critical patients.

Treatments are usually performed over a four- to six-hour period each day, over the course of days. IHD machines also create their own dialysate, combining processed water (reverse osmosis), acids, and electrolytes.

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Traditionally, CRRT patients are hooked up to the machine, which continuously filters the blood until a goal is reached; these treatments often last 24 to 48 hours. While this platform has higher requirements for staffing and monitoring, it produces more gradual shifts in fluid and solutes, and tends to be safer in critical patients. In veterinary medicine, those programs with a CRRT machine will often do a hybrid treatment where a combination of convection and diffusion is used to increase the efficiency of the treatment and shorten treatment time. CRRT machines require bags of pre-made dialysate instead of the machine making it.

Regardless of the platform used, the workhorse of dialysis is the dialyzer, or filter. Many different types of dialyzers are available, again based on which solute(s) need to be removed and the patient's size. Within the filter are hundreds of tiny tubes, each of which is a semipermeable membrane. The pores in these membranes vary in size but are generally effective in allowing small- to medium-sized solutes to pass through (either through diffusion or convection). Larger molecules, or solutes bound to proteins in the blood, are not effectively removed by dialysis. These membranes keep the blood and the dialysate separate, which creates the concentration gradient during diffusion. The structure of the dialyzers creates a huge surface area, mimicking the kidney and allowing for the efficient removal of solutes and toxins.

Indications for Hemodialysis

As discussed, hemodialysis is effective for removing small- to medium-sized solutes and toxins, which is why it is effective in treating AKI (urea, creatinine, and electrolytes are fairly small). Currently, the most common reason we treat patients with hemodialysis is Leptospirosis; however, other causes of AKI, such as pyelonephritis and toxin exposure, are possible. It used to be thought that patients with ureteral obstruction should be treated with hemodialysis, but this has fallen out of favor, and current recommendations support immediate surgical intervention with subcutaneous ureteral bypass (SUB) placement.

Causes and Progression of AKI

AKI and azotemia can develop due to three main causes: prerenal (such as hypotension, dehydration), intrinsic renal disease (infection, immune-

mediated disease, toxicity), or postrenal (urethral obstruction). It is essential to determine which one is causing renal failure in a given patient, as the treatment for each cause will vary.

There are four identified phases of AKI: initiation, extension, maintenance, and recovery. Initiation is the inciting event (development of infection, exposure to toxin) and usually lasts hours. The extension phase includes sustained injury or exposure to a toxin and can last days to weeks. During the maintenance phase, renal response to and repair from the injury occurs and may last for months. The recovery phase can last for years; if too much damage has occurred and recovery is not achieved, renal death will occur. We do not see an increase in BUN and creatinine on routine blood work until 75% of renal function has been lost. Currently, we tend to identify AKI during the maintenance phase when treatment addresses the systemic effects of renal failure and is less effective in definitive healing. Ideally, we would identify AKI during the initiation or extension phases, when intervention could play a more significant role in preventing ongoing and further renal damage. This is why there is so much investigation into renal biomarkers, such as symmetric dimethylarginine (SDMA), which may be more sensitive in identifying early AKI. More recent evidence suggests that approximately half of patients who sustain a significant AKI will develop chronic kidney disease at some point during their life. Being able to identify a kidney injury early, allowing earlier intervention and prevention of further injury, may reduce the risk of CKD in some patients.

Hemodialysis is recommended in patients with AKI who have developed severe and/or progressive azotemia, despite adequate fluid resuscitation. Additional criteria include severe hyperkalemia or acid-base disturbances, fluid overload, and oliguria/anuria. Ideally, hemodialysis would be initiated before these life-threatening sequelae develop in our patients.



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**Fluids and AKI**

The kidneys typically receive approximately one-quarter of cardiac output and normally exist in a relatively low-oxygen state. This makes them extremely sensitive to decreases in perfusion and oxygen delivery and poorly capable of dealing with hypoxemia. While fluid therapy can effectively improve perfusion and oxygen delivery to the kidneys, it requires a careful balance because too much fluid volume can negatively impact renal function. When interstitial edema develops, it increases the distance that oxygen must diffuse to reach the renal cells, promoting renal hypoxia.

Additionally, the kidneys are enclosed in a capsule that cannot expand. As renal interstitial edema develops, it also creates increased pressure within the kidney, leading to damage to the renal cells and decreasing renal blood flow. There has been a demonstrated association in human medicine between fluid overload and worsening patient outcomes, and it is reasonable to assume the same holds true for our patients. In a recent study, approximately 70% of veterinary patients presenting for hemodialysis were significantly fluid-overloaded. The historical belief that more fluids are better for AKI has led to a new movement of mindful fluid therapy stewardship.

Total Plasma Exchange (TPE)

In TPE, patient blood is separated from the plasma to remove larger and/or protein-bound molecules or toxins. This can be done either by a filter that uses a membrane or via centrifugation. If the separation occurs via a filter, the patient plasma is removed (including the substance we want to remove) and discarded; donor plasma is combined with the rest of the patient's blood components before returning it to the patient. Alternately, the components in a patient's plasma can be removed via centrifugation, and the patient's plasma is then returned to the patient. This process is called plasmapheresis. The modality chosen depends on the machine available. This discussion will focus on TPE; however, both modalities are primarily used to remove larger and/or highly protein-bound substances from the blood. In veterinary medicine, TPE is used mainly to remove antibodies in immune-mediated diseases and specific toxins.

During total plasma exchange, a treatment is based on processing a certain volume of the patient's plasma. Research has found that treating one-and-a-half plasma volumes (based on patient size and packed cell volume, or PCV) will reduce the substance by approximately 85%, and this is what most treatment protocols are based on. For immune-mediated diseases, current treatment guidelines recommend daily treatment for three days in a row,

followed by a rest day and subsequent treatments, as indicated by the patient's clinical progression. One treatment may be sufficient for the removal of a toxin.

Currently, the main indications for TPE in veterinary patients include immune-mediated diseases and exogenous toxicities. Immune-mediated diseases result from auto-antibodies against a particular cell type or receptor. These antibodies circulate in the plasma before binding to a specific cell or receptor. By removing patient plasma, TPE effectively also removes circulating auto-antibodies, essentially taking immediate control of the disease process in the hopes of allowing standard immunosuppressive therapy to take action. The use of TPE in treating IMHA has been most commonly reported in veterinary literature, with a landmark case series reported in 2019 by Cowgill et al.

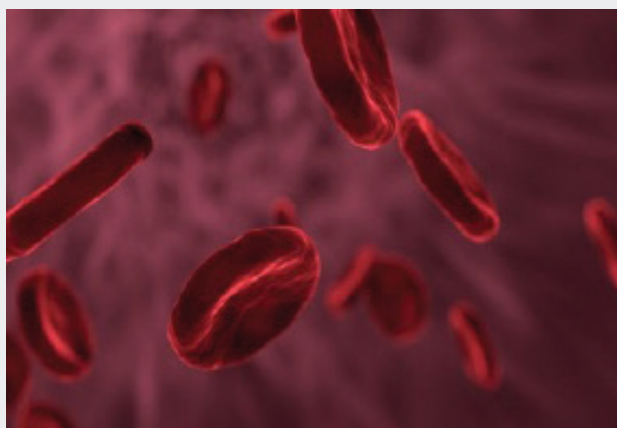
This paper showed a significant improvement in survival in patients who received TPE compared to traditional medical management alone. TPE does not replace the current recommended standard of treatment with immunosuppressive therapy; it aims to rapidly control the disease to allow those therapies to take effect. TPE has also been evaluated in treating immune-mediated thrombocytopenia, fulminant myasthenia gravis syndrome, and rattlesnake envenomation in veterinary patients.

Hemoperfusion

Hemoperfusion is another modality becoming more popular, particularly when treating exogenous toxicities in veterinary patients. This modality uses the process of adsorption (not to be confused with absorption), where molecules or solutes bind to a medium when exposed to it. Instead of a filter, the patient's blood is passed through a cartridge containing a substance such as charcoal, carbon, or polymer. Adsorption is non-specific for a particular substance, and its efficacy is not determined by molecular size or degree of protein binding. Hemoperfusion also binds molecules that we do not necessarily want to remove from the blood, such as glucose, albumin, electrolytes, and platelets. To counteract this, blood passing through a hemoperfusion cartridge is often passed through a dialyzer to re-equilibrate electrolytes and glucose before returning the blood to the patient.

Toxicities

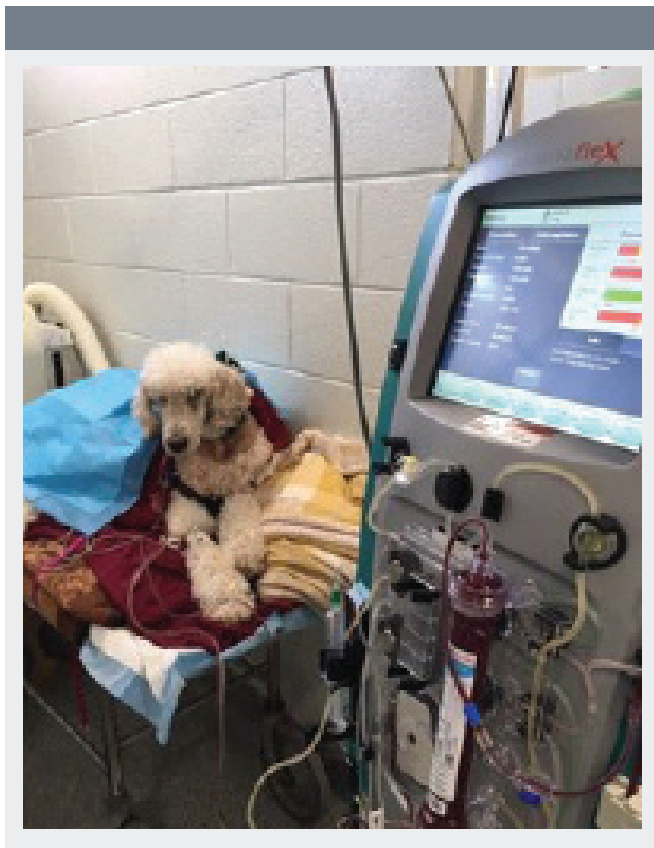
Based on recent ASPCA poison control reports, the most common types of exogenous toxins that veterinary patients are exposed to include ibuprofen, human antidepressants, human cardiac medications and grapes/raisins. Immediate intervention to decontaminate is recommended, but in cases



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where the overdose is significant, extracorporeal therapy may improve patient outcomes by removing the toxin in a matter of hours. The modality used will depend on the characteristics of the exogenous toxin, most importantly, its molecular size and degree of protein binding. Hemodialysis is preferred for small molecular weight, non-protein-bound toxins, such as phenobarbital, whereas TPE is preferred for larger, protein-bound substances, such as non-steroidal anti-inflammatories.

Tips for Transfer

With an increased knowledge about these modalities and their uses in veterinary medicine, our referral community plays a significant role in identifying patients who might benefit from them and in preparing the clients with appropriate expectations. Immediate intervention is ideal in cases of toxin exposure, with these modalities being most effective six to 12 hours post-exposure. Patients with azotemia should be evaluated carefully, including urine output monitoring and tailored fluid therapy. With cases of suspected AKI, clients should be urged to consider hemodialysis earlier, as opposed to later, if the patient is developing progressive azotemia, oliguria, or electrolyte or acid/base disturbances. For patients with IMHA who require a second transfusion, clients should be made aware that if any further transfusions are needed, TPE will be the suggested course at that time.

For patients who may be appropriate candidates for extracorporeal therapy, we recommend avoiding jugular venipuncture, as the jugular veins need to be preserved for the hemodialysis/TPE catheter. Some patient characteristics are also essential to consider. Patients under 5kg can increase the challenges and risks of extracorporeal therapy, while large patients often require more supplies and subsequently incur a higher cost for therapy. Ideally, patients are behaviorally amenable to close contact with staff, as they are closely monitored and frequently handled during the treatments. Any concurrent

comorbidities that may complicate extracorporeal therapy should also be considered. A final consideration in determining if therapy can proceed is the availability of blood products, which are often required (packed red blood cells in smaller or anemic patients, and plasma for all TPE patients).

Hemodialysis, TPE, and hemoperfusion require a significant investment from the client, not only financially but also in terms of time and emotional energy. These modalities do come with some risks, including catheter complications, coagulation complications, and failure to respond to therapy. However, these modalities can be life-saving in appropriate patients, and, if clients are aware of the short-term commitment, it can be gratifying.

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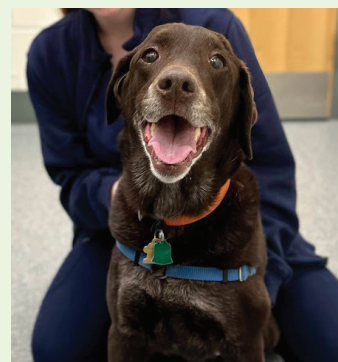
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Leptospirosis: Current Insights and Practical Approaches for Veterinarians

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Pathophysiology and Multi-Organ Manifestations

Leptospirosis is a zoonotic bacterial infection caused by spirochaetes of the genus *Leptospira*. Infection occurs via mucous membranes or abraded skin when exposed to contaminated urine, soil, or water. Rodents are a primary reservoir, and all dogs are at risk regardless of location or lifestyle. Leptospire invade host tissues, weaken endothelial barriers, and evade immune responses. Clinical complications include acute kidney injury (AKI) (95%), cholestatic hepatopathy (92%), and leptospiral pulmonary hemorrhage syndrome (LPHS) (58%). Other complications include vasculitis, myocarditis, coagulopathy, uveitis, myositis, enteritis, and pancreatitis. All dogs are at risk regardless of signalment, geographic location, and time of year.

Diagnosis

Clinical signs of leptospirosis in dogs are initially nonspecific, presenting as a febrile illness that can progress to renal and hepatic dysfunction, pulmonary involvement, and coagulopathy. Laboratory findings commonly include neutrophilia, thrombocytopenia, and non-regenerative anemia on a complete blood count. Chemistry panels may reveal azotemia, cholestatic hepatopathy with hyperbilirubinemia, and mild to moderate hypoalbuminemia. Urinalysis findings often show isosthenuria, renal glucosuria, and proteinuria. Coagulation testing may indicate prolonged Prothrombin Time/Partial Thromboplastin Time (PT/PTT) or hypercoagulability.

Imaging plays a crucial role in diagnosis and disease monitoring. Radiographs should be performed even in the absence of respiratory signs, as dogs with leptospirosis may have underlying pulmonary pathology, such as leptospirosis-associated pulmonary hemorrhage syndrome (LPHS), vasculitis, or aspiration pneumonia, in which imaging abnormalities could precede clinical signs. Identifying pulmonary changes aids in diagnosing LPHS, assessing fluid therapy, and monitoring respiratory function.

While no pathognomonic ultrasound findings exist for leptospirosis, a normal ultrasound does not rule out the disease. Furthermore, ultrasound is useful in excluding other causes of azotemia.

Diagnostic testing includes serology, PCR, and rapid tests. In serological testing using the microscopic agglutination test (MAT), infection is suggested when titers reach $\geq 1:800$, and a fourfold increase confirms the diagnosis. Testing for multiple serovars increases sensitivity, but it does not identify the infecting serovar. It should be noted that vaccination can influence MAT results.

PCR testing, which detects both viable and non-viable organisms, is the preferred method for early diagnosis of leptospirosis, and testing both blood and urine enhances sensitivity. Recent antibiotic administration may cause false negatives; however, vaccination does not interfere with results.

Rapid tests, such as SNAP Lepto and WITNESS Lepto, serve as useful screening tools by detecting antibodies. These tests may yield negative results early in the disease process and do require additional diagnostics for

confirmation. As mentioned above, a confirmatory diagnosis of leptospirosis is achieved through a fourfold or greater rise in *Leptospira* agglutination titers at a single laboratory, detection of pathogenic *Leptospira* via PCR in blood or isolation of *Leptospira* from a clinical specimen by a reference laboratory, though the latter is not clinically practical.

Antimicrobial Therapy

There is no universally optimal antibiotic regimen for the treatment of canine leptospirosis. For dogs that are eating well, the first-line treatment is doxycycline at a dose of 5 mg/kg orally every 12 hours for two weeks to prevent intra-renal persistence of the bacteria. In dogs not eating, ampicillin is administered intravenously at 20 to 30 mg/kg every six to eight hours to suppress bacteremia. If a urine culture is pending, Unasyn at 30 mg/kg intravenously every eight hours is recommended until the urine culture returns negative. In cases of severe acute kidney injury (AKI) classified by the International Renal Interest Society (IRIS) as Grade IV, where creatinine levels exceed 5 mg/dL, doubling the administration interval of Unasyn is advised. Early and aggressive treatment is crucial to prevent complications and improve outcomes.

Supportive Care

Supportive care for canine leptospirosis includes fluid therapy tailored to the dog's hydration status, urine output, and respiratory function. Dogs that are anuric, oliguric, have vasculitis or have severe hypoalbuminemia warrant extreme caution with fluid therapy due to the concern for possible fluid overload, and they should be referred to a tertiary facility. Dogs with leptospirosis can become extremely polyuric and require large fluid volumes to maintain hydration.

Supportive care involves gastrointestinal support, including antiemetics and gastroprotectants. If necessary, nutritional support should be provided via enteral feeding to maintain adequate caloric intake. Intensive monitoring of respiratory rate, blood pressure, electrolytes, complete blood count, and chemistry panels is crucial for the early detection of complications. In severe cases, advanced therapies, such as mechanical ventilation and dialysis, may be required to manage respiratory distress and acute kidney injury effectively.

When to Refer

Early dialytic intervention is recommended to prevent morbidity associated with acute kidney injury (AKI) rather than being used as a delayed salvage measure. Dialysis does not directly repair the kidneys but helps manage the clinical manifestations of uremia while allowing time for renal recovery after antibiotic therapy. Some dogs require multiple dialysis treatments, and a subset will not recover despite intervention. Referral for hemodialysis is indicated in dogs with IRIS AKI Grade 4, which is characterized by a serum creatinine level greater than 5 mg/dL. Traditionally, other indications for dialysis include severe acidosis, electrolyte disturbances, intoxication, fluid overload, and severe uremia. If there is a concern for declining respiratory status, severe hyperbilirubinemia, or progressive multisystemic disease, these patients should also be referred.

INTERNAL MEDICINE

Leptospirosis: Current Insights and Practical Approaches for Veterinarians

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Prognosis

The literature documents several survival rates for dogs with leptospirosis. As seen in a 2017 evaluation by Knöpfler et al., dogs treated conservatively had a 60% survival rate to discharge.⁵ However, according to the 2024 study by Ioannou et al., dogs receiving dialysis had a higher survival rate of 73%.⁴ It is possible that if the dogs treated conservatively had had access to renal replacement therapies, their prognosis may have been different.

The Ioannou et al. study also found that among dialysis survivors, 75% of dogs that survived to discharge were alive at long-term follow-up (≥ 6 months). However, 50% of those long-term survivors developed chronic kidney disease (CKD), requiring ongoing monitoring. Notably, the degree of azotemia was not associated with survival to discharge, but it was seen that the presence of multisystemic disease and severe hyperbilirubinemia may indicate a worse prognosis. Owners, therefore, should be warned about the possibility of CKD in these patients.

Vaccination

Leptospirosis vaccination is now a core vaccine. Annual vaccination is recommended for all dogs starting at 12 weeks of age, with priority given to those in endemic areas. If a dog was previously vaccinated but is no longer up to date, then the dog will need to have an initial vaccine followed by a booster. Dogs recovering from leptospirosis should be vaccinated as soon as possible after recovery as there are multiple infecting serovars, and it is unknown how long immunity lasts following natural infection.

While vaccination significantly reduces the risk of disease, infection can still occur in vaccinated dogs. Notably, in the study by Ioannou et al. on dogs with leptospirosis requiring hemodialysis, none of the affected dogs were fully vaccinated against the disease.

Conclusion

Canine leptospirosis remains a significant veterinary and public health concern. Early diagnosis, aggressive treatment, and comprehensive prevention strategies are critical in improving patient outcomes and reducing zoonotic risk. Early dialytic intervention is now recommended to reduce mortality. The leptospirosis vaccine is now a core vaccine. Ongoing research

and updated American College of Veterinary Internal Medicine (ACVIM) guidelines provide valuable insights for managing this complex disease.

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Computed Tomography Angiography (CTA)

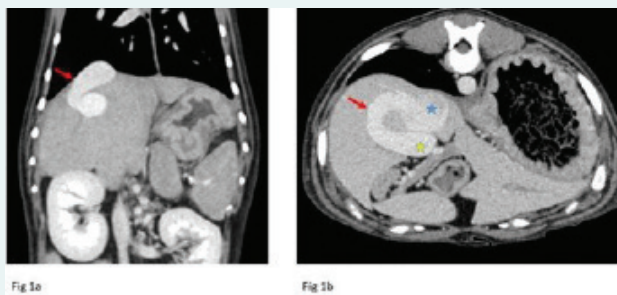
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Introduction

Computed Tomography (CT) has become a mainstay in veterinary medicine. With the now pervasiveness of this diagnostic tool, it has become vital to make certain distinctions between the various CT protocols. Routine CT scans include a pre-contrast scan, followed by the administration of a contrast medium and a post-contrast scan. These scans are performed to optimize contrast enhancement of the organ parenchyma. Compare this to CT angiography (CTA), which includes a pre-contrast scan followed by post-contrast scans that are timed to achieve specific vascular phases (i.e., arterial, portal, venous, and delayed). CTA is the recommended imaging technique to diagnose various vascular abnormalities, characterize various vascular parenchymal lesions, as well as evaluate tumors, as discussed below.

FIGURE 1

› **Figure 1a:** Example of an intrahepatic portosystemic shunt. Red arrow denotes the shunting vessel. **Figure 1b:** The blue star marks the caudal vena cava and the yellow star marks the portal vein.



CTA for Portosystemic Shunts/Portal Vascular Diseases

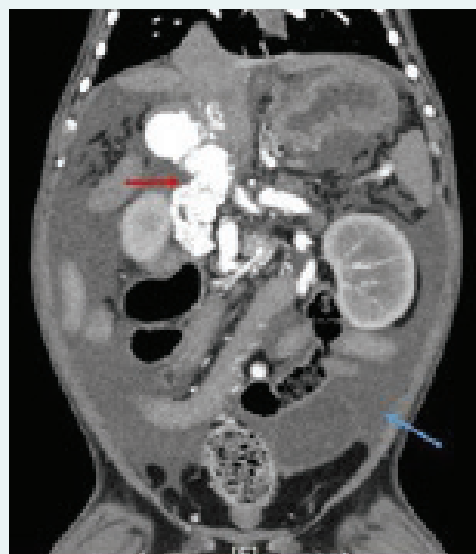
One of the most common indications for CTA is the evaluation of portosystemic shunts (PSS). Congenital intrahepatic PSS occurs more frequently in large-breed dogs, while congenital extrahepatic PSS is more common in small-breed dogs. It is recommended to further classify either type of shunting prior to surgical intervention (Fig. 1). CTA has been shown to be the best modality for characterizing the origin and termination of the shunting vessel, as well as evaluating for possible additional shunting vessels that can be missed on ultrasonography or intraoperative mesenteric portovenography.¹ Other portal vascular diseases that can be evaluated on CTA include arterioportal fistulas, acquired portosystemic shunting, and portal vein thrombosis, which can mimic portal hypertension secondary to liver disease. CTA is the modality of choice for portal vein disease with improved detection compared to ultrasound (Fig. 2).²

Other Vascular Anomalies

Other vascular anomalies that are evaluated with CTA include vascular ring anomalies, coronary artery anomalies, arteriovenous fistulas, and

FIGURE 2

› Example of nest of tortuous vessels (red arrow) secondary to an arterioportal fistula documented in an arterial phase. Due to the secondary portal hypertension, there is a large accumulation of ascites (blue arrow).



thromboembolism, including pulmonary thromboembolism. Vascular ring anomalies are often present in young patients with regurgitation and focal esophageal enlargement cranial to the heart base. There are various types of vascular ring anomalies, including persistent right aortic arch, aberrant subclavian arteries, and double aortic arch, and they can all present with similar clinical signs. CTA will allow for classification of the type of vascular ring anomaly and surgical planning (Fig. 3). Treatment for pulmonary valve stenosis is balloon valvuloplasty; however, the presence of certain coronary artery anomalies is a contraindication for this treatment and can increase the risk of sudden death during the procedure. CTA of the thorax is required for those breeds with a higher risk of this anomaly to map the coronary artery anatomy prior to the valvuloplasty procedure.³ Evaluation of the arterial phase is required to diagnose an arteriovenous fistula. Thus, a routine CT is not sufficient to diagnose this anomaly. A CTA scan is needed to diagnose thromboemboli, especially in the thorax, where the vasculature cannot be evaluated on ultrasound.

Insulinomas

CTA is also the preferred diagnostic test for evaluating insulinomas. Insulinomas are often small and can be difficult to detect on abdominal ultrasound, especially in large or deep-chested dogs, where the entire pancreas is a baseline difficult to image. It has been documented that insulinomas exhibit increased contrast uptake on the arterial phase on CTA, and then most become

DIAGNOSTIC IMAGING

Computed Tomography Angiography (CTA)

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FIGURE 3

➤ Example of a persistent right aortic arch (red arrow). The green outline denotes a focal, severely distended esophagus with gas and fluid.



similarly contrast-enhancing to the pancreas parenchyma on the delayed post-contrast phase (Fig. 4).^{4,5} This imaging characteristic makes it difficult to detect small tumors on routine CT, with CTA being a required diagnostic test for insulinomas. In addition, during the arterial phase, marked contrast-enhancing lesions within the liver and lymph nodes can strongly indicate regional metastasis.⁵

FIGURE 5

➤ **Figure 5a and 5b.** The red oval marks a large left-sided hepatic mass (presumptive hepatocellular carcinoma).

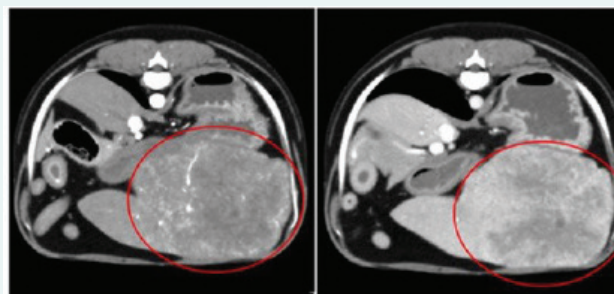


Fig 5a

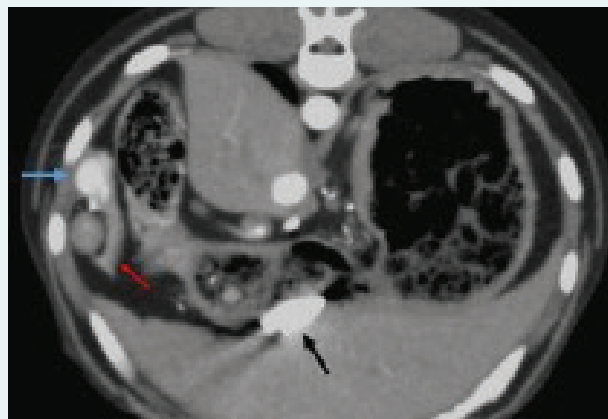
Fig 5b

Tumor Evaluation and Surgical Planning

Lastly, CTA has been used for surgical planning of tumor resection and possibly for evaluating certain tumor types. Using preoperative CTA for isolated hepatic masses has allowed for a more accurate diagnosis of the location within the liver, as well as vascular and multilobar involvement, which can better predict surgical difficulty and resectability. Ultrasonography has been shown to correctly localize solitary hepatic masses in only about 50% of cases.⁶ The most common malignant hepatic tumor type in dogs is hepatocellular carcinoma, which is often described as a large mass with central cyst-like or necrotic regions on CT, compared to nodular hyperplasia, which is smaller and has a more homogeneous appearance post-contrast. On CTA, hepatocellular carcinomas tend to exhibit heterogeneous contrast enhancement with irregular central or marginal contrast enhancement in the arterial phase (Fig. 5). In addition, hepatocellular carcinomas tend to be

FIGURE 4

➤ Example of an early post-contrast scan. The blue arrow marks a markedly contrast-enhancing nodule within the pancreas (red arrow) that was confirmed to be an insulinoma. The black arrows mark metallic hemoclips secondary to a prior liver biopsy.



more hypoattenuating compared to the liver parenchyma on the more delayed phases.⁷ Nodular hyperplasia, on the other hand, tends to exhibit more homogeneous hyperattenuation or isoattenuation compared to the liver parenchyma and other primary hepatic masses on the various post-contrast phases, and hepatic metastatic lesions more commonly are homogeneously hypoattenuating in the earlier post-contrast phases.⁸ Despite these imaging characteristics helping to prioritize malignant versus benign lesions, other reports document more of an overlap between tumor types, and ultimately, histopathology remains the gold standard for diagnosis.⁹

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Common Canine Retinal Conditions

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Introduction

The evaluation and diagnosis of retinal conditions are challenging for many veterinary practitioners. These difficulties are due to numerous reasons, including the infrequent use of specialized equipment made for the examination of the ocular fundus, the learning curve that exists to get a reliably detailed view of the retina and choroid, and the recollection of specific knowledge needed to interpret the type of lesions present. For etiological characterization, fundus lesions can be divided into two categories: those that are caused by primary ocular disease and those that are an extension of systemic disease, or Ocular Manifestation of Systemic Disease (OMSD). By reviewing the relative structure and function of the retina and ocular fundus, as well as ocular examination techniques, veterinarians can understand the most common categories of retinal conditions presented by dogs.

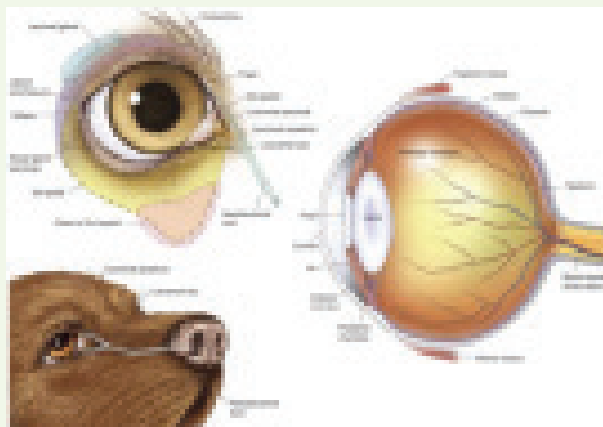
Structure of the Retina and Ocular Fundus

The retina is the innermost layer of the ocular fundus (fundus meaning the deep end of the eye opposite the cornea and pupil), or posterior portion (Figure 1). The neuroretina consists of multiple layers that capture an image and transmit it to the brain through a series of neural connections beginning with the photoreceptor layer and ending with the ganglion cell layer that leads to the optic nerve and brain (Figure 2). The retina itself, upon direct examination, is largely invisible to the naked eye due to its thinness, visible only through the blood vessels that course through the retinal tissue and form the characteristic radiant pattern that emerges from the optic nerve and supplies blood to the retina in an arborizing pattern to the retinal periphery (Figure 3).

The retina is only fixed to the eye in two places: at the optic nerve and at the equator, or the periphery ocular fundus. The rest of the retinal surface area is physically held to the ocular fundus by apposition with the vitreous, pressing

FIGURE 1

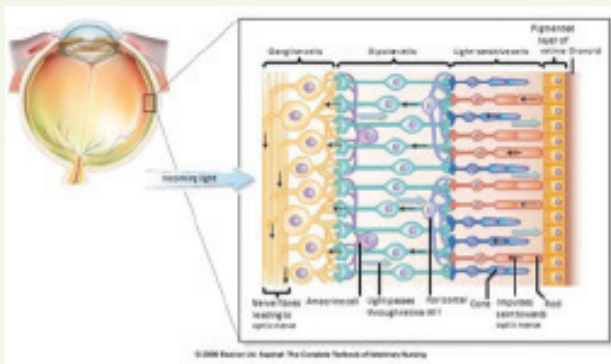
» Orientation of retina and visual axis.



the retina up against the fundus to maintain its connection to the retinal pigmented epithelium (RPE layer), which is the deepest portion of the retina and the part of the ocular fundus that we primarily visualize. Behind the RPE, as seen through the pupil, is the choroid, with the scleral tissue further back. Normally, the sclera and much of the choroid are not visible unless specific eye conditions allow direct viewing of the choroid through the visual axis (e.g., subalbinotic or merle dogs). In many dogs, the RPE is lacking pigment in a specific central area, often triangular, known as the tapetum lucidum, or shining layer. The tapetal region is usually brightly colored and serves to enhance light vision in low-light conditions (Figures 1 and 3).

FIGURE 2, 3

» **Figure 2 (left):** Neuroretinal layout. **Figure 3 (right):** Normal canine retina.



OPHTHALMOLOGY

Common Canine Retinal Conditions

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FIGURE 4

» Ocular ultrasound. Courtesy Matt Chandler.



How the Retina Functions

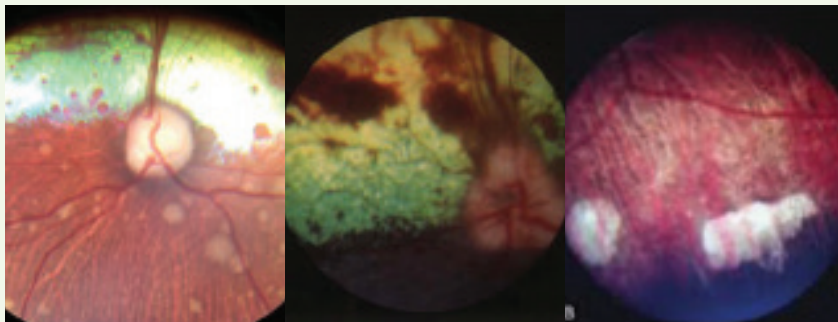
When light travels into the eye, it is initially perceived by the photoreceptors that lie deepest in the retina, furthest from the cornea and pupil, abutting the RPE layer (Figure 2). The neural transmission then travels from the photoreceptors rostrally through the neural retina to the ganglion cell and nerve fiber layer, which is the anterior-most layer that abuts the vitreous. The nerve fiber layer transmits the nerve impulse to the optic disk and optic nerve, which then carries the signal to the occipital cortex in the brain. Retinal physiology is a highly metabolic process that requires substantial energy and metabolism, with the extremely high retinal blood flow velocity to maintain retinal function. (The retina has the second-highest blood flow velocity in the body, just after the kidney). When the retinal function is disrupted, either physically or metabolically through disease, the visual neural pathway is broken, and partial or total vision loss can result.

Techniques in Retinal Evaluation

The neuroretina is extremely fragile when subject to physical stress and will tear easily. The normal canine retina is about 180 to 200 microns thick at the center. The retina is as fragile as wet tissue

FIGURE 7, 8, 9

» **Figure 7 (left):** Infiltrative retinal disease (fungal). **Figure 8 (center):** Intraretinal bleeding (polycythemia). **Figure 9 (right):** Uveodermatologic syndrome (VKH).



paper, making it difficult to handle; therefore, only a few veterinary ophthalmology subspecialists with posterior segment surgery equipment can perform retinal reattachment surgery in dogs. Many veterinary ophthalmologists can stem the advance of retinal tears with laser retinopexy using a diode laser to weld the retina to the RPE and choroid.

Retinal disease is typically diagnosed with ophthalmoscopy. Indirect ophthalmoscopy is often preferred for a comprehensive view of the fundus, while direct ophthalmoscopy provides a detailed view of smaller sections of the retina. The retinal examination includes not only ophthalmoscopy, but also other aspects of the ocular examination that play a critical role, including pupillary light reflexes, color-specific testing to differentiate between rod and cone disease, maze testing, tonometry, and menace/dazzle reflex testing. When the visual axes are not clear or the nature of the retinal disease is not discernible by examination alone, other diagnostics may include ultrasonography (Figure 4) or electroretinography. Ultrasound can be used to confirm the nature and extent of retinal detachments or intraocular masses when they are

not directly visible due to other ocular changes, such as cataracts, globe position, corneal cloudiness, or eyelid swelling.

Vision breakdown related to retinal disease can be divided into two categories: 1) physical separation of the retina from the ocular fundus (a.k.a. detachment) and 2) primary or secondary conditions in the retina itself that disrupt the neural tissue function and vital visual pathways. These two causes are not mutually exclusive and may overlap at times.

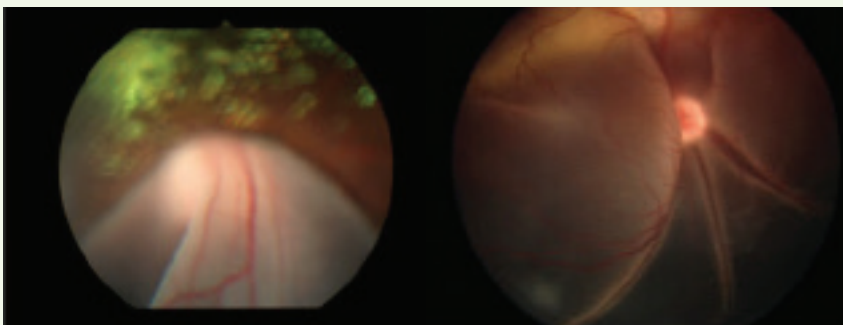
Retinal Detachments

Diseases in which there is a physical separation of the retina from RPE and choroid are categorized as retinal detachments. They can occur due to physical trauma to the eye and retina, as well as diseases that cause the retina to pull away from the ocular fundus through metabolic or biochemical changes, often as a manifestation of systemic disease. Traumatic or physical pulling of the retina from the ocular fundus can have various causes in dogs. The ones we see most frequently are related to head trauma, lens instability and subluxation (almost always anterior), and marked vitreal degeneration. Within this last vitreal degeneration category, there are some breeds where the retina spontaneously detaches peripherally from its border attachments, referred to as disinsertion. We see this almost exclusively in Shih Tzus. Mass effect of intraocular tumor, either primary or metastatic, may also cause the retina to detach if the tumor is pushing or pulling at the retina as it expands in size.

Retinal detachments can be subdivided into two categories relative to the state of intactness: rhegmatogenous and non-rhegmatogenous. Rhegmatogenous detachments are those that have tears or holes, leaving the retina incomplete. Often, these detachments have pulled away from their natural position and frequently lie in the inferior globe, like a drape or veil, over the optic disk (Figure 5). Non-rhegmatogenous detachments

FIGURE 5, 6

» **Figure 5 (left):** Retinal detachment or disinsertion. **Figure 6 (right):** Bullous retinal detachment.



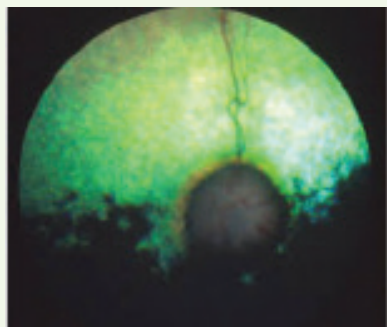
OPHTHALMOLOGY

Common Canine Retinal Conditions

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FIGURE 10

» Retinal degeneration.



have no tears and can be further characterized into bullous detachments that billow anteriorly, giving a “morning glory” or “parachute” appearance (Figure 6). Bullous detachments can tear and become rhegmatogenous detachments in time.

Metabolic or biochemical changes in the eyes also may or may not lead to retinal detachment. These comprise both vascular and infiltrative conditions, including systemic hypertension, infiltrative cancer, infectious diseases (e.g., fungal retinopathies like aspergillus, histoplasmosis, blastomycosis, and cryptococcosis), toxoplasmosis, heartworm disease or other parasitic oculomyiasis of insect larvae (Figure 7), and other vasculopathies related to hematologic disorders, such as coagulopathy or polycythemia (Figure 8). The most common conditions we see in dogs that are related to infiltrative disease are cancers, such as lymphoma or meningioma, steroid-responsive idiopathic bullous retinal detachments, fungal and toxoplasma infections, and autoimmune diseases, including uveodermatologic syndrome/Vogt-Koyanagi-Harada Syndrome (VKH), which is commonly seen in Akitas and other Northern breeds (Figure 9).

Retinal Degeneration

Retinal diseases that do not involve detachment are typically related to degenerative diseases or biochemical loss of retinal function. These

conditions include the retinal degenerations familiar to us as a collection of diseases termed progressive retinal atrophy (PRA), or progressive rod-cone degeneration (PRCD) (Figure 10). Most, but not all, retinal degenerations are bilateral and have a genetic component. They can be early or late onset in age, and the rate of progression can vary. Most rod-cone degenerations present as a loss of night vision first, followed by day vision, while cone-rod degenerations can occur with day vision lost first, followed by night vision. Sudden Acquired Retinal Degeneration Syndrome (SARDS) is a subset of these conditions that has a particular onset in midlife and is rapid in progression. It is sometimes associated with hyperadrenocorticism (Cushing's), but the cause-and-effect relationship of this association is unclear. There is usually no treatment for SARDS or other retinal degenerations. These conditions are painless, but some dogs may develop cataracts, which can cause uveitis in certain cases. The Orthopedic Foundation for Animals' Blue Book lists retinal degeneration across all OFA breeds. Additionally, a variety of genetic tests are available to confirm the diagnosis of several genetic variants of canine retinal degeneration.

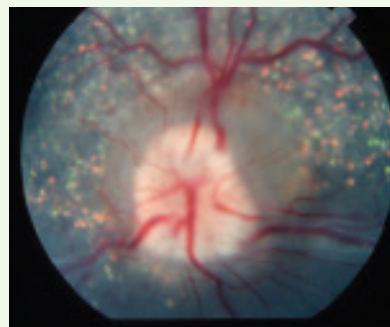
Over time, retinal degenerations are characterized by retinal thinning that leads to increased tapetal reflectivity, attenuation of retinal blood vessels, and diminished pupillary light reflexes. Loss of pupil movement in red light indicates reduced rod function (night vision), while loss of movement in blue light reflects decreased cone function (day vision). In early stages of disease, or when the retina is not visible due to cataracts, an electroretinogram is often used to rule in retinal degeneration as a cause of vision loss. To further confirm a retinal degeneration diagnosis, which differs from retinal detachment, techniques such as ocular ultrasound or optical coherence tomography (OCT) (Figure 11) are used to differentiate.

Other Causes of Vision Loss

It is worth mentioning that there can be causes of blindness with a clear visual axis that are not related to retinal disease, mainly diseases of the optic nerve and visual cortex of the brain. Optic

FIGURE 12

» Optic Neuritis.



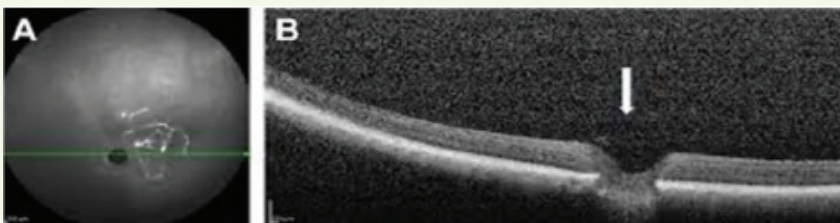
neuritis is a set of conditions where the optic nerve is inflamed, which blocks the transmission of vision signals to the brain and presents as blindness. Often, the retinal examination is unremarkable, and pupillary reflexes are variable but frequently remain intact with all light colors, suggesting the retina is intact and functioning. Meningitis (MUE, or GME), optic nerve inflammation from cancer, vascular disruption to the optic nerve, and trauma are frequent causes of optic nerve dysfunction. The optic nerve may look inflamed or swollen (Figure 12) with ophthalmoscopy or OCT. Treatment with anti-inflammatories, advanced imaging of the brain, and cerebrospinal fluid analysis with full neurological evaluation are often recommended to fully evaluate and care for vision loss associated with optic nerve disease.

Summary

Diseases of the retina can be caused either by retinal detachment and tear, or by conditions where the retina remains in place but loses function due to other degenerative, infiltrative, or vascular diseases. Ophthalmoscopy and enhanced imaging, including electroretinography, ocular ultrasound, and other imaging (MRI, OCT), can assist in better characterizing retinal disease. Serology and, in some cases, ocular aspirates (or ocular histopathology) can rule in infectious diseases where more in-depth neurological evaluation and care can be done to diagnose and manage diseases of the optic nerve and visual cortex.

FIGURE 11

» Ocular coherence tomography.



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- **Cytology of Emerging Canine and Feline Infectious Diseases** - Patty Ewing, DVM, MS, DACVP
(Clinical and Anatomic Pathology)
- **Navigating the Supplement Maze: Evidence-Based Approaches to Managing Allergies in Dogs** - Meagan Painter, DVM, DACVD
- **Fracture Repair Techniques: How to Choose the Best Option for Each Case** - Jennifer Peterson-Levitt, DVM, MS, DACVS

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