

# Partners In Care

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## DERMATOLOGY



### Update in treatment and diagnosis of canine demodicosis

Klaus Loft, DVM

[angell.org/dermatology](http://angell.org/dermatology)  
[dermatology@angell.org](mailto:dermatology@angell.org)  
617 524-5733

Even since the large and exhaustive review published by Dr. Muller et al in early 2012, “Treatment of demodicosis in dogs: 2011 clinical practice guidelines,” there have been several new treatments and diagnostics introduced into our clinical reality, necessitating a short update on the diagnosis and treatment of canine demodicosis.

This update will start with a brief history and background on canine demodicosis, followed by a concise review of the latest published insight into the demodex mites. It will conclude with a review of current treatments.

Although they were historically described in 1842 as an obligate parasite of the pilosebaceous glands of humans, we consider the *demodex canis*, *d. injai*, and *d. cornei* to be part of the normal canine skin microbiome. Mites are transferred via maternal contact very early in life to the offspring, as offspring born via cesarean and kept isolated will have no demodex mites and the mite is not considered contagious. The different canine demodex mites are closely related, with no known cross-species zoonotic risk, but are, according to newer publications, genetically different. The full phylogenetic classification continues to evolve.

The proliferation of the mites in patients displaying dermatologic symptoms is thought to be a genetic and/or immunological issue, with lack of appropriate inflammatory response and disease control. The mites cannot survive *in vivo*,

(CONTINUED ON NEXT PAGE)

## DIAGNOSTIC IMAGING



### Clinical Radiation Safety

Steven Tsai, DVM, DACVR

[angell.org/diagnosticimaging](http://angell.org/diagnosticimaging)  
[diagnosticimaging@angell.org](mailto:diagnosticimaging@angell.org)  
617 541-5139

Diagnostic X-ray studies are ubiquitous in veterinary healthcare facilities, and with the rise of digital radiography, the ease of acquiring images is exponentially improved relative to the era of hard film, chemical developers, and dark rooms. With near-instantaneous availability of a review image following exposure, repeat exposures are easy and effectively free of cost. The hidden cost, however, is increased radiation exposure.

X-rays are high energy photons which can pass through materials and often collide with atoms in the body, potentially removing orbital electrons (ionization). Damage can affect DNA directly (less common) or indirectly through interaction with water and creation of free radicals (more common). While the cellular DNA repair mechanism is quite reliable, it is not perfect, and failure to repair DNA damage correctly can lead to mutation, which may lead to cancer.

Although the risk of developing cancer from a single X-ray exposure is small, current epidemiological data suggest that there is no minimum threshold below which there is zero risk of cancer. Additionally, this risk is considered to be cumulative over a person’s lifetime. A 2004 study estimated the attributable risk for a US resident of developing cancer due to medical X-ray exposure to be 0.9 percent, resulting in 5,695 cancer deaths per year.<sup>1</sup> Therefore, reducing radiation exposure as much as possible (As Low As

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FIGURE 1

↘ Demodex mite.



which complicates research and full understanding of the pathobiology of the mite. Each mammalian species has its own species of demodex mites, making it difficult to extrapolate findings.

Clinical symptoms arise when the patients experience an excessive proliferation of the mites in the hair follicles and sebaceous glands, resulting in an inflammatory skin disease. The inflammation is often complicated by secondary bacterial and/or yeast infections. In dogs, generalized demodicosis is differentiated clinically with the localized form, which is defined by having fewer than five sites affected. Demodicosis can be further subdivided based on age of onset: juvenile-onset (onset of symptoms under age 18 months) or adult-onset. The localized form resolves spontaneously in most cases, and treatment does not appear to accelerate the healing process. Based on the published “clinical practice guidelines,” treatment with a miticidal agent is not recommended in localized demodicosis, because it is possible that in a small number of cases, the localized demodicosis will progress into generalized demodicosis, and this development could be “hidden” if all localized patients are treated. Generalized canine demodicosis is considered hereditary and can become a very severe skin disease, often complicated by secondary bacterial and/or yeast infections. In very severe cases this can lead to sepsis and patient death. This generalized form of demodicosis will need to be treated intensively and appropriately. In several countries, the breeding of animals suffering from hereditary generalized demodicosis is not recommended and actually may be prohibited by national breeding associations.

**Diagnosis** of the mites is classically done via multiple deep skin scrapings from affected areas of skin (alopecic, seborrhea sicca/oleosa pustules and furuncles). A dulled scalpel or spatula with mineral oil can be used to collect skin scrapings from patients. The sample site should be squeezed to raise the mites closer to the skin surface and the

scraping made until capillary bleeding is noted on the scraped skin. The collected material is then microscopically examined at low magnification (4x to 10x) with the condenser lowered and the light source turned down. If done appropriately, the diagnosis is straightforward. Areas close to lip margins or eyes, webbed skin on paws, or areas with deep complicated pyoderma can be difficult to scrape. Plucking hair and a tape squeeze technique can be helpful in these harder-to-sample locations and will, if done well, yield a good diagnostic return.

**Treatment** of canine demodicosis is designed to resolve inflammation and thereby reduce any secondary skin infections and/or fur loss and ideally to remove the mites from the skin. The majority of localized cases will spontaneously resolve with no antiparasitic treatments needed. Generalized demodicosis is not necessarily a marker of general immunosuppression (Cushing’s syndrome, hypothyroidism, neoplastic malignancy, or immune suppressive treatments). In patients with demodicosis and a comorbidity of immune altering conditions, the resolution of the parasites will often be faster with fewer setbacks if the immune altering conditions can be controlled. The gold standard of treatment is to reach two negative skin scrapings from previously affected sites within a 30-day interval. Treatments should be tailored to fit the patient’s signalment and to avoid using protocols interfering with medications needed for any concurrent health issues (seizures, MDR-1 defect, or food adverse reaction). Many commonly used demodex treatments (e.g., Ivomec, Milbemycin) are not registered specifically for canine demodicosis by the FDA or the EPA. Several of the newer products have started to receive labeling for demodicosis (at least in Europe) or have been shown to have good antiparasitic effect in recent peer-reviewed publications.

**Amitraz (Mitaban®) dip:** Mitaban dip immersion bath is a FDA-approved drug for this disease, but toy and teacup breeds should not receive full treatment concentration. Dips are usually applied either weekly or every two weeks. Whole body clipping is often required for long-haired dogs throughout treatment, so that the dip solution can reach the mites down in the hair follicle. Side effects of Mitaban include sedation, decreased body temperature, loss of appetite, vomiting, and diarrhea, and the odor of the dips is not pleasant. Treatment with an antidote, Yohimbine, can be used to decrease the severity of some Mitaban dip side effects. Eye lubrication may be needed if significant conjunctivitis is a problem of treatment. Spot-on Amitraz products (e.g., Certifect®) used primarily for flea and tick prevention also have some reported effect on demodicosis, and an earlier similar product (Promeris) was discontinued after an association with outbreaks of pemphigus was shown to occur in a small number of patients treated.

**Lime sulfur dips** can be used for demodicosis, and are particularly effective in the earlier phases of treatment of very severe cases of generalized demodicosis with severe furunculosis forming deep pyoderma. Its low cost and minimal toxicity makes it a safe but foul-smelling option in very young animals and animals at risk for toxicity or side effects, as well as in cases with multi-ectoparasitic disease.

**Ivermectin (Ivomec®, Eqvalan®)** is available as an injectable liquid or oral paste as a deworming agent for production animals. It can be given orally daily as a liquid to dogs to treat demodicosis. Subcutaneous injections will often cause injection site discomfort and can result in sterile abscess formation after long-term use. Some dogs, especially herding breed dogs and

FIGURE 2

↘ Skin scraper spatulas



## DERMATOLOGY

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animals with drugs affecting the blood-brain barrier p-glycoprotein function, can have central nervous system sensitivity to the higher doses of ivermectin (MDR-1 or ABCB-1 gene defect) needed for demodex treatments. Signs of ivermectin sensitivity include sedation, drooling, loss of balance, vomiting, seizures, and, rarely, blindness. A genetic test is available to help determine if an individual dog may be sensitive to ivermectin. Dogs are first started on a low dose of ivermectin and then gradually given a higher dose while monitoring for side effects. Antifungal azoles such as fluconazole, itraconazole, and ketoconazole can affect the absorption, clearance, and metabolism of ivermectin.

**Doramectin® and moxidectin (Cydectin®, Advantage Multi®)** are available in combination as a liquid deworming (pour-on and injectable) agent for sheep and cows and as part of canine flea and heartworm preventive spot-on products. This agent can be given daily as an oral liquid to dogs to treat demodicosis, and is also available as part of a spot-on product (Advantage Multi®) for weekly application, but is registered for demodicosis only outside the US. This product can be used in ivermectin-sensitive breeds, as it is not a p-glycoprotein substrate, but other forms of neurotoxic-like side effects have anecdotally been reported with the use of the injectable form.

**Selamectin (Revolution®)** topically is less effective, although a few studies looking at oral use (evaporated on bread to avoid carrier vehicles) have shown some efficacy. Still, it is likely not the most practical option in clinical settings.

**Milbemycin oxime (Trifexis®, Sentinel®, and Interceptor®)** is available as a heartworm preventive pill for dogs. It can be given daily to dogs to treat demodicosis. Some breeds can have central nervous system sensitivity to high doses of milbemycin, but this is less common than a sensitivity to ivermectin. Signs of milbemycin sensitivity include sedation, drooling, loss of balance, vomiting, seizures, and rarely blindness. Cost will often become an issue.

**Fluralaner (Bravecto™, Nexgard®, and Simpirica®)** is a new class of antiparasitic drugs targeting fleas and ticks, but lately has been shown to have a promising effect on some forms of sarcoptic and demodectic mange in dogs. Depending on the product, the treatment will have to be repeated monthly or every 90 days until the two negative skin scrapings have been accomplished.

With more findings in PCR and DNA sequencing of mites as well as new treatments for canine demodicosis, the possibilities are promising from the perspective of clinicians. More options for

treatment will likely allow us to tailor the demodicosis treatments to high-risk patients—for example, those with known adverse reactions to antiparasitic classes of drugs or with complex health issues (seizure disorders, liver disease, etc.). We see this as a good opportunity to work with clients and hopefully breeders as well, so we can find ways to reduce the overall incidence of this disease in our pets.

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## DIAGNOSTIC IMAGING

CONTINUED FROM PAGE 1

Reasonably Achievable, or ALARA) is the guiding principle.

The three factors in minimizing radiation exposure to veterinary personnel are time, distance, and shielding.

**Time**—Reduce the amount of time exposed to radiation. Generally speaking, this means trying not to be in the room at all during exposure. Experienced radiology technicians generally achieve this by using either tape or sandbags to hold the patient in place while they run out of the room for the exposure. Judicious use of chemical sedation for X-ray studies also greatly improves ease of acquisition and reduces stress both for the patient and for the techs. In some states, it is actually illegal for a human to be in the room during X-ray exposure for a veterinary patient.

**Distance**—Radiation levels decrease with distance according to the inverse square law, which is to say it decreases exponentially as you move away from the source. Thus, even small increases in distance can result in significant drops in exposure. Generally speaking, exposure is considered to be effectively zero if you are >6 feet away from the X-ray source.

**Shielding**—If you must be in the room for exposure, lead shielding is imperative. At minimum, a lead apron, thyroid shield, and lead gloves should be worn. A common misconception is that adequate hand protection can be achieved by draping a lead glove over your hand while you directly grasp the patient's leg. This is not adequate protection, since the primary cause of radiation exposure to personnel is scatter (assuming the personnel are not within the primary X-ray beam), and the primary source of scatter is not the X-ray tube, but the patient itself! Since the patient is not 100 percent transparent to X-rays, some of the X-rays inevitably are scattered within the body and bounce around, sometimes exiting the body in a completely random direction. Effectively, the patient is “glowing” with scattered X-rays, so the hand holding the patient's limb is directly exposed to the X-ray “glow” coming off the patient (Figure 1). Thus, your hand should always be *inside* the lead glove, not just underneath it.

Tight collimation to the body part of interest also reduces scatter radiation, with the added bonus of improving image quality. Since X-rays pass through and are scattered by the materials they reach, a wide open beam results in a large amount of excess scattered radiation. If you imagine a flashlight pointing at a disco ball, a wide beam of light that hits the whole disco ball scatters a great deal of light throughout the room, while a tightly focused beam of light would not result in nearly as much scattered ambient light. Scattered X-rays are also captured by the digital detector and result

FIGURE 1

▶ **A.** Frosted glass ornament. **B.** Laser pointer aimed at the belly. Note that the entire ornament, from the ears to the distal extremities, glows with green light, even with just a pinpoint exposure to the primary beam. This is analogous to how a patient scatters the x-ray beam.

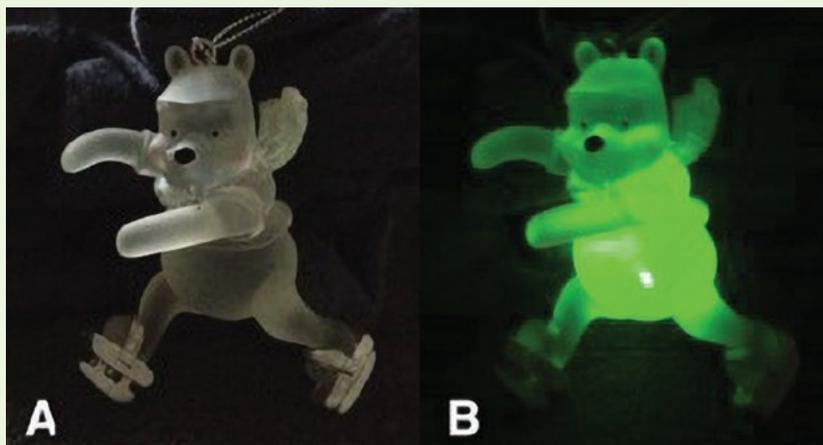
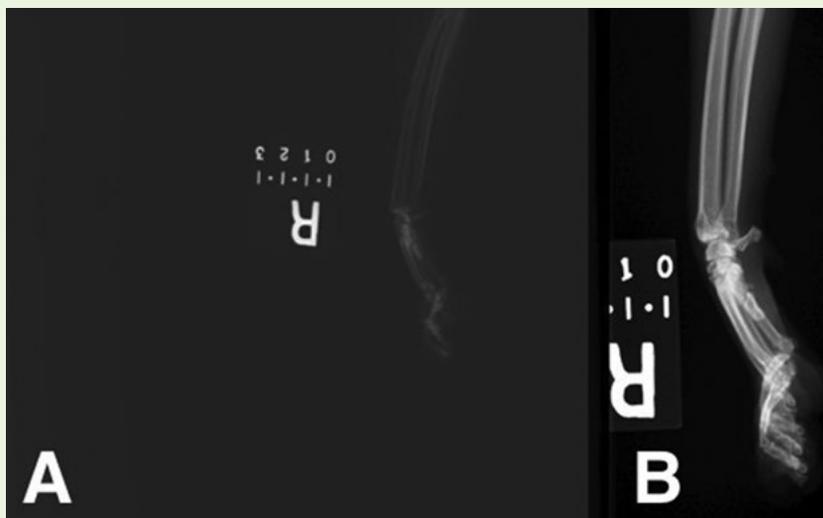


FIGURE 2

▶ **A.** Poor quality image of the antebrachium with wide-open collimation. The loss of detail is attributable almost entirely to scatter. **B.** Same patient, with same exposure parameters, but appropriate collimation.



in noise and blurriness (Figure 2). Thus, more focused collimation reduces room scatter and improves image quality.

Medical X-ray use has greatly improved our diagnostic capabilities over the past century, and is steadily increasing with the advent of digital radiography and spread of teleradiology. It is crucial that all veterinary healthcare personnel who are involved with ordering or performing X-ray studies be aware of the potential risks and

employ appropriate techniques to minimize radiation exposure.

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## ➤ Physical Rehabilitation Now Available at MSPCA-Angell West

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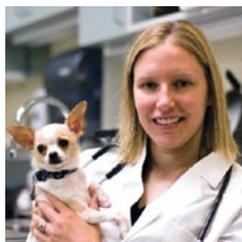
Open weekdays from 7:45am-4:00pm throughout the year, the clinic does not provide overnight care, specialty service care, nor 24/7 emergency service as Angell's Boston and Waltham facilities do, but will refer cases as appropriate to surrounding specialty veterinary referral hospitals.

To reach the clinic, please call **978 577-5992**. The clinic is located at: 100 Littleton Road, Westford, Massachusetts.

For more information, visit [angell.org/nashoba](http://angell.org/nashoba).



➤ Medical Director Dr. Laurence Sawyer and her team provide routine care to Ragdoll kittens.



## A Closer Look at Tissue Plasminogen Activator in Small Animals

Megan Whelan, DVM, DACVECC, CVA

angell.org/emergency | emergency@angell.org | 617 522-7282

**A**rterial thromboembolism (ATE) in cats can be a devastating disease with a poor outcome, often in seemingly “healthy” young cats. One study showed that among 250 cats with ATE that presented to general practices, there was a 61.2 percent euthanasia rate at presentation.<sup>1</sup> Cats are typically treated conservatively with supportive care including pain medication, nursing care, and treating the underlying cause. The pictures below show one ATE cat that was medically managed, and the end-stage damage of ischemic necrosis that can occur to the hind limbs.

Other interventions are being used in ATE cats, including human recombinant Tissue Plasminogen Activator (TPA) or Alteplase, which is a sterile lyophilized powder that should be kept refrigerated. This product is readily available, and contains an enzyme that has the property of fibrin-enhanced conversion of plasminogen to plasmin.<sup>2</sup> This conversion results in local fibrinolysis or clot breakdown.

There have been multiple studies in cats and dogs dating back decades, including one study

in 1992 describing the use of TPA for intraocular fibrinolysis in dogs.<sup>3</sup> The last prospective study of TPA use was in 11 cats with ATE. There were different dosing protocols; Group A received CRI of 5 mg IV of TPA over 4 hours, or an accelerated administration protocol; Group B received 1 mg TPA bolused IV, then 2.5 g IV over 30 minutes, then 1.5 mg IV over 1 hour. The investigator could also administer another 5 mg dose of TPA if no side effects were noted and there was no clinical improvement. The study was terminated due to high complication rates after administration of the drug. Adverse effects were seen in all 11 cats, and included azotemia (n=5), neurological signs (n=5), cardiac arrhythmias (n=5), hyperkalemia (n=4), acidosis (n=2), and sudden death (n=1). In this study, ATE cats were included if the thromboembolic event was within 12 hours.<sup>4</sup>

Since the prior 2010 study, Alteplase had been used in dogs and cats in various ways, including to improve catheter function in patients receiving extracorporeal renal replacement therapy.<sup>5</sup> There is also a case report using Alteplase intravesically in a dog to dissolve a urinary bladder clot.<sup>6</sup>

Each small vial of Alteplase is reconstituted with 2.2 mL of sterile water, and gently swirled, not shaken, until complete dissolution of the powder occurs. The cost per vial is about \$150 and the concentration is 1 mg/mL. This “clot-buster” drug has been used in people with central venous access devices to help restore catheter function and in cases of myocardial infarction within 6 hours of clinical signs. The reconstituted TPA is good for 8 hours, and has been frozen and then used for up to 6 months duration. The solution has no preservatives, so sterility cannot be guaranteed past the 8-hour time frame.<sup>7</sup>

Currently, Angell Animal Medical Center is participating in an international, multi-institutional, randomized, double-blind clinical study evaluating the use of TPA in 40 cats with ATE. The primary investigator of the Bilateral Lysis of Aortic Saddle Thrombus with Early TPA (BLASTT) study is Dr. Julien Guillaumin from The Ohio State University College of Veterinary Medicine. There are specific exclusion criteria, but the main inclusion criteria is that two to three limbs need to be affected and the event must have occurred within the past 6 hours, so that treatment can be initiated within 6 hours of the event. Then, either TPA or placebo (0.9% NaCl) is administered at 1 mg/kg with a maximum dose of 6 mg. Ten percent of the dose is given as a small bolus over a minute, followed by a 60-minute CRI. A limb scoring scheme is then used every 12 hours to determine any notable changes to the pulses and motor function in the limbs. The study is still being conducted, but the interim analysis of the 33 cats and the accompanying initial data are indicating that the earlier the intervention, the better.

The use of TPA remains controversial and should be decided on an individual case basis after discussion with the owner of the potential benefits and possible adverse side effects in his/her cat. For those clients wanting to be more aggressive in treatment, TPA may be the drug to help cats. It is clear that more studies need to be conducted to elucidate the ideal timing and dose.

### FIGURES 1 & 2

▼ An ATE cat that was medically managed and the end-stage damage of ischemic necrosis. Photos courtesy of Dr. Therese O'Toole.



## EMERGENCY

CONTINUED FROM PAGE 6

## FIGURES 3 &amp; 4



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## Novel Oral Anticoagulants (NOACs) in Veterinary Medicine

Rebecca Quinn, DVM, DACVIM

angell.org/cardiology | cardiology@angell.org | 617 541-5038

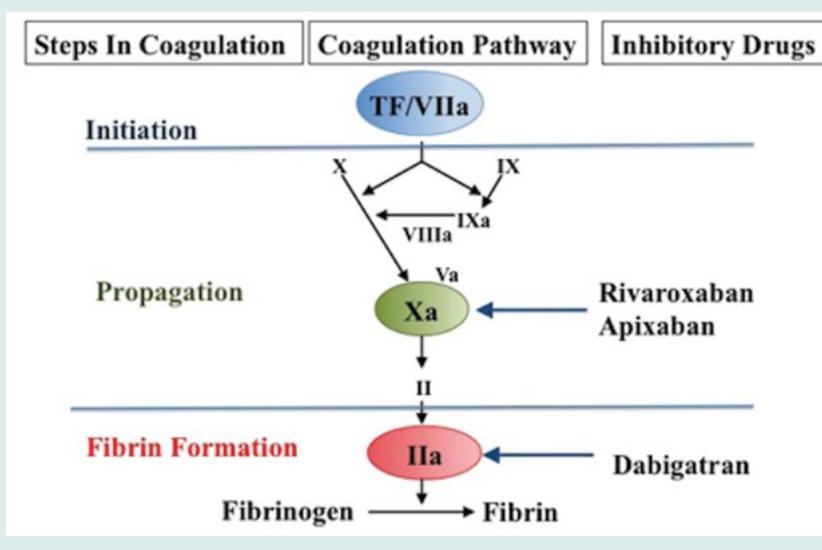
**T**hrombotic disease has been a well-recognized complication of human health for centuries. Millennia, in fact, if one considers that Hippocrates was the first to identify a stroke in a human patient in the year 400 BC.<sup>1</sup> Other than ischemic strokes, human thrombotic disease includes such conditions as myocardial infarction, deep vein thrombosis, and Budd-Chiari syndrome. It is therefore interesting to take note of the timeline of anticoagulant therapies, which didn't blossom until the use of heparin in the 1930s.<sup>2</sup> Over nearly 80 years, the medical community moved on to discover oral vitamin K antagonists (1940s), low-molecular-weight heparin (1980s), and finally the first novel oral anticoagulant (NOAC) in 2008. Reviewing this timeline, it is easy to understand why warfarin became almost a daily household name: For over 60 years, warfarin was the only oral anticoagulant available to not only human but veterinary patients.

In veterinary medicine, thrombosis is most often secondary to cardiac disease in cats (acquired cardiomyopathies), and associated with non-cardiac illness in dogs (most commonly immune-mediated hemolytic anemia, sepsis, neoplasia, Cushing's disease, and glomerular disease).<sup>3</sup> Historically, the veterinary community relied on warfarin for at-home treatment of many thromboembolic diseases, and encountered challenges in terms of administration, monitoring, safety, and side effects.<sup>4,5</sup> While other oral therapies have become available, mainly in the form of antiplatelet medications such as aspirin and clopidogrel (Plavix), another true oral anticoagulant has not been available until the introduction of the NOACs.

The NOACs, which are also referred to as "non-vitamin K oral anticoagulants," inhibit the traditionally named extrinsic coagulation pathway.<sup>6</sup> Specifically, they work by either inhibiting factor Xa (rivaroxaban and apixaban) or by directly inhibiting thrombin formation (dabigatran) (See Figure 1<sup>6,7</sup>). By interfering with the coagulation pathway at these levels, NOACs prevent the formation of fibrin and cross-linked fibrin clots.<sup>6</sup> Ultimately, they help prevent thrombus formation with fewer side effects

FIGURE 1.

This figure was borrowed from the journal *Arteriosclerosis, Thrombosis, and Vascular Biology*<sup>6</sup>, and demonstrates the site of action of the NOACs rivaroxaban, apixaban, and dabigatran.



compared to warfarin.<sup>8</sup> As a result, over the last 10 years, NOACs have largely replaced warfarin in human medicine. In observing our human counterparts and their overwhelmingly positive experiences with NOACs, we in the veterinary community are now exploring clinical opportunities in our patients.

Rivaroxaban (Xarelto) is the most well-studied NOAC in veterinary medicine. It inhibits factor Xa, which therefore prevents the conversion of prothrombin to thrombin.<sup>6</sup> Rivaroxaban has been established to effectively suppress thrombin formation in canine and feline patients, and has been demonstrated as a safe therapy (canine median dose 0.89 mg/kg by mouth once daily, feline dose range 1.25–5.0 mg per cat by mouth once to twice daily).<sup>9,10,11</sup> In a more clinical setting, rivaroxaban has been used to treat dogs with pulmonary embolism (PTE), arterial thromboembolism, and jugular vein embolism.<sup>12</sup>

In this particular case study, rivaroxaban was given at a dose range of 0.6–1.0 mg/kg by mouth once a day, with no obvious side effects and apparent resolution of the thrombi.<sup>12</sup>

A feline study is currently underway investigating the effectiveness of rivaroxaban as compared to clopidogrel in cats with cardiogenic thromboembolism. This study, called SUPER-CAT (Study of the Utility of Rivaroxaban or Clopidogrel for prevention and recurrent Arterial Thromboembolism in cats), is ongoing and organized by the University of Georgia with support from the Morris Animal Foundation. Anecdotal, rivaroxaban at doses of 2.5–5.0 mg per cat by mouth once to twice daily has been used effectively and safely in cats with or at risk of cardiogenic thromboembolism. Interestingly, clinicians have found that rivaroxaban can be combined with aspirin and/or clopidogrel therapy in feline patients who are at very high risk for

thromboembolic events. This offers potential multimodal anticoagulation, and the risk of bleeding appears to be low. Monitoring rivaroxaban in the laboratory setting is a challenge in veterinary medicine, as the species-specific anti-factor Xa assay available to human patients is not available to cats or dogs.<sup>13</sup> Prothrombin time can be measured to monitor for adverse effects, but this test is often only pursued if the veterinarian is concerned about excessive anticoagulation, or if the recommended doses are exceeded. It is important to understand that the reversal agents available in human medicine have not been studied in veterinary patients, and are likely to be cost prohibitive; should a patient treated with rivaroxaban develop significant bleeding, this may be very difficult to control.<sup>14</sup>

Like rivaroxaban, apixaban (Eliquis) is a factor Xa inhibitor. The differences between the two drugs can be found when reviewing the human-based pharmacokinetic properties. Of note, apixaban has lower oral bioavailability and less renal excretion compared to rivaroxaban.<sup>15</sup> Very little is known about apixaban use in dogs; there have been several pharmacokinetic studies in cats, indicating that apixaban at a dose of 0.625 mg per cat by mouth twice a day or 0.2 mg/kg by mouth once a day effectively inhibits factor Xa.<sup>16,17</sup> Preliminary data suggest that cats may develop nausea or vomiting with apixaban therapy, and patients should be monitored for gastrointestinal side effects.<sup>16</sup> To date, there are no published or ongoing clinical studies assessing apixaban use in dogs or cats.

Dabigatran varies from the other members of the NOAC family in that it directly inhibits thrombin, which therefore prevents the conversion of fibrinogen to fibrin.<sup>6</sup> It has the lowest oral bioavailability, is predominantly renally excreted, and must be metabolized from its prodrug to active form.<sup>15</sup> While studies indicate that dabigatran is an effective anticoagulant in dogs, there is no clinical data supporting its use in either dogs or cats in the hospital setting.<sup>18</sup>

Novel oral anticoagulant therapy use in veterinary medicine is on the rise, and will likely continue to trend in that direction. At the present time, rivaroxaban use is most supported, but therapy should be approached thoughtfully. As we continue to learn, one cannot help but hope that we will discover more effective means of treating our canine and feline patients with devastating thromboembolic disease.

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# Discospondylitis

Michele James, DVM, DACVIM

angell.org/neurology | neurology@angell.org | 617 541-5140

## OVERVIEW

Discospondylitis is an inflammatory disease of the spine characterized by infection of the intervertebral disc, adjacent vertebral end plates, and vertebral bodies. The infection typically develops secondary to hematogenous spread of bacteria from a distant site, such as the urinary tract, prostate, mouth, or skin. In rare cases, the infection can originate from a migrating foreign body (e.g., a grass lawn), penetrating trauma, surgery, or epidural injection. The most commonly reported bacterial etiologic agents are *Staphylococcus spp* (*S. aureus* and *S. intermedius*); however, *Streptococcus spp*, *Brucella canis*, and *Escherichia coli* have been routinely isolated. *Salmonella*, *Enterobacter*, *Nocardia*, and variable fungal organisms have also been reported. Dogs are more commonly affected, though discospondylitis has been described in cats, horses, ruminants, and pigs. In a large study by Burkett et al. of more than 500 dogs, examining the signalment and clinical features of discospondylitis, male dogs, older dogs, and purebred dogs were more likely to be affected with the disease than their counterparts. Great Danes, German Shepherds, and Labrador Retrievers appear to be overrepresented in the literature.

## PRESENTATION

Patients with discospondylitis can have variable clinical signs, though spinal hyperesthesia is the most commonly reported physical exam finding. Some patients may also present with non-specific systemic signs, including lethargy, fever, and decreased appetite. Owners may report waxing and waning clinical signs that appear to worsen with physical activity. Depending on the location, duration, and severity of the lesion, some patients may present with minimal to no appreciable pain, whereas others may have more severe neurologic dysfunction ranging from ataxia to plegia. Neurologic deficits, such as paresis/plegia, are rarely observed in the absence of more serious complications such as vertebral luxations, pathologic fractures, and/or spinal empyema.

## DIAGNOSIS

Discospondylitis is clinically diagnosed by radiography, though radiographic changes may lag several days to weeks after onset of infection.

Radiographic findings include osteolysis of vertebral end plates, sclerosis of adjacent bone, and narrowing of the intervertebral disc space (Figure 1). Advanced imaging modalities, namely CT and MRI, are able to identify discospondylitis sites earlier in the disease process prior to radiographic diagnosis. The lumbosacral disc space is reported to be one of the most commonly affected sites, though the thoracolumbar and cervical spine are also frequently affected.

Positive microbial culture results not only help to support a diagnosis of discospondylitis, but also help to guide treatment. Urinalysis and urine cultures should be submitted in all patients suspected of having discospondylitis. Blood cultures may also be submitted; however, in the author's experience these have been of low yield, particularly if the patient is not febrile at the time of diagnosis. Fine needle aspirate of an affected

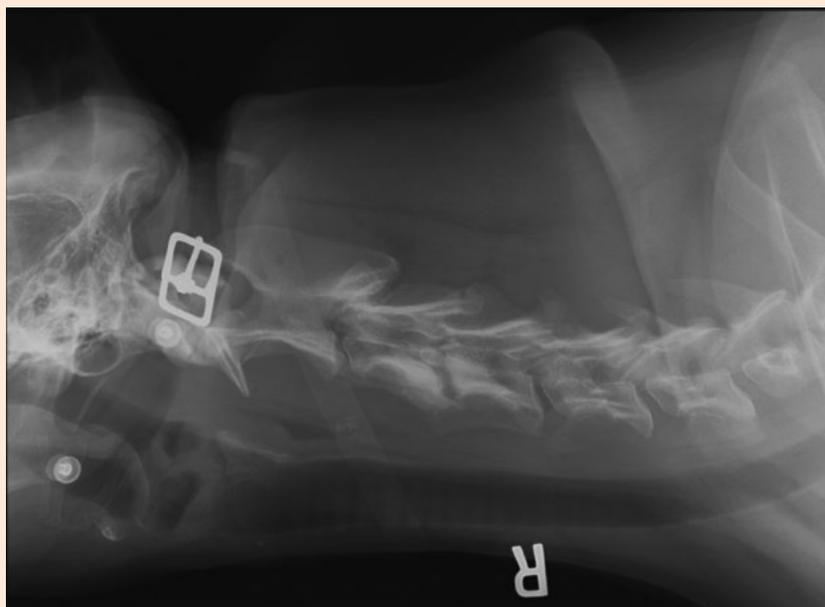
disc space may be considered in hopes of determining the microbial profile of an infection, but diagnostic yield is reported to vary greatly, and in the author's experience has also been low yield. Though *Brucella* accounts for less than 10 percent of discospondylitis cases in dogs, due to the risk of zoonotic potential of brucellosis, *Brucella* titers should be performed in all canine patients suspected of discospondylitis. *Brucella* is more prevalent in male intact dogs in the southeastern United States.

## TREATMENT

Medical management with targeted antibiotic therapy, ideally based on isolation of an organism, is the mainstay of treatment for discospondylitis. Rarely, in cases with severe neurologic deficits, surgical decompression and/or stabilization may also be warranted. Since isolation of an organism may not occur in 50-60 percent of

FIGURE 1

↳ Lateral cervical radiograph of a 10-month-old German Shepherd dog with discospondylitis at C3-4. There is osteolysis of the vertebral end plates and moderate sclerosis of the adjacent bone.



cases, empirical treatment with antibiotics known to have good bone penetration and known to be effective against *Staphylococcus* and *Streptococcus* is acceptable and recommended. This includes potentiated cephalosporins (e.g., Cephalexin 30 mg/kg PO Q8hr) or potentiated penicillins (e.g., Clavamox/Augmentin 15 mg/kg PO Q12hr). Analgesics such as Gabapentin, Tramadol, and/or NSAIDs may also be prescribed early in treatment to help address hyperesthesia. Patients should also be exercise-restricted for 4 to 6 weeks following diagnosis.

Patients should experience a resolution of clinical signs within 3-5 days of starting antibiotic therapy. If this does not occur, then additional diagnostic workup (e.g., fine needle aspirate of the disc space, surgical biopsy, and/or fungal testing) may be indicated to determine if a change in antimicrobial therapy is needed in the event of a resistant bacterial infection or underlying fungal infection.

Patients should be treated for ideally 2-4 weeks following resolution of both clinical signs and radiographic resolution of the infection, as premature discontinuation of antimicrobial therapy results in a relapse of clinical signs. For many animals, radiographic resolution may take several months or longer to occur; therefore most patients will need to be on antibiotic therapy for 6 months to a year or longer. The author recommends recheck radiographs 2 months following initiation of treatment and then every 3-4 months until evidence of bony lysis has disappeared and/or vertebral fusion has occurred on two consecutive recheck radiographs. Healed discospondylitis on radiographs is characterized by ankylosis and the replacement of lytic bone with osseous proliferation (Figure 2).

Fungal infections should be considered in patients not responding to empirical antibiotic therapy. German Shepherds are predisposed to *Aspergillus* infection. In cases of fungal discospondylitis, early treatment with an antifungal microbial is recommended (e.g., itraconazole 5 mg/kg PO Q24hr).

Brucellosis is an incurable disease; however, some infected dogs may respond favorably to combination antimicrobial therapy (e.g., Enrofloxacin 10-20 mg/kg PO Q24hr and Doxycycline 5 mg/kg PO Q12hr). It is recommended that infected patients be castrated or spayed, and owners and other people in contact with the patient should take appropriate measures to prevent exposure (e.g., appropriate hand washing, etc.). Brucellosis is a reportable disease in many states, including Massachusetts.

FIGURE 2

↘ Lateral cervical radiograph of the same German Shepherd dog pictured in Figure 1 following 6 months of antibiotic treatment for previously diagnosed discospondylitis. There is ankylosis of the C3 and C4 vertebral bodies and reduction in the degree of osteosclerosis of the C3-4 vertebral end plates.



### PROGNOSIS

For a large majority of cases, the prognosis for bacterial discospondylitis is very good, particularly if treated early and prior to severe neurologic deficits. However, owners should be counseled on the need for long-term antibiotic therapy and repeat radiographs. Although brucellosis is incurable, patients may be able to be managed long term with lifelong antibiotic treatment. Unfortunately, fungal discospondylitis has a guarded to poor prognosis due to the risk of disseminated disease.

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## TECH TIP

# Management and Complications of the Newly Diagnosed Diabetic Dog

■ Caitlan Koontz, DVM

[angell.org/emergency](http://angell.org/emergency) | [emergency@angell.org](mailto:emergency@angell.org) | 617 522-7282

**D**iabetes mellitus is a commonly diagnosed endocrine disorder in dogs. In humans, diabetes is classified most simply as type I or type II, based on the pathophysiology of the disease and requirement for exogenous insulin. Diabetes in our canine patients most closely resembles type I diabetes. However, it is more appropriately classified as “insulin-dependent diabetes mellitus” (IDDM), as nearly all diabetic dogs require daily insulin therapy.<sup>1</sup> In the insulin-dependent diabetic dog, there is a relative or absolute deficiency of insulin. The etiologies vary but generally include a combination of genetic predisposition, immune-mediated destruction of beta islet cells, pancreatitis, and concurrent diseases and environmental factors leading to beta cell destruction and/or insulin resistance.<sup>1</sup> A few breeds at higher risk for developing diabetes include the Schnauzer, Toy and Miniature Poodle, Terrier breeds and mixes, Beagle, and Dachshund.<sup>3</sup>

The most common clinical signs of diabetes in the dog include polyuria (PU), polydipsia (PD), polyphagia, and weight loss.<sup>1</sup> Insulin deficiency and resistance lead to hyperglycemia due to decreased tissue utilization of glucose. Tissues are essentially “starved” of glucose, thus stimulating hepatic gluconeogenesis and glycogenolysis, worsening the hyperglycemia. Glucose from circulation spills over into the urine as the threshold for reabsorption of glucose by the renal tubules is exceeded. Glucose in the urine acts as an osmotic diuretic, leading to polyuria and subsequent polydipsia. In the untreated or poorly regulated diabetic dog, where these common clinical signs are mild or go unnoticed by the owner, diabetic ketoacidosis (DKA) may ultimately develop (DKA will be discussed further in the following paragraphs). Many canine patients often present to their general practitioner or an emergency service suffering from DKA. Common clinical signs include lethargy or weakness, hyporexia or anorexia, vomiting, labored breathing, and collapse.<sup>2</sup>

Diabetes in the dog is diagnosed in a variety of situations. Hyperglycemia may be discovered incidentally via routine blood



work, or a patient may be presented to a veterinarian for the common clinical signs, particularly PU/PD. In these patients, the diagnosis is ultimately made based on the presence of persistent fasting hyperglycemia and glycosuria.<sup>1</sup> In patients who are suffering from diabetic ketosis or ketoacidosis, the diagnosis is usually rapidly made based on the presence of marked hyperglycemia, ketonuria, and metabolic acidosis.<sup>2</sup> When hyperglycemia is mild, or to clarify discrepancies on serial blood glucose (BG) measurements and differentiate stress from true hyperglycemia, serum fructosamine may be measured.<sup>1</sup> Various other ancillary tests exist, including measurement of serum insulin concentrations and anti-beta cell autoantibodies.<sup>5</sup> Physical exam abnormalities are often absent in the otherwise healthy, non-ketotic patient; however, muscle wasting, thin body condition, cranial organomegaly, poor hair coat, and mild dehydration may be present.<sup>1</sup> In the sick, undiagnosed diabetic patient who may be suffering from diabetic ketoacidosis, additional exam findings often include more severe dehydration and evidence of hypovolemia, such as tachycardia and poor pulse quality, dull mentation and generalized weakness, sweet-smelling breath that resembles acetone, nausea, abdominal pain, and slow deep breathing (Kussmaul respiration).<sup>2</sup>

Treatment of the newly diagnosed diabetic dog depends on whether the patient is ketotic or not. For the purposes of this review, we will focus on beginning insulin therapy in the non-ketotic patient. For the dog suffering from diabetic ketosis or ketoacidosis, hospitalization at a 24-hour facility is recommended. These patients require short-acting regular insulin in the initial treatment period to eliminate ketones and correct what is usually a marked hyperglycemia. IV fluids and supplemental electrolytes are generally required to correct dehydration and acid/base and electrolyte derangements. Supportive care for other clinical signs or concurrent diseases is also often required.

In the non-ketotic patient, intermediate-acting insulin can be started, assuming that the patient is eating well and any concurrent diseases have been identified and managed. There are several types of intermediate-acting insulins. The two most commonly used in dogs are porcine lente insulin (Vetsulin) and recombinant human NPH insulin, with porcine insulin having a slightly longer duration of action.<sup>3</sup> Porcine insulin has the same amino acid sequence as canine insulin, and thus could be considered the more appropriate choice, as it theoretically would be less likely to stimulate insulin auto-antibodies.<sup>4</sup> However, recombinant human

sourced insulin is very similar, and a study showed that only 5 percent of dogs treated with it developed antibodies.<sup>1</sup> Determining which insulin to use ultimately depends on availability, personal preference, and patient response (i.e., control of hyperglycemia and associated clinical signs at home).

The recommended starting dosage for intermediate-acting insulin is 0.25U/kg twice a day. However, higher doses are often eventually required, especially in patients with concurrent illnesses or environmental factors causing insulin resistance.<sup>1</sup> Insulin therapy should be initiated in hospital and blood sugars monitored using a typical BG curve over 12-24 hours. The goal at this time is not to obtain perfect glycemic control, and the client should be informed that this ultimately can take a month or longer. During the initial treatment period and first BG curve, the objective is to identify the nadir (lowest BG reading) and at what time during the day it occurs. If the nadir is less than 80 mg/dL, the insulin dose should be reduced.<sup>1,3</sup> If the nadir is greater than 150 mg/dL, the dose should be increased.<sup>1,3</sup> A safe unit of increase is approximately 1U/injection; however, the size of the dog should be considered and the exact U/kg dose calculated.<sup>6</sup> If the dose exceeds 1U/kg without adequate glycemic control, it is likely that there are other factors—such as concurrent illness or improper insulin handling—at play, and these causes should be investigated.<sup>3</sup> The nadir should ideally fall between 100 and 125 mg/dL.<sup>3</sup>

The duration of effect of the insulin should also be evaluated on the BG curve. If the duration is less than 10 hours, a longer-acting insulin should be used (e.g., if NPH is being used, the insulin could be switched to Lente) and vice versa if the duration of action is longer than 14 hours.<sup>1,3</sup> The long-term, ideal BG concentrations in a well-controlled diabetic dog should fall between 100 and 250 mg/dL.<sup>1,3</sup> After an initial dose is determined

and the patient is discharged, a recheck BG curve should be scheduled for the following week, and either weekly or biweekly thereafter until adequate glycemic control is achieved and the owner sees resolution of clinical signs at home. Gradual increases in insulin doses and close monitoring are important to avoid complications, including hypoglycemia and Somogyi response.

Dietary and lifestyle adjustments are also important when managing the newly diagnosed diabetic dog and should be maintained for the long term. Typical diets recommended for the diabetic dog are those high in fiber and low in fat. The main mechanisms by which these diets help improve glycemic control include the fiber creating a viscous gel within the intestine, thus inhibiting absorption of glucose, delaying gastric emptying, and delaying absorption of nutrients.<sup>1</sup> The higher the percentage of crude fiber, the lower the calorie content. Therefore, these diets should be used with caution in thin or underweight diabetic dogs. However, they are important and effective in overweight or obese patients. Regular exercise should also be implemented, as it helps promote weight loss and thus reduce the insulin resistance brought on by obesity, and help mobilize insulin from the site of injection.

Despite seemingly straightforward therapeutic recommendations, diabetes can be a very difficult disease to treat and control in dogs (and cats). As mentioned briefly above, several complications can occur. Hypoglycemia can be seen with large increases in insulin or overlap of insulin action in dogs being treated twice daily, after excessive exercise, or when there is significant hyporexia/anorexia, vomiting, or diarrhea. Insulin overdosage can lead to hypoglycemia and subsequently the Somogyi response, in which the body secretes insulin counter-regulatory hormones such as glucagon, cortisol, and epinephrine in response to hypoglycemia.<sup>1</sup> This leads to a profound *hyperglycemia*. Twelve-hour BG

curves are important if the Somogyi response is suspected, since increasing the insulin dose based on a single BG measurement where significant or worsening hyperglycemia is found can ultimately be deleterious. Correcting the Somogyi response requires decreasing the insulin dose.

Diabetic ketoacidosis is a condition more commonly encountered in the undiagnosed or untreated diabetic, but can occur in underdosed and poorly regulated diabetic dogs, especially when there is concurrent illness causing insulin resistance. With DKA, the body mobilizes stored fat to be used as an energy source. The metabolism of stored fatty acids results in the production of ketone bodies, which are acidic, and accumulation can lead to a metabolic acidosis and profound illness. As discussed above, dogs suffering from DKA require hospitalization and 24-hour care to correct the acidosis and eliminate the ketones. Other complications commonly encountered when treating the diabetic dog include concurrent illnesses or conditions that induce insulin resistance. Some examples include infection, such as urinary tract infections, pancreatitis, hyperadrenocorticism, hypothyroidism, diestrus in intact female dogs, and renal and liver insufficiency.<sup>3</sup> Use of diabetogenic drugs (most commonly glucocorticoids) will also make glycemic control difficult. Thus, when diagnosing diabetes and initiating insulin therapy, a full systemic workup and control or correction of these diseases and conditions must be made a priority.

The prognosis of dogs with diabetes depends on a variety of factors, including age, ease of glycemic control, owner compliance and willingness to treat, and control or reversal of concurrent illnesses. Long-term complications and quality of life are also important factors. However, with good glycemic control, proper care by the owners at home, and regular veterinary rechecks, the diabetic dog can lead a generally happy and healthy life.

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## STAFF DOCTORS AND RESIDENTS

■ 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

## CHIEF OF STAFF

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

## 24-HOUR EMERGENCY &amp; CRITICAL CARE MEDICINE, BOSTON

**Alison Allukian, DVM**  
aallukian@angell.org

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

**Maria Brandifino, DVM**  
mbrandifino@angell.org

**Kate Dorsey, DVM**  
kdorsey@angell.org

**Morgan Kelley, DVM**  
mkelley@angell.org

**Audrey Koid, DVM**  
akoid@angell.org

**Caitlin Koontz, DVM**  
ckoontz@angell.org

**William (Glenn) Lane, DVM**  
wlane@angell.org

**Susan Magestro, DVM**  
smagestro@angell.org

**Hannah Marshall, DVM**  
hmarshall@angell.org

**Emiliana Meroni, DVM**  
emeroni@angell.org

**Virginia Sinnott-Stutzman, DVM, DACVECC**  
vsinnottstutzman@angell.org

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

## 24-HOUR EMERGENCY &amp; CRITICAL CARE MEDICINE, WALTHAM

**Lauren Baker, DVM**  
lbaker@angell.org

**Alyssa Blaustien, VMD**  
ablaustein@angell.org

**Jordana Fetto, DVM**  
jfetto@angell.org

**Emily Finn, DVM**  
efinn@angell.org

**Amanda Lohin, DVM**  
alohin@angell.org

**Kate Mueller, DVM**  
kmueller@angell.org

**Courtney Peck, DVM, DACVECC**  
cpeck@angell.org

**Jessica Seid, DVM**  
jseid@angell.org

**Susan Smith, DVM**  
ssmith@angell.org

**Catherine Sumner, DVM, DACVECC**  
*Chief Medical Officer, Waltham*  
csumner@angell.org

## ANESTHESIOLOGY

**Stephanie Krein, DVM, DACVAA**  
skrein@angell.org

## AVIAN &amp; EXOTIC MEDICINE (W/B)

**Brendan Noonan, DVM, DABVP (Avian Practice)**  
(Boston & Waltham)  
bnoonan@angell.org

**Elisabeth Simone-Freilicher, DVM, DABVP (Avian Practice)**  
esimonefreilicher@angell.org

**Anne Staudenmaier, VMD**  
astaudenmaier@angell.org

**Patrick Sullivan, DVM**  
psullivan@angell.org

## BEHAVIOR (W/B)

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

## CARDIOLOGY (W/B)

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

**Julia Lindholm, DVM**  
jlindholm@angell.org

**Rebecca Malakoff, DVM, DACVIM (Cardiology)**  
(Waltham)  
rmalakoff@angell.org

**Natalie Morgan, DVM**  
nmorgan@angell.org

**Rebecca Quinn, DVM, DACVIM (Cardiology and Internal Medicine)**  
rquinn@angell.org

**Joseph Zarin, DVM**  
jzarin@angell.org

## DENTISTRY

**Erin Abrahams, DVM**  
eabrahams@angell.org

**Colleen McCarthy, DVM**  
cmccarthy@angell.org

**Jessica Riehl, DVM, DAVDC**  
jriehl@angell.org

## DERMATOLOGY

**Klaus Loft, DVM**  
kloft@angell.org

## DIAGNOSTIC IMAGING (W/B)

**Naomi Ford, DVM, DACVR**  
nford@angell.org

**Heather Spain, DVM**  
hspain@angell.org

**Steven Tsai, DVM, DACVR**  
stsai@angell.org

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

## INTERNAL MEDICINE (W/B)

**Douglas Brum, DVM**  
dbrum@angell.org

**Alyssa Bryce, DVM**  
abryce@angell.org

**Maureen Carroll, DVM, DACVIM**  
mccarroll@angell.org

**Zach Crouse, DVM, DACVIM**  
zcrouse@angell.org

**Erika de Papp, DVM, DACVIM**  
edepapp@angell.org

**Jean Duddy, DVM**  
jduddy@angell.org

**Lisa Gorman, DVM, DACVIM**  
lgorman@angell.org

**Kirstin Johnson, DVM, DACVIM**  
kcjohnson@angell.org

**Shawn Kearns, DVM, DACVIM**  
skearns@angell.org

**Evan Mariotti, DVM**  
emariotti@angell.org

**Susan O'Bell, DVM, DACVIM**  
*Service Director*  
sobell@angell.org

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

## STAFF DOCTORS AND RESIDENTS

## NEUROLOGY (W/B)

**Rob Daniel, DVM, DACVIM (Neurology)**  
(Boston & Waltham)  
rdaniel@angell.org

**Michele James, DVM, DACVIM (Neurology)**  
(Boston & Waltham)  
mjames@angell.org

**Jennifer Michaels, DVM, DACVIM (Neurology)**  
(Boston & Waltham)  
jmichaels@angell.org

## NUTRITION

**Dana Hutchinson, DVM, DACVN**  
dhutchinson@angell.org

## ONCOLOGY

**Lyndsay Kubicek, DVM, DACVR (Radiation Oncology)**  
lkubicek@angell.org

**J. Lee Talbott, DVM, DACVIM (Medical Oncology)**  
jtalbott@angell.org

## OPHTHALMOLOGY

**Ken Abrams, DVM, DACVO**  
kabrams@angell.org

**Daniel Biros, DVM, DACVO**  
dbiros@angell.org

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

## PAIN MEDICINE

**Lisa Moses, VMD, DACVIM, CVMA**  
lmoses@angell.org

## PATHOLOGY

## (CLINICAL &amp; ANATOMIC)\*

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

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

## PHYSICAL REHABILITATION

**Jennifer Palmer, DVM, CCRT**  
jpalmer@angell.org

**Amy Straut, DVM, CCRT**  
astraut@angell.org

## SURGERY (W/B)

**Sue Casale, DVM, DACVS**  
scasale@angell.org

**Megan Cray, VMD**  
mcray@angell.org

**Andrew Goodman, DVM, DACVS**  
agoodman@angell.org

**Michael Pavletic, DVM, DACVS**  
mpavletic@angell.org

**Nicholas Trout MA, VET MB, MRCVS, DACVS, DECVS**  
ntrout@angell.org

**Emily Ulfelder, BVetMed**  
eulfelder@angell.org

**Spencer Yeh, DVM**  
syeh@angell.org

## ANGELL AT NASHOBA

**Laurence Sawyer, DVM**  
lsawyer@angell.org

(W/B) Services also available at our Waltham location

## Our Service Locations

## BOSTON &amp; WALTHAM

**Avian & Exotic Medicine**  
617 989-1561

**Behavior**  
617 989-1520

**Cardiology**  
617 541-5038

**Diagnostic Imaging**  
617 541-5139

**Internal Medicine**  
617 541-5186

**Neurology**  
617 541-5140

**Physical Rehabilitation\*\***  
781 902-8400

**Surgery**  
617 541-5048

## BOSTON ONLY

**Anesthesiology**  
617 541-5048

**Dentistry**  
617 522-7282

**Dermatology**  
617 524-5733

**Nutrition**  
617 522-5011

**Oncology**  
617 541-5136

**Ophthalmology**  
617 541-5095

**Pain & Palliative Care**  
617 541-5140

**Pathology\***  
614 541-5014



### 24/7 Emergency & Critical Care

■ Boston: 617 522-5011 ■ Waltham: 781 902-8400

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



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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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MSPCA-ANGELL

350 South Huntington Avenue  
Boston, MA 02130  
617 522-5011  
[angell.org](http://angell.org)

MSPCA-ANGELL  
WEST

293 Second Avenue  
Waltham, MA 02451  
781 902-8400  
[angell.org/waltham](http://angell.org/waltham)

ANGELL-AT  
NASHOBA

100 Littleton Road  
Westford, MA 01886  
978 577-5992  
[angell.org/nashoba](http://angell.org/nashoba)

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

■ [angell.org/directions](http://angell.org/directions) (free parking) ■ [angell.org/hours](http://angell.org/hours) ■ [angell.org/ce](http://angell.org/ce)



## ➤ Have you referred to Angell's Pain and Palliative Care service?

Angell's Pain and Palliative Care service is for companion animals with discomfort and disability that negatively affects their quality of life, regardless of their age or the cause of their distress. Many of our patients have complex medical issues that complicate treatment for pain.

Lisa Moses, VMD, DACVIM, CVA, uses her advanced training in pain medicine and palliative care to provide treatment options including (but not limited to) novel drug therapy and non-drug modalities of treatment, including medical acupuncture and mobility exercises.