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## ANESTHESIA

# Anesthesia and Analgesia for the Cesarean Section

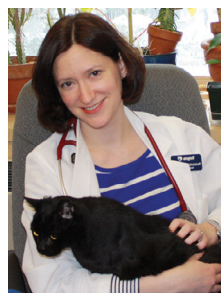
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**I**n considering an anesthetic protocol for both emergent and elective cesarean sections (C-sections), it's important to first understand maternal physiologic changes associated with pregnancy and fetal physiology considerations. Major body systems affected by pregnancy include the cardiovascular (CV), respiratory, gastrointestinal, and renal systems — all of which are equally important to consider during any anesthetic event. A brief summary of how these systems are impacted during pregnancy and anesthesia is outlined in Table 1 on page 2. In regard to fetal physiology, it is imperative to know that drugs that cross the blood-brain

barrier also cross the blood-placental barrier. Enzymes responsible for hepatic metabolism are ineffective until 3-5 weeks of age. Finally, the fetal oxyhemoglobin dissociation curve is shifted to the left, meaning that there is less unloading of oxygen to tissues.<sup>1,2,3</sup>

An ideal protocol for C-section patients provides adequate analgesia to the mother, optimal operating conditions, rapid recovery, and minimal fetal depression (Table 2). It is important to note that maternal and puppy or kitten mortality increases (CONTINUED ON PAGE 2)



## CARDIOLOGY

# Oral Antiarrhythmic Therapy

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**W**hen your patient's heart beats too fast or irregularly, it's enough to make a veterinarian's heart speed in response. My hope is that this review of the most commonly used oral antiarrhythmic medications in veterinary medicine will help to reduce the heart rate of patient and doctor alike. On page 4 you will find a quick reference of the actions, side effects, and considerations for selection of the four drugs you

are most likely to prescribe or encounter. (Dosages for the medications can be found in Plumb's Veterinary Drugs).

## QUICK REVIEW OF ANTIARRHYTHMIC CLASSIFICATIONS

You may not have spent much time thinking about the Vaughan-Williams classification scheme for antiarrhythmic (CONTINUED ON PAGE 4)

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## Anesthesia and Analgesia for the Cesarean Section

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TABLE 1 — MATERNAL PHYSIOLOGIC CHANGES AND THEIR ANESTHETIC IMPACT.<sup>1,2,3</sup>

Body System	Changes During Pregnancy	Anesthetic Impact
<b>Cardiovascular</b>	<ul style="list-style-type: none"> <li>• Blood volume increases ~40% with larger increase in plasma volume); thus, relative anemia develops</li> <li>• Increased cardiac output (CO) due to increased heart rate and stroke volume</li> <li>• Right shift of oxyhemoglobin dissociation curves facilitates oxygen delivery to fetus</li> <li>• Increased myocardial work and reduced cardiac reserve — susceptible animals incapable of mounting a significant response to CV stressors</li> </ul>	<ul style="list-style-type: none"> <li>• Common to battle hypotension with patients in dorsal recumbency as venous return is compromised by pressure of large uterus on vena cava</li> <li>• Following removal of the uterus, abdominal vessels may vasodilate, leading to systemic hypotension</li> <li>• Judicious use of IV fluids recommended unless patient is at risk for developing CHF +/- use of vasopressors</li> <li>• Animals with preexisting myocardial dysfunction may decompensate and develop heart failure</li> </ul>
<b>Respiratory</b>	<ul style="list-style-type: none"> <li>• Oxygen consumption increases by ~20% (due to developing fetus, placenta, uterine muscle, and mammary tissue)</li> <li>• Functional residual capacity (FRC) and total lung capacity reduced</li> <li>• Increased tidal volume and respiratory rate result in increased minute ventilation</li> </ul>	<ul style="list-style-type: none"> <li>• Patients predisposed to hypoxemia — pre-oxygenation IMPERATIVE</li> <li>• Increase in alveolar ventilation and reduced FRC result in reduced MAC requirements</li> <li>• Reduced P<sub>a</sub>CO<sub>2</sub>, generally in the range of 30-33 mmHg</li> </ul>
<b>Gastrointestinal</b>	<ul style="list-style-type: none"> <li>• Positioning of uterus and increased progesterone result in reduced gastric emptying and reduced gastric motility</li> </ul>	<ul style="list-style-type: none"> <li>• Increased risk of regurgitation and aspiration</li> <li>• Higher percentage of brachycephalic breeds requiring C-section adds to this risk</li> <li>• Rapid sequence induction and intubation to quickly protect the airway</li> <li>• Consider use of metoclopramide and/or an H<sub>2</sub>-receptor antagonist</li> </ul>
<b>Renal</b>	<ul style="list-style-type: none"> <li>• Renal blood flow and GFR increased</li> <li>• Patients often have low or low normal BUN and creatinine</li> </ul>	<ul style="list-style-type: none"> <li>• Elevated renal values may indicate dehydration or underlying kidney disease</li> <li>• Pre-surgical fluid resuscitation and adjusted fluid therapy/use of vasopressors during anesthesia necessary to maintain kidney perfusion</li> </ul>

TABLE 2 — ELECTIVE AND EMERGENT C-SECTION PROTOCOL SUGGESTIONS IN DOGS AND CATS.<sup>3</sup>

Species	Anesthetic Protocol	Analgesia
<b>Dog</b>	<ol style="list-style-type: none"> <li>1. Pre-oxygenate, clip, and prep</li> <li>2. Propofol (4-6 mg/kg) or alfaxalone (2-3 mg/kg) titrated to effect IV</li> <li>3. Maintenance on isoflurane or sevoflurane</li> <li>4. Balanced crystalloid IVF at 5-10 mL/kg/hr (adjust if preexisting heart disease)</li> </ol>	<ol style="list-style-type: none"> <li>1. Line block with bupivacaine (1 mg/kg) or lidocaine (2 mg/kg)</li> <li>2. Buprenorphine (0.01 mg/kg) or hydromorphone (0.05-0.1 mg/kg) IV after removal of puppies</li> <li>3. Single dose of carprofen (4.4 mg/kg) or meloxicam (0.1 mg/kg) SC</li> </ol>
<b>Cat</b>	<ol style="list-style-type: none"> <li>1. Same as above</li> </ol>	<ol style="list-style-type: none"> <li>1. Same as above aside from these changes</li> <li>2. Buprenorphine (0.02 mg/kg) IV after removal of kittens</li> <li>3. Single dose of meloxicam (0.1 mg/kg) or robenacoxib (2 mg/kg) SC</li> </ol>

with emergent versus planned C-section.<sup>4,5</sup> Fetal and neonatal mortality can be minimized by avoiding placental hypoperfusion and hypoxemia and providing proper neonatal care upon delivery.<sup>6,7,8</sup> Prior to anesthesia, the mother should have a full physical exam, thorough history taking, and a minimum database of blood work (e.g., PCV/TP/Azo/iCa/Glu). In debilitated patients or those with comorbidities, a complete database of lab work should be performed prior to anesthesia. Correction of electrolyte and/or hydration/volume deficits should occur before induction.

Most dystocia patients are easy to handle and calm, allowing for IV catheter placement and

initial abdominal clipping and prepping *before* administration of anesthetic agents. Pre-anesthetic medications can and should be avoided unless the patient is overly fractious or unmanageable. If dealing with an aggressive patient, consider short-acting and reversible drugs such as fentanyl and midazolam. Alpha-two adrenergic agonists, particularly xylazine, should be avoided as they have been linked to decreased survival in the neonate.<sup>4</sup> All patients should be pre-oxygenated for at least five minutes and induced in the *or* when possible. The primary determinant of fetal viability is the time from anesthetic induction to delivery, with a target of < 15 minutes.<sup>4,5,6,7,8</sup> Propofol or alfaxalone are induction agents of choice, both short-acting and with similar CV and respiratory

effects. Both drugs cross the placenta, but there is rapid clearance and minimal fetal depression. Newer studies have shown improved APGAR scores within the first 60 minutes following delivery in neonates with an alfaxalone induction compared to propofol.<sup>9,10</sup> Survival out to 3 months was similar between groups, however, indicating that either induction agent is appropriate.<sup>9</sup>

Following induction, maintenance can be achieved with isoflurane or sevoflurane, keeping in mind that maternal anesthetic requirements are often reduced due to pregnancy-induced changes. Occasional small boluses of propofol or alfaxalone can be used if patient depth suddenly lightens. Multimodal analgesia is important and possible

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in the mother. Prior to surgery, a line block can be performed quickly with either bupivacaine or lidocaine. After delivery (Figure 1), an opioid and chosen intra-operatively antibiotic can be administered. Inhalants should be adjusted following removal of the puppies/kittens since the opioid effectively reduces anesthetic requirements. Buprenorphine, a partial mu agonist, is recommended as it results in minimal sedation and adequate analgesia. A full mu agonist (e.g., hydromorphone or methadone) can be used as an alternative, but lower doses are recommended to minimize CV and respiratory depression as well as limit the degree of sedation. An epidural can be considered *postoperatively* with morphine alone or morphine and lidocaine. If an epidural is performed, owners should be made aware of possible urinary retention and hind end weakness (short duration) if lidocaine is used.<sup>1,2,3</sup> Finally, a single postoperative dose of an NSAID can be considered in patients with normal blood work and adequate intraoperative blood pressures (MAP > 65 mmHg).

Neonatal management and resuscitation begins by having one person dedicated to each neonate when possible. Early and frequent rubbing to dry helps stimulate respiration and keep the neonates warm. A bulb syringe can be used to suction oral and airway secretions. Swinging the neonate is *not* recommended as this has been shown to result in cerebral hemorrhage.<sup>1,2,3,6,7,8</sup> Neonates that are thriving can be placed in an incubator for heat support until the mother recovers. In neonates that may be struggling, ventilation is key as it 1) allows for elimination of gas anesthetics and 2) aids in increasing HR as bradycardia is caused by hypoxia.<sup>6,7,8</sup> Attempts can be made to stimulate ventilation by activating the acupuncture point, GV26, with a small gauge needle inserted into the nasal philtrum. Oxygen supplementation can be provided via mask or intubation. The most common arrest rhythm for neonates is asystole and should be treated with basic life support measures, including epinephrine +/- atropine. Consider sublingual use of reversal agents (e.g., flumazenil, naloxone) if benzodiazepines or opioids were used prior to fetal delivery.

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FIGURE 1

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## CARDIOLOGY

## Oral Antiarrhythmic Therapy

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drugs since veterinary school, but it can be useful for a reminder of both the intended effects and side effects of various medications. For example, any drug with beta blocking or calcium channel blocking action will be both negatively chronotropic (lowering the heart rate) but also negatively inotropic (reducing contractility). The scheme divides antiarrhythmic drugs into classes according to the ion channels they block:

<b>CLASS I:</b>	Sodium channel blockers (e.g., lidocaine, procainamide, mexiletine)
<b>CLASS II:</b>	Beta blockers (e.g., atenolol, metoprolol)
<b>CLASS III:</b>	Potassium channel blockers (sotalol and amiodarone both have class III activity in addition to other channel blockade(s), making them mixed agents)
<b>CLASS IV:</b>	Calcium channel blockers (e.g., Diltiazem, verapamil)

## ATENOLOL

In addition to being used for heart rate reduction in cases of outflow tract obstructions (e.g., hypertrophic obstructive cardiomyopathy, pulmonic or aortic stenosis), atenolol is a commonly used medication either alone or in combination with other drugs to treat supraventricular or ventricular arrhythmias. Although it is not particularly efficacious as a solo drug for ventricular tachycardia in dogs, it can be more useful in treating ventricular premature complexes or tachycardia in cats. Atenolol is a beta blocker (class II agent), and one that is relatively specific for the beta 1 receptors (found in the heart muscle). However, it does have some beta 2 activity (affecting bronchial and smooth muscle), and, as such, caution should be taken when using the drug in poorly controlled asthmatic patients due to potential bronchoconstriction. It could mask physiologic response to hypoglycemia, so caution in poorly controlled diabetics is also warranted. Because atenolol will also have negative inotropic effects, caution should be used with reduced systolic function, and it is best to avoid starting the drug during active congestive heart failure.<sup>1</sup>

Atenolol has the advantage of being inexpensive, and generally well-tolerated. Due to renal metabolism, doses should be adjusted when significantly reduced renal function is present. Avoid stopping the drug suddenly or missing doses with chronic use. Since the number of beta

receptors increases with long-term administration, stopping suddenly could lead to hyper-reaction to physiologic adrenergic stimulation.

## Atenolol Highlights

- Inexpensive
- Does not work well alone for ventricular tachycardia in dogs (but may in cats)
- Caution with systolic dysfunction, CHF, AV block
- Caution in poorly controlled asthmatics, diabetics
- Reduce dose with severe renal dysfunction
- Avoid stopping suddenly after chronic use

## SOTALOL

Sotalol is one of the most commonly used oral antiarrhythmic agents for ventricular tachycardia, for good reason. Sotalol has the benefits of being relatively inexpensive, fast-acting, tends to be well tolerated, and only requires twice-daily administration. A mixed agent, with both beta blocking (class II) and potassium channel blocking (class III) properties, sotalol tends to have more potent effect against ventricular arrhythmias than a solo beta blocker such as atenolol in dogs.<sup>2</sup> What many veterinarians may not realize is that sotalol can also be effective in treating supraventricular tachycardia.

Due to negative inotropic effects, caution must be taken when the patient has concurrent congestive heart failure, especially with reduced systolic function such as with dilated cardiomyopathy. As sotalol is a nonselective beta blocker (albeit a relatively weak one; most of its pharmacologic actions are related to its class III potassium channel blockade), it may also cause bronchoconstriction and should be used with caution in patients with lower airway disease. Elimination of sotalol is almost entirely via the kidney, and dosage intervals may need to be extended in patients with renal dysfunction. Sotalol has a quick onset of action, reaching peak plasma levels two to four hours after dose administration, providing enhanced usefulness in the ER setting.<sup>3</sup>

## Sotalol Highlights

- Inexpensive
- Twice-daily dosing
- Well-tolerated
- Quick onset of action
- Caution with systolic dysfunction, congestive heart failure, AV block

## MEXILETINE

Mexiletine is a class I agent (sodium channel blocker) with similar properties to lidocaine. Mexiletine is most commonly used along with a beta blocker such as atenolol or sotalol, which further enhances arrhythmic suppression through cooperative drug action.<sup>4</sup> When used as a solo agent, higher doses are often required, which increases risk of adverse effects. Mexiletine does have disadvantages compared to sotalol monotherapy, including increased cost, and typically three times daily dosing (some patients may respond adequately to twice-daily dosing when given with sotalol<sup>5</sup>). Given routine availability in only three different doses of capsules (150 mg, 200 mg, 250 mg), mexiletine can be challenging to dose, especially in smaller patients. Mexiletine does have the advantage of not possessing negative inotropic actions, making it a safer choice in patients with significant systolic dysfunction (even if it requires concurrent beta blocker use, it may allow reduced doses of those agents to be used).

Mexiletine has higher potential to cause adverse side effects compared to sotalol, most commonly GI upset (reduced appetite, vomiting and/or diarrhea). Giving the drug with food reduces this risk. Potential CNS effects (trembling, unsteadiness, dizziness, depression) in people taking the drug have been reported. Similar signs have been seen in dogs, with a subset of patients exhibiting mildly dull or disoriented behavior. In my experience, this reaction does not appear to be dose-dependent and does resolve if the drug is stopped.

Due to liver metabolism, the half-life of mexiletine may be significantly increased in patients with moderate to severe hepatic disease. It is recommended to test dogs of breeds susceptible to the MDR1 gene mutation prior to using the drug, as those homozygous for the MDR1 gene defect may be susceptible to mexiletine toxicity.<sup>6</sup>

## Mexiletine Highlights

- More effective with concurrent atenolol or sotalol
- Typically requires TID dosing
- No negative inotropic effects
- Higher risk of side effects (GI upset, CNS signs)
- Caution in dogs with MDR1 gene mutation

## DILTIAZEM

Calcium channel blockers are very useful agents for treatment of supraventricular arrhythmias

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such as atrial fibrillation or atrial tachycardia, and diltiazem is the most commonly used oral calcium channel blocker in veterinary medicine. Studies have shown it is most effective for heart rate control with atrial fibrillation when used in conjunction with digoxin,<sup>7</sup> but it may be adequately effective on its own. It is important to be aware that oral diltiazem comes in numerous human dosage forms, including standard release tablets (e.g., Cardizem®) and various sustained release products (e.g., Dilacor XR®, Cardizem CD® and LA®). Standard release forms are typically used three times daily for arrhythmia control in dogs, whereas the sustained release products are typically used twice daily (although there is limited pharmacokinetic data for use of the sustained release products in veterinary patients). The most critical thing to note is that the recommended or utilized mg/kg dose for the two forms is not equivalent (sustained release products are typically used at a higher dose BID), and they are not simply interchangeable.

Standard forms of diltiazem are relatively inexpensive, whereas the sustained release forms can be more costly. Both forms have been associated with decreased appetite and weight loss, especially in cats. Diltiazem possesses negative inotropic properties, and so caution must be used with systolic dysfunction. Ideally,

the drug would not be started during active congestive heart failure. It also should not be started with severe hypotension, sick sinus syndrome, or significant AV block. Caution is warranted with use in geriatric patients, or those with serious hepatic or renal impairment.<sup>8</sup>

## Diltiazem Highlights

- Comes in standard (TID) and sustained release (BID) forms; dosing is not equivalent
- GI side effects common in cats
- Some negative inotropic effects
- Better heart rate reduction in atrial fibrillation when used in conjunction with digoxin

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# Diagnosis and Treatment of Chronic Hepatitis in Dogs: Summary of the 2019 ACVIM Consensus

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**T**his consensus was generated from published veterinary and selected human studies summarized by a panel of seven specialists with extensive experience and training in canine hepatology.

When hepatic biopsy reveals inflammation, we must be careful to differentiate between primary and secondary hepatopathy. In both cases, inflammatory infiltrates are present, including lymphocytic, plasmacytic, or granulomatous inflammation. The key difference is that in primary hepatopathies, evidence of hepatocyte cell death is seen along with variable degrees of fibrosis and regeneration. Secondary or “reactive” hepatopathies occur due to a primary disease process elsewhere in the body, often in the gastrointestinal tract (GIT), that causes inflammation in the liver without necrosis and fibrosis. This is a very important consideration when interpreting liver biopsies because the primary disease needs to be investigated and addressed. We will discuss primary chronic hepatitis in dogs (CH).

## ETIOLOGY

Although there is evidence of infectious, metabolic, toxic, and immune causes of CH, most cases are idiopathic (Table 1).

### Infectious Causes

In contrast to human medicine, there is no strong evidence of a viral etiology in canine CH. Leptospirosis causes acute hepatitis but can also induce a chronic pyogranulomatous response. *Ehrlichia canis* has been associated with CH, and experimentally infection with *Anaplasmosis spp.* can cause subacute hepatitis. Multiple other systemic diseases can have hepatic involvement, but lesions are typically acute and necrotizing and part of a multisystemic disorder.

### Drugs and Toxins

Several drugs and toxins have been implicated in liver injury, including carprofen, oxi-bendazole, amiodarone, aflatoxin, and cycasin. Most often they cause acute hepatopathy, but in some instances CH or cirrhosis are potential sequelae. Strong

TABLE 1—ETIOLOGIC FACTORS IMPLICATED IN CH AND RELATIVE STRENGTH OF EVIDENCE BASED ON LITERATURE.

ETIOLOGY	SUBCATEGORY	EVIDENCE
Immune		Moderate-strong
Toxic	Copper	Strong
Metabolic	Protoporphyrria	Moderate (rare)
	Alpha-1-anti-trypsin	Weak
Infectious	Leptospirosis	Moderate
	Leishmaniasis	Moderate-strong
	Rickettsial	Weak
	Mycobacteria	Moderate
	Histoplasmosis	Moderate
	Bartonella	Weak
	Protozoal (Neospora, Sarcocystis, Toxoplasma)	Moderate
	Viral	Negligible

evidence indicates that phenobarbital, primidone, phenytoin, and lomustine can result in CH. In humans, it is estimated that herbal and dietary supplements are responsible for up to 18% of drug-induced liver injury! Supplement toxicity is usually difficult to prove in veterinary medicine, but a complete drug history—including supplements—is vital.

The most common toxic injury causing CH in dogs is copper-associated CH; therefore, every liver biopsy should be evaluated for abnormal hepatic copper content. Altered hepatic copper excretion in bile, excessive dietary intake, or both are suspected. The panel believes that current dietary guidelines (no maximum limit for dietary copper), along with a change to more bioavailable Cu premixes in the 1990s, are linked to increased hepatic Cu accumulation in dogs as noted in multiple studies.

### Metabolic conditions

Alpha-1 antitrypsin (AAT) deficiency, caused by abnormal hepatic processing of AAT, results in hepatocyte retention of abnormally folded proteins causing CH in American and English

cocker spaniels. It is unknown whether accumulation of hepatic AAT causes liver disease or merely reflects liver injury.

### Immune-mediated CH

In humans, the diagnosis of autoimmune hepatitis relies on several criteria, including serum markers (enzymes, IgG, and antinuclear, anti-mitochondrial, anti-liver and kidney microsomal antibodies), exclusion of other causes, typical histology, and response to immunosuppressive treatment. It is thought to occur in genetically predisposed individuals exposed to certain triggers (pathogens, drugs, vaccinations, toxins, or GI microbiome changes).

Based on available veterinary studies, an immune basis for CH is suggested by several criteria (lymphocytic infiltrate, abnormal expression of MHC class II, positive serum autoantibodies, familial history, and female predisposition). Presumptive clinical diagnosis of immune-mediated CH requires elimination of other etiologies and a favorable response to immunosuppressive treatment.

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## SIGNALMENT

There is strong evidence for an increased prevalence of CH in Bedlington terriers, Doberman pinschers, Labrador retrievers, dalmatians, American and English cocker spaniels, English springer spaniels, West Highland white terriers, and standard poodles. The overall mean age of onset of clinical signs is 7.2 years.

## CLINICAL PATHOLOGY

Persistent (> 2 months) unexplained increases in ALT with or without other laboratory changes is the best screening test currently available for early detection of CH.

If both ALT and ALP are increased, the magnitude of ALT increase often exceeds that of ALP. There is a long subclinical phase when diagnosis should be pursued with the best chance for intervention. Once overt signs develop, they often represent complications of late-stage disease with poor prognosis (portal hypertension, ascites, HE, coagulation disorders, infection, and gastroduodenal ulceration).

Hyperbilirubinemia is reported in 50% of dogs with CH and is a negative prognostic indicator. Hypoalbuminemia is a late marker of hepatic synthetic failure. Decreased BUN and cholesterol develop in approximately 40% of dogs with CH, usually once cirrhosis develops. Hypoglycemia is more often associated with acute liver failure. Serum bile acids are the most sensitive hepatic function test. However, they are not sensitive for early stages of CH and should not be used as the basis for deciding to pursue liver biopsy.

## IMAGING

Abdominal ultrasound is the most useful and informative imaging modality for dogs with suspected chronic hepatitis (CH), but is highly operator dependent, its sensitivity is low, and no changes are pathognomonic or diagnostic for CH.

## BIOPSY

The primary concern for any hepatic sampling is hemorrhage. Published studies, including a heterogeneous group of hepatic disorders, indicate a relatively low incidence of bleeding complications of 1.2-3.3%. Tests used to assess risk of hemorrhage include PCV, platelet count, PT, aPTT, fibrinogen, BMBT, and vWF in predisposed breeds. High-risk dogs (PCV < 30%, Platelets < 50,000, either PT or aPTT > 1.5 x upper limit, fibrinogen < 100 mg/dl, BMBT > 5 min, vWF < 50%) should have laparoscopic liver biopsy where tissue injury is

minor compared to surgery and hemostasis can be more tightly controlled compared to ultrasound-guided needle biopsy. Patients should be hospitalized overnight after a liver biopsy to monitor for hemorrhage or other complications. There is not enough evidence to recommend routine prophylaxis with fresh frozen plasma, other blood products, or vitamin K, and their use should be considered on a case-by-case basis.

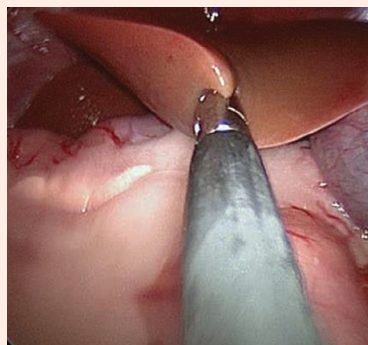
Fine needle aspirates have no role in the definitive diagnosis of CH, because they often miss inflammatory infiltrates, extent of fibrosis, or abnormal copper accumulation.

Laparotomy is indicated if there is a concern for extra-hepatic biliary duct obstruction (EHBO), severe gallbladder pathology, or a vascular anomaly.

Laparoscopy is the method of choice for liver biopsy in dogs with suspected CH, as this minimally invasive method enables gross evaluation of the liver, extra-hepatic biliary system, and safe acquisition of large targeted biopsies from multiple liver lobes (Figure 1). A minimum of five biopsies from at least two liver lobes should be obtained for histopathology (3), aerobic/anaerobic culture (1), and quantitative copper analysis (1). Ultrasound-guided hepatic biopsy is least invasive, but small sample size frequently compromises diagnosis.

FIGURE 1

▲ Laparoscopic liver biopsy in a 2-year-old male castrated Havanese (McDevitt: Short-term clinical outcome of laparoscopic liver biopsy in dogs: 106 cases; JAVMA 248, No. 1, January 2016).



## TREATMENT

If thorough diagnostic investigation fails to find an etiology, then treatment with nonspecific hepatoprotective agents such as ursodeoxycholic acid and S-adenosylmethionine may be indicated. Beneficial effects of Silymarin were

not proven in human studies; therefore, it is not recommended. There is limited evidence of the efficacy of vitamin E in CH in dogs.

Any increase in hepatic copper should be treated with D-penicillamine (the chelator of choice) and a copper-restricted diet, likely for months to years.

Studies support the existence of a subset of dogs with CH that respond to immunosuppressive treatments. However, not enough evidence is available to recommend an optimal immunosuppressive protocol. Corticosteroids are efficacious as a first-line treatment, but carry many side effects problematic in dogs with advanced liver injury (sodium and water retention that provoke ascites, catabolism, risk for enteric ulceration precipitating hepatic encephalopathy, hypercoagulability). Some panel members combine corticosteroids with azathioprine or cyclosporine to enable more rapid tapering of steroids to every other day anti-inflammatory doses. For most experts, maintenance on the second drug alone was the goal. Some experts use single agent cyclosporine twice daily as first-line treatment to avoid the adverse effects of corticosteroids. My personal experience with cyclosporine (Atopica) for treatment of CH has been excellent. Mycophenolate also has been used by panel members as a first- or second-line treatment and in combination with steroids. The length of time to remission and whether or not lifelong maintenance therapy is necessary are undefined.

## PROGNOSIS

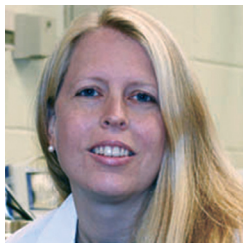
Dogs with CH do not typically go into spontaneous remission, and there is a large amount of evidence that once diagnosed, histological lesions of CH progress and many dogs die from causes related to their hepatic disease. In multiple studies, mean survival time was 561 ± 268 days. In dogs with cirrhosis, survival is considerably shorter (23 ± 23 days). Factors associated with poor prognosis include hyperbilirubinemia, prolonged PT and aPTT, hypoalbuminemia, the presence of ascites, and the degree of fibrosis on biopsy.

## TAKE-HOME POINTS

*Many dogs have an increase in ALT. When should I be worried?*

- Predisposed breeds
- Progressive increase during serial evaluations
- ALT greater than three times the upper limit of normal
- Any increase in bilirubin





## Idiopathic Chylothorax in the Dog

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**C**hylothorax, the accumulation of chylous fluid in the thoracic cavity, is a rare disorder that is seen in both dogs and cats. In most cases, there is no underlying etiology, and it is considered idiopathic. Other causes of chylothorax have been reported, including right-sided heart failure, heartworm disease, vena caval obstruction, lung lobe torsion, and neoplasia.<sup>1</sup> Afghan hounds, Shiba Inus and Oriental breeds of cats are predisposed to developing chylothorax.<sup>1,2</sup>

The lymphatic system is a network of permeable capillaries which allows extravascular protein, tissue fluid, and foreign particles to be returned to the circulatory system via ducts that empty into the jugular or precaval vein. The pelvic limbs, via the lumbar lymph trunks, and abdominal viscera, via the intestinal trunk, drain into the cisterna chyli. The cisterna chyli is an elongated sac that runs from the fourth to the first lumbar vertebrae, dorsal to the aorta.<sup>3</sup> The cisterna chyli narrows cranially at the diaphragm and continues into the thorax as the thoracic duct. In dogs, the thoracic duct courses on the right side, dorsal to the aorta, before crossing over to the left side around the fifth thoracic vertebra, and terminates at the left jugular vein and cranial vena cava, although there is some variation to its termination.<sup>3</sup> The thoracic duct is not always a single structure as multiple collaterals may be present in both the caudal and mid thorax.<sup>3</sup> During digestion, triglycerides are transported through this lymphatic system within chylomicrons to be released into the venous circulation. When disruption of lymphatic drainage occurs, there can be an accumulation of chyle in the thoracic cavity resulting in a chylothorax. Altered pressure within the lymphatic system is thought to allow transmural leakage of chyle.<sup>4,5</sup> Although traumatic disruption to the thoracic duct can cause chylothorax, when the thoracic duct is experimentally lacerated or transected, the resulting chylothorax has been shown to resolve in five to 10 days.<sup>6</sup> Experimental obstruction of the thoracic duct rarely results in chylothorax, while ligation of the cranial vena cava causes chylothorax in more than half of the dogs.<sup>7</sup>

Chyle is milky fluid which has a triglyceride level that is higher than serum, often 10 to 100x

FIGURE 1

⌵ Thoracic radiographs showing pleural effusion in a dog with a chylothorax.

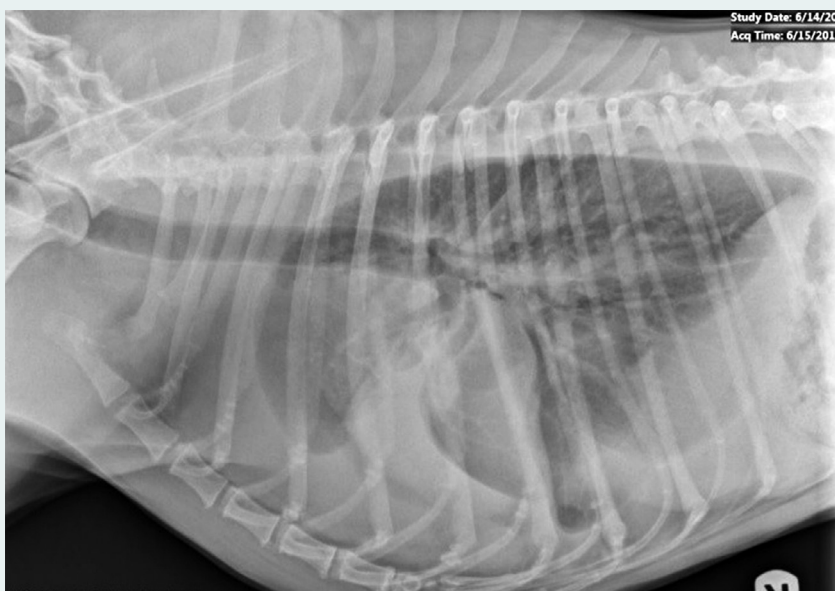
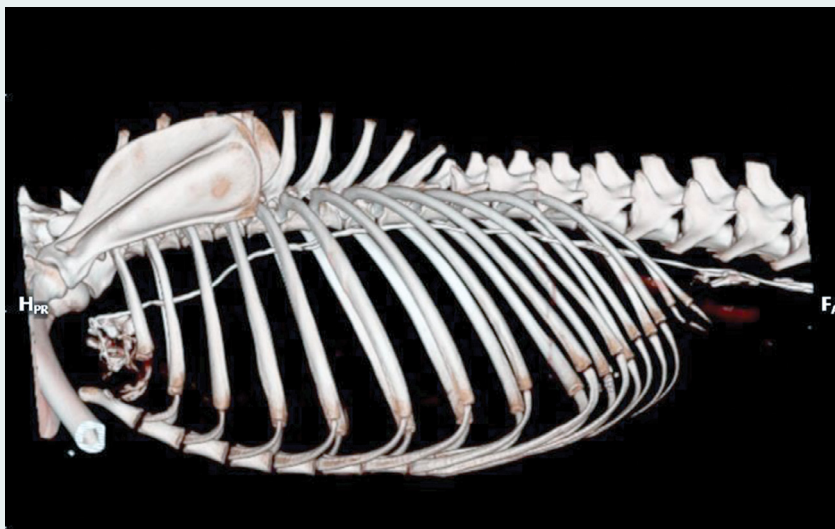


FIGURE 2

⌵ Lymphangiogram showing the thoracic duct running just below the spine and leaking in the cranial thorax.





## SURGERY

## Idiopathic Chylothorax in the Dog

CONTINUED FROM PAGE 8

greater, and a cholesterol level that is less than serum.<sup>8</sup> It is a modified transudate that contains mostly small and large lymphocytes. Upon standing, chyle retains its milky appearance, although it may have a milky top layer.<sup>8</sup> If the patient is anorexic, the fluid will not be as milky or may appear serosanguinous.<sup>9</sup> Accumulation of chyle within the thorax can have devastating results. Patients often present with dyspnea, and radiographs reveal pleural effusion and thoracocentesis reveals chyle.

Medical management with repeated thoracocentesis is the initial treatment for chylothorax but is not a long-term solution. Fibrin development can result in restrictive pleuritis and adhesions, and the effusion often becomes pocketed and harder to fully drain.<sup>9,10,11</sup> The lungs become less tolerant of accidental laceration and pneumothorax can result. Drainage of this fluid can lead to malnutrition because of the loss of lipid and protein. Hypoproteinemia can result as well as dehydration and electrolyte imbalance, including fat-soluble vitamins such as vitamin K.<sup>9,12</sup> Initial conservative therapy for 10 days is reasonable to see if the fluid diminishes or resolves. If the patient requires infrequent (every few months) thoracocentesis, and there does not appear to be a large amount of restrictive disease, conservative therapy can be continued. Low-fat diets can decrease the triglyceride content of the chyle but will not decrease the volume.<sup>10</sup> Rutin, a benzopyrone, has been recommended, but the reported benefit is slight.<sup>13</sup> If frequent taps are

required with no decrease in the amount of fluid produced, surgery should be recommended.

Surgery for chylothorax is not always successful but gives the best chance to resolve the effusion. Multiple procedures have been described for chylothorax, including omentalization and pleuroperitoneal shunts, but the most commonly performed and most successful treatment is thoracic duct ligation (TDL) which is often combined with a subtotal pericardectomy and cisterna chyli ablation (CCA).<sup>14-19</sup> Prior to surgery, a CT lymphangiogram is helpful to look for branches of the thoracic duct.<sup>20</sup> This can be achieved by injecting iohexol into a popliteal lymph node. Contrast will appear in the thoracic duct within two to 13 minutes.<sup>20</sup> The thoracic duct is seen as it runs along the dorsal surface of the aorta, and branches can be identified and surgery can be planned.

At surgery, the thoracic duct and its branches may be difficult to identify as it is typically clear. Feeding cream prior to surgery increases the fat content of the chyle and will make the thoracic duct appear white, which can help in identification. Injection of the popliteal or mesenteric lymph nodes at surgery with a small volume of methylene blue will highlight the thoracic duct within 10 minutes making identification simpler when compared to surrounding structures.<sup>21</sup> The blue color will persist for up to 60 minutes. Methylene blue can be diluted prior to injection. Traditional surgery involves ligating the thoracic duct through a

lateral thoracotomy at the 10th intercostal space. The duct can be ligated en bloc with suture, with surgical clips, or with devices like the ultrasonically activated shears or Ligasure™.<sup>22,23</sup> More recently, thoracoscopic TDL has gained popularity.<sup>24,25</sup> Thoracoscopic TDL is achieved with the patient in sternal recumbency, and three ports are placed in the caudal thorax for the camera and instruments.<sup>24</sup> Dissection dorsal to the aorta reveals the thoracic duct, which is ligated with endoscopic clips or a sealing device. Pericardectomy can also be performed with minimally invasive techniques.<sup>17,25</sup>

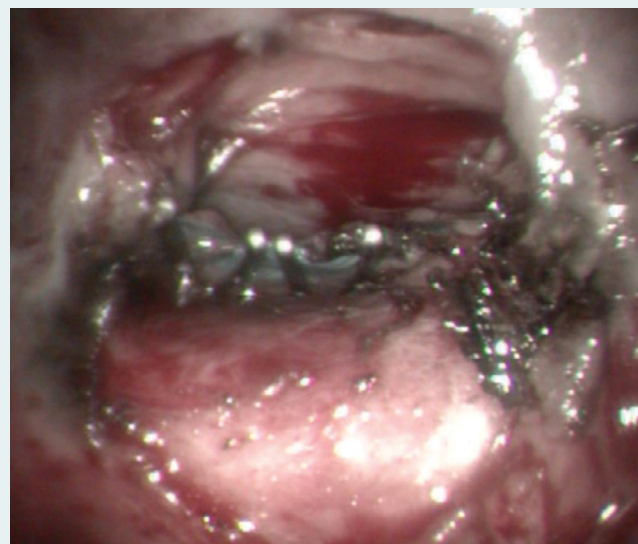
Postoperative care of dogs with chylothorax is similar to other thoracic procedures. A chest tube is left in place for 24 to 48 hours and fluid production monitored. After that, the chest tube is removed and the patient discharged on a low-fat diet. Recheck thoracic radiographs are taken one to two weeks after surgery to assess for recurrence of pleural effusion. If no effusion is present, the patient is monitored regularly for recurrence. If there is continued effusion, treatment with anti-inflammatory doses of steroids and placement of a pleural port can be considered.<sup>27,28</sup>

TD ligation was reported to have about a 50% success rate in dogs.<sup>1,9,11,26</sup> The combination of thoracic duct ligation and pericardectomy has a reported success rate of 60-85% in dogs.<sup>2,3,14,17-19</sup> In addition to the TD ligation and pericardectomy, CCA is often performed. CCA

(CONTINUED ON PAGE 10)

## FIGURES 3 + 4

☐ Intraoperative injection of a mesenteric LN with methylene blue will highlight the TD and branches can be seen. The photo on the left shows the thoracic duct above the aorta. The second photo shows multiple surgical clips occluding the thoracic duct.



## SURGERY

## Idiopathic Chylothorax in the Dog

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allows drainage of lymphatic fluid into the abdomen and prevents pressure buildup in the thoracic duct after ligation, which may lead to collateral ducts being formed or opened.<sup>29</sup> TDL and CCA has shown similar success to TDL and pericardectomy with 83% of dogs having resolution of their chylothorax.<sup>2</sup> At Angell Animal Medical Center, we currently perform TDL, CCA, and pericardectomy for dogs with chylothorax. Success rate with all three procedures performed simultaneously has not yet been reported in dogs. Although chylothorax is a devastating condition, improvement in surgical techniques and the use of minimally invasive surgery have resulted in less morbidity and a better success rate in dogs.

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## Addison's Disease

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**I**t's a Saturday night a week before Christmas and a 4-year-old, female, spayed Labradoodle presents to your emergency room for a one-day history of vomiting and decreased appetite. Her owner is also worried that she seems weak and is not acting like herself. As you go up front to triage her, what differentials are running through your mind? Maybe she ate a stocking or holiday decoration that is causing an intestinal obstruction. It could be that she got into the trash and ate cooking leftovers, and she is starting to brew a case of pancreatitis. However, endocrine disease is a sometimes-overlooked differential, and this patient's history contains classic markers of Addison's disease, or hypoadrenocorticism.

Addison's disease results from a lack of mineralocorticoids and glucocorticoids. These substances are produced by the adrenal glands. The most common type of hypoadrenocorticism is caused by immune-mediated destruction of the adrenal cortices. This disease can also be iatrogenic (caused by a veterinarian) in cases where Cushingoid dogs have been given medications to destroy portions of their adrenal glands.

The most common signalment for this disease is a young to middle-aged female dog, although it can appear at any age and in male dogs as well as females. Besides poodles, other breeds that are predisposed include Wheaten terriers, Portuguese water dogs, Great Danes, West Highland white terriers, and Rottweilers. However, many dogs affected by Addison's disease are of mixed breeding. It can also rarely occur in cats.

When dogs with Addison's disease present to the emergency hospital, it is typically because of an "Addisonian crisis." This often includes severe gastrointestinal distress with vomiting, bloody diarrhea, and signs of hypovolemic shock. There are many other ailments that can cause these signs, so it is important to obtain point-of-care bloodwork as a part of a minimum database in these patients.

Clinicopathological abnormalities in dogs with hypoadrenocorticism are attributable to the



body's inability to produce sufficient glucocorticoids and mineralocorticoids. The most common findings include low sodium and high potassium, with a sodium to potassium ratio of less than 28:1. Other chemistry panel findings can help confirm the diagnosis, especially in light of a low sodium to potassium ratio. These include azotemia (pre-renal secondary to dehydration) and often hypoglycemia. Urinalysis frequently reveals a low urine-specific gravity, especially noteworthy in light of dehydration and azotemia, as a result of the body's inability to concentrate urine due to inadequate sodium retention.

The CBC can also be supportive of Addison's disease in several ways. Addisonian patients are often anemic. Due to the body's inability to mount an appropriate stress response in the face of illness, many will lack a "stress leukogram" that is typically seen in ill animals. Rather than exhibiting neutrophilia and lymphopenia, an Addisonian dog may have a normal leukogram or even a reverse stress leukogram, with neutropenia and lymphocytosis.

Treatment of Addison's disease in the emergency setting initially involves aggressive intravenous fluid therapy to address hypovolemia and electrolyte abnormalities. Prompt diagnosis is important, however, because hormone replacement therapy should begin as soon as possible. Definitive diagnosis is made via the ACTH stimulation test; ACTH is the substance that regulates the release of cortisol in the body. In a normal dog, exogenous administration of this compound will cause a significant increase in blood cortisol. In a dog with Addison's disease, the cortisol level will not increase after ACTH administration because the body is unable to produce cortisol.

Once diagnosis is confirmed, treatment is aimed at replacing both mineralocorticoids (usually this takes the form of a once-a-month injection) as well as glucocorticoids, typically given daily in the form of prednisone. Dogs who are promptly diagnosed and aggressively treated in an Addisonian crisis, and who are placed on an appropriate long-term monitoring and therapeutic regimen, typically do very well and





# Canine and Feline Extractions and Dealing with Common Complications

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**T**eeth are extracted for a number of reasons in our canine and feline patients. One of the most common reasons to perform extractions is advanced periodontal disease. Multiple studies have shown that periodontal disease has a prevalence of 80-85% in dogs and cats over 2 years of age.<sup>1,2</sup> Early or mild periodontal disease does not always necessitate extraction of the affected teeth, but advanced or end-stage periodontal disease often requires extraction for treatment.<sup>1,3</sup>

Persistent deciduous teeth are found frequently in toy/small-breed dogs and should be addressed when the dogs are still young. Left in place, the continued presence of deciduous teeth can cause irreversible periodontal disease of the adjacent permanent tooth. Deciduous teeth should exfoliate once the permanent successive tooth has fully erupted; if they are still present, they should be removed.<sup>1</sup> Often timing of removal coincides with spay/neuter surgery as most of the adult dentition should erupt by the age of 5-7 months.<sup>1,3</sup>

Fractured teeth are seen with an incidence of nearly 25% in our canine patients.<sup>2</sup> Unfortunately, many of these fractures are caused by inappropriate chew items such as Nylabones, antlers, and marrow bones.<sup>4</sup> Root canal treatment is an option for complicated crown fractures, while extractions are often required for complicated crown root fractures or long-standing complicated crown fractures.<sup>4,5</sup>

Malocclusion is another frequent finding in our toy/small breed and some purebred dogs. While orthodontic appliances can resolve the issue, selective extraction can also be successful. The goal is to provide a pain-free functional occlusion.<sup>1</sup> Crowding/supernumary teeth can also be treated with selective extraction. This gives the remaining teeth room for a healthy periodontium.

Any “missing” teeth should be investigated in dogs and cats. A great time to investigate is during a spay/neuter as the adult dentition should be erupted or in the process of erupting by 5-7 months.<sup>1</sup> Any unerupted or impacted teeth can be treated at that time. Bony impaction will require extraction, while soft tissue impaction can be treated with an operculectomy to allow

FIGURE 1

➤ This is an example of a complicated crown root fracture of the right maxillary 4th premolar tooth, commonly called a “slab” fracture.



the tooth to continue its normal eruption.<sup>1</sup> Addressing these teeth at a younger age can prevent the development of dentigerous cysts, which can cause significant bone loss, damage to nearby structures, and potential jaw fractures.<sup>1</sup>

Cats (and some dogs) often present with tooth resorption. Unfortunately, there is no way to stop or reverse the tooth resorption process. Tooth resorption in cats can be classified as Type 1, 2, or 3.<sup>6</sup> Type 1 tooth resorption in cats requires extraction of all remaining tooth structures, while Type 2 tooth resorption in cats can be treated with crown amputation. Type 3 tooth resorption occurs when one tooth is affected by both Types 1 and 2 tooth resorption, necessitating different approaches to different regions of the tooth. Intraoral radiographs must be obtained prior to treatment to determine the type and extent (or stage) of tooth resorption.<sup>6</sup> Dogs can also present with tooth resorption, but theirs is not classified as Type 1, 2, and 3. The most common type of tooth resorption seen in dogs is external replacement resorption, which resembles Type 2 tooth resorption in cats.<sup>7,8,9</sup> This type does not always require treatment

unless it involves the crown or has severely damaged the integrity of the tooth.<sup>7,8,9</sup> Like Type 2 in cats, this type of tooth resorption may be treated with crown amputation when indicated.<sup>7,8,9</sup> The second most common type of resorption found in dogs is external inflammatory resorption, often seen in relation to tooth root abscesses due to endodontic disease and/or advanced periodontal disease.<sup>7,8,9</sup> These teeth require extraction due to the periapical lucencies indicative of endodontic disease.<sup>7,8</sup>

Feline chronic gingivostomatitis is an uncommon but debilitating disease affecting our feline patients. Total or caudal mouth extraction procedures do not always provide an immediate cure, but they are recommended as the best chance for a cure.<sup>10</sup> The majority of cats do improve with about 30% having refractory disease and requiring medication for a period of time.<sup>10,11</sup> Studies have found cyclosporine to be a good adjunct treatment after caudal/partial/total mouth extractions are performed.<sup>10</sup>

Prior to performing extractions, be sure to get permission from the client. Estimates should be

## DENTISTRY

## Canine and Feline Extractions and Dealing with Common Complications

CONTINUED FROM PAGE 12

provided with a range that allows for multiple extractions. Often the extent of treatment needed cannot be appreciated until an examination, charting, and intraoral radiographs are performed under general anesthesia. Have a plan in place after obtaining all the pertinent information. Does the owner need to be called and informed of all the findings or only unexpected findings? What if the owner can't be reached? Would the owner prefer you to extract all the necessary teeth or just perform a complete dental prophylaxis and return another day for extractions? Some owners become very upset if teeth are extracted without permission. This is often due to a lack of understanding about the disease process...or cost. What happens if the required work will go over the high end of your estimate?

Local anesthetic blocks are an important part of dentistry and oral surgery; they help prevent windup, decrease anesthetic requirements, and provide postoperative pain control.<sup>1,3</sup> Lidocaine and Bupivacaine are common local anesthetics used in our canine and feline patients. These can be purchased as formulations with vasoconstrictors or they can be mixed with opioids.<sup>1,3</sup> Do not exceed 4 mg/kg of Lidocaine or 2 mg/kg of Bupivacaine for dogs and cats.<sup>3</sup> The volume of local anesthetic also needs to be taken into consideration.<sup>12,13</sup> Larger volumes can be given per site in larger dogs and cats (see charts in the slides that follow).<sup>12,13</sup> Commonly performed local anesthetic blocks include the infraorbital, caudal maxillary, rostral mandibular (mental), and caudal mandibular (inferior alveolar).<sup>1,3</sup> Specific landmarks and guidelines will be discussed during the presentation.

Equipping the dental operator with proper equipment and instruments is imperative for success. Recommended supplies include: high-speed delivery unit with integrated water coolant, burs for high-speed handpiece (1/2, 1, 2, 4, 700, 701, 701L, cylindrical diamond, conical white stone), scalpel blades (#11, #15, #15C), periosteal elevators (molt #2, molt #4, freer), dental elevators (winged elevators #1-4), luxators in various sizes, extraction forceps, small alveolar curette, tissue forceps: Brown-Adson 7X7 tooth, Olsen-Hegar needle holders, scissors (Dean), root tip picks, dental radiographs (mandatory), excellent lighting, suture (Monocryl in 3-0 to 5-0 sizes, with reverse cutting or tapered needles), and Gel-foam of Vetspon for hemostasis.<sup>1,3</sup>

Dental extractions can be divided into two major categories: "simple/closed" versus "surgical" extractions.<sup>1,3</sup> Simple extractions do not require removal of alveolar bone or sectioning of multirooted teeth, while surgical extractions do. Mucoperiosteal flaps are created for surgical extractions, and they should be closed without tension.<sup>1,3</sup> Absorbable sutures are recommended for extraction site closure, using a simple interrupted suture pattern.<sup>1,3</sup>

Extraction complications can occur for a number of reasons; the best approach for handling complications is prevention.<sup>1,3</sup> Preventive measures include preoperative intraoral radiographs, proper technique, good lighting, +/- magnification, postoperative radiographs, and occasional intraoperative radiographs.<sup>14,15</sup> Proper technique includes using the correct instruments as intended and technique improves with appropriate training.<sup>14,15,16</sup> Common complications include retained tooth roots, pushing tooth roots into the nasal cavity/mandibular canal, orbital penetration, hemorrhage, and dehiscence.<sup>14,15</sup> Each of these complications will be discussed in today's presentation in addition to their prevention and treatment. Remember to ask for help, be honest, and learn from your mistakes.

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# Patellar Groove Replacement

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**C**anine patellar luxation is an extremely common orthopedic condition that, at the time of corrective surgery, necessitates evaluation of the patellar groove with respect to alignment, depth, shape, and cartilage quality. For the most part, groove abnormalities can be addressed with either a wedge recession or block trochleoplasty; however, there are occasions in which the patella groove is so severely deformed or damaged that the surgeon is forced to perform a sulcoplasty, burring or rasping away what cartilage exists to expose bleeding subchondral bone in order to create a faux groove to “capture” the patella. Sulcoplasty is painful, results in a slower return to function, and, compared to trochleoplasty, ensures suboptimal development of fibro-cartilage over a larger surface area.

An appropriately sized, anatomically shaped, low-friction prosthesis, patellar groove replacement (PGR, KYON) offers the surgeon an alternative to sulcoplasty in cases of severe femoro-patellar osteoarthritis. Dogs should be greater than six months of age, and trochlear deformities can be addressed secondary to either lateral or medial patellar luxation. Careful

preoperative radiographic planning is essential in order to choose the correct size of prosthesis.

The stifle joint is approached routinely, the patella luxated, and the groove exposed. An osteotomy to remove the diseased groove is performed using an oscillating saw along a line from the origin of the long digital extensor tendon to the proximal margin of the trochlear groove (Figure 1). This will leave a flat, smooth, bony bed (Figure 2) on which to secure an implant base plate (Figure 3). Screws secure the plate in the appropriate position. The prosthesis has three prongs that engage the plate before being carefully and gently hammered into the locked position (Figures 4 & 5). Prior to final prosthesis choice and placement, use of trial implants, together with patella reduction and stifle flexion/extension, is vital to ensure the correct size, a “snug” relationship between patella and implant, smooth tracking, and absence of a click or hitch as the patella glides.

It is important to note that the PGR is not, in itself, a cure for patella luxation. All the other standard concurrent treatments — joint capsule release/imbrication, tibial tuberosity transposition, distal

femoral or proximal tibial corrective osteotomies — are still essential to a positive outcome.

One of the advantages to PGR over sulcoplasty is the lack of innervation to a prosthesis, with owners noting increased comfort and earlier return to function. KYON suggests a two-week period of postoperative restriction, introduction of five-minute leash walks at week three, and return to normal by weeks eight to ten. Radiographic follow-up to ensure stable implants can be useful at this time; however, bone ingrowth into the base plate has been reported to take nine months.

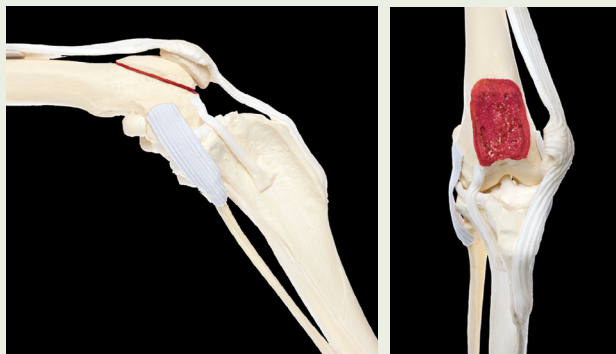
## REFERENCE:

Patellar groove replacement in patellar luxation with severe femoro-patellar osteoarthritis. Z. Dokic, D. Lorinson, JP Weigel, A Vezzoni. *Vet Comp Orthop Traumatol* 2/2015; 124-130.

All figures are courtesy of KYON ([www.kyon.ch](http://www.kyon.ch)).

### FIGURES 1 & 2

❏ (Left) Bone model, lateral position, red-inked line delineating the axis of a required osteotomy from the origin of the long digital extensor tendon to the proximal margin of the trochlear groove. (Right) Bone model, A/P position, representing the appearance of a smooth flat bone surface required for bone plate application.



### FIGURES 3, 4, & 5

❏ (Left) Base plate screwed into the appropriate position. (Middle) Bone model, A/P position, demonstrating PGR (patella luxated). (Right) Bone model, lateral position, demonstrating PGR (patella reduced)





## ➤ Angell Offers Two Low-Cost Clinic Locations

Angell at Essex (Danvers, MA) and Angell at Nashoba (Westford, MA) clinics are dedicated to providing quality care to the general public as well as offering deeply discounted services for qualified low-income families. The clinics provide primary veterinary care, spay and neuter services, vaccinations, and surgery and dental services.

Angell at Essex and Angell at Nashoba are open weekdays from 7:45am – 4:00pm throughout the year. The clinics do not provide overnight care, specialty service care, or 24/7 emergency care. Appropriate cases will be referred to Angell's Boston or Waltham facilities or a surrounding specialty veterinary referral hospital.

### To Financially Qualify for Discounted Services

Angell clinics welcome all pet owners and reserve a sizable portion of appointment times for discounted services. For those interested in discounted services, clients must financially qualify by presenting a photo ID and one of the following:

- Women, Infants, and Children (WIC) Program card
- Supplemental Nutrition Assistance Program (SNAP) card (formerly known as Food Stamps/EBT)
- Spay and Neuter Assistance Program certificate
- Letter/lease from the owner's local housing authority showing that the owner is a participant in public housing
- Veterans card

### **angell at nashoba**

To schedule an appointment with the Angell at Nashoba clinic, please call **978-577-5992**.



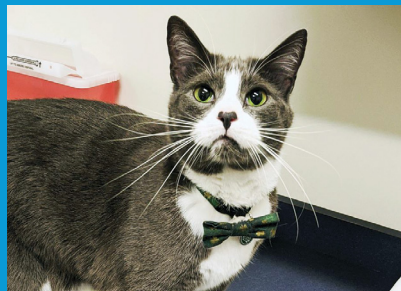
Left to Right: Dr. Laurence Sawyer. Angell at Nashoba on campus at Nashoba Valley Technical High School (Westford, MA).

### **angell at essex**

To schedule an appointment with the Angell at Essex clinic, please call **978-304-4648**.



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



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

Main Phone: 617-522-7282 (Boston) | 781-902-8400 (Waltham) | Veterinary Referrals: 617-522-5011 | Angell at Nashoba: 978-577-5992

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





## ➤ Courtesy Shuttle for Patients Needing Further Specialized Care

Angell Animal Medical Center offers the convenience of our MSPCA-Angell West facility in Waltham, MA. With 24/7 Emergency and Critical Care service and two board-certified criticalists on staff, the Waltham facility also offers specialized service appointments Monday through Saturday. If needed, an oxygen-equipped courtesy shuttle can transport animals to Boston for further specialized care and then take them back 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.

## ➤ Physical Rehabilitation at MSPCA-Angell West

Canine and feline physical rehabilitation is used to treat a wide variety of orthopedic and neurological conditions. Whether recovering from an injury, or cross training, or facing a mobility issue, dogs and cats can substantially benefit from physical rehabilitation.

MSPCA-Angell West Physical Rehabilitation offers services seven days a week. Jennifer Palmer, DVM, Certified Canine Rehabilitation Therapist (CCRT), and Amy Straut, DVM, CCRT, lead our Physical Rehab team. Visit [angell.org/rehab](http://angell.org/rehab) for details and video footage of the impact their work on our patients.

Currently physical rehabilitation services include:

- Hydrotherapy
- Land-based exercise
- Manual therapy
- Therapeutic laser treatment
- Massage
- Consultation and fitting of assistive devices
- Chiropractic







We mail one complimentary copy of our newsletter to each of our referring partners. Please circulate this copy within your practice.

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Please consider adding Angell's main numbers to your after-hours phone message.



## ➤ Angell Fall Continuing Education — **Registration is Open!**

Management of Ear Disease and Food Allergies —  
Everything You're Itching to Know

Wednesday, October 14, 2020  
6:15pm – 8:45pm  
2 Interactive CE Credits (pending RACE approval)

### TOPICS INCLUDE:

- Management of Ear Disease: Acute vs Chronic  
*Klaus Loft, DVM*
- When, Why, and How — Making the Most of the Diet Trial  
as a Diagnostic Test  
*Meagan Rock Painter, DVM, DACVD*

**JOIN US FOR AN ONLINE LIVE INTERACTIVE WEBINAR!**

To register for this online webinar, please visit [angell.org/ce](http://angell.org/ce)