



NEUROLOGY
PAGE 1 Masticatory Muscle Myositis
INTERNAL MEDICINE
PAGE 1 An Update on Extracorporeal Therapies for Exogenous and Endogenous Toxins
PATHOLOGY
PAGE 5 Color Atlas: Feline Gastrointestinal Tumors
EMERGENCY & CRITICAL CARE
PAGE 8 Marijuana Toxicosis
DERMATOLOGY
PAGE 10 Non-Neoplastic Anal Sac Disease: How to Help Patients Who Scoot, Lick, Leak, or Smell
AVIAN & EXOTIC MEDICINE
PAGE 14 Introduction to Dermatology in the Exotic Animal Patient
EMERGENCY & CRITICAL CARE
PAGE 17 Respiratory Emergencies



NEUROLOGY

Masticatory Muscle Myositis

Rob Daniel, DVM, DACVIM
angell.org/neurology | neurology@angell.org | 617-541-5140

Masticatory Muscle Myositis (MMM) is a relatively common inflammatory disorder affecting primarily dogs, rarely cats. This autoimmune disorder targets the muscles of mastication, specifically the temporalis, masseter, and medial and lateral pterygoid muscles. Pets present with common clinical signs at various stages of disease, with the acute phases of disease, in particular, calling for a prompt diagnosis, early intervention, and the best chances for improvement.

Common signalments are younger, larger-breed dogs, including the German Shepherd, Labrador Retriever, Golden Retriever, and Doberman Pinscher, although MMM can affect any dog at any age. A possible MMM variant has been reported to affect young (~12 weeks old) Cavalier King Charles Spaniels; MMM is rarely reported in cats. Common historical complaints and clinical signs in the early phases of the disease include lethargy, hyporexia/anorexia, exophthalmos, third-eyelid protrusion, trismus, regional lymphadenomegaly, and dysphagia. Fever is possible. Swelling of the pterygoid muscles

(CONTINUED ON PAGE 2)



INTERNAL MEDICINE

An Update on Extracorporeal Therapies for Exogenous and Endogenous Toxins

Shawn Kearns, DVM, DACVIM
angell.org/internalmedicine | internalmedicine@angell.org | 617-541-5186

Extracorporeal therapies (ECT) in veterinary medicine are an advanced modality for the treatment of acute kidney injury (hemodialysis; HD), autoimmune diseases (therapeutic plasma exchange; TPE), and toxicities. Since 2015, the number of toxicity cases treated with advanced modalities has significantly increased, likely due to increased access to equipment, increased training, and drawing

on experiences from human medicine. In human medicine, the use of ECTs has also increased over the years from ~0.1% use in all poisoning to ~0.45% use.^{1,2} Many substances have a large safety margin and are safe when exposed in isolation. There may be medical means for treatment, but ECT may be considered when this is not the case.

(CONTINUED ON PAGE 4)



might explain the exophthalmos, whereas muscle spasms might explain the reduction in jaw movement seen in trismus. In chronic stages of disease, masticatory muscle atrophy can be severe, with loss of muscle support for the eye leading to enophthalmos and varying limits to open mouth. Interestingly, there have been anecdotal observations of a relative 'latent' period between the acute and chronic phases, whereby the pet seems partially improved compared to the acute phase.

Differential diagnoses for MMM include inflammatory causes (retrobulbar abscess, generalized polymyositis, extraocular myositis), musculoskeletal (temporomandibular joint disorder), endocrine (hyperadrenocorticism, hypothyroidism), neoplasia (cachexia, trigeminal nerve sheath tumor) and sarcopenia.

The pathogenesis of MMM has been detailed. The muscles of mastication, specifically the temporalis, masseter, and medial and lateral pterygoid muscles, differ in their particular isoforms from their appendicular counterparts. The masticatory muscles originate from the branchial arch mesoderm, whereas those of the appendicular skeletal originate from the paraxial mesoderm. The masticatory muscles have a unique myosin isoform, the Type 2M myofiber, which is subsequently targeted by autoantibodies in MMM, leading to inflammation and phagocytosis, with resultant myonecrosis and end-stage fibrosis. Lymphocytes, particularly T cells, are key players in MMM. The exact etiology of MMM is unknown, although molecular mimicry between Type 2M myosin isoform and an unknown infectious agent has been theorized.

A minimum database should be submitted following a general physical examination that suspects MMM, including a CBC, serum chemistry with CK, urinalysis, and thyroid panel. An inflammatory leukogram may be present, with a chemistry supporting muscle involvement (hyperCKemia, elevated ALT and AST enzyme activity). The serologic test for 2M autoantibodies is the gold standard for MMM and should be submitted early in the diagnostic workup before starting corticosteroid therapy to minimize false negative results. A temporalis muscle biopsy should be considered early in diagnostic workup, considering the longer turnaround time for serum 2M autoantibody testing and the lack of information pertaining to both severity and prognosis on serum testing alone. Advanced imaging, namely MRI or CT, can be beneficial and sensitive in early stages of disease, helping to identify affected regions of a particular muscle and identifying affected muscle that can be targeted for biopsy to minimize false negative results. Electrophysiology, namely electromyogram (EMG), can be performed with predictable abnormalities that might be identified in the affected muscle.

Response to treatment can be good if instituted early in the disease. Autoimmune therapy is the cornerstone of therapy, with autoimmune corticosteroid dosing commonly instituted until jaw mobility and jaw pain normalize. Slow tapering of corticosteroids over extended months is encouraged to minimize relapse. Adjunctive immunomodulatory drugs, such as leflunomide, mycophenolate, and/or ciclosporin, to name a few, can be beneficial, either replacing the need for corticosteroid altogether or to help lower the dosing of corticosteroid quicker than might be realized is using corticosteroid monotherapy.

Follow-up with these patients' families and follow-up physical examinations are important, paying particular attention to drug side effects, drug monitoring, body weight, degree of muscle atrophy, jaw comfort, and mobility.

REFERENCES

- 1 Textbook of Veterinary Internal Medicine Chapters 272, 354
- 2 Practical Guide to Canine and Feline Neurology 3rd Ed Dewey, da Costa
- 3 Atypical and Chronic Masticatory Muscle Myositis in a 5 month old CKCS. Clinical and diagnostic Findings, treatment and successful outcome. *Frontiers in Veterinary Science*. *Front. Vet. Sci.* September 14 2022
- 4 Canine Inflammatory Myopathies: A Clinicopathologic Review of 200 cases. Evans, Levesque, Shelton 2004 *JVIM*; 18 679691.
- 5 Gatineau M, El-Warrak AO, Marretta SM, Kamiya D, Moreau M. Locked jaw syndrome in dogs and cats: 37 cases (1998–2005). *J Vet Dent.* (2008) 25:16–22
- 6 Clinical presentation, MRI, histopathology and outcome in a cat with immune-mediated masticatory myositis. Marco Armellini¹, Lluís Sánchez², Andrea Lorek³, G Diane Shelton⁴ and Luisa De Risio⁵. *JFMS* 2021 Oct 6: 7(2)
- 7 Use of MRI for the Early Diagnosis of Masticatory Muscle Myositis Cauduro, Favole et al *Case Report AAHA* 2013 49:5 Sep/Oct 2013



INTERNAL MEDICINE

An Update on Extracorporeal Therapies for Exogenous and Endogenous Toxins

CONTINUED FROM PAGE 1

> The canine patient (not pictured) received lipids prior to treatment, resulting in the milky fluid on the effluent/collection bag.



Deciding whether to consider hemodialysis, TPE, hemoperfusion (HP), or a combination of therapies for toxicity should be based on several factors. Keeping in mind that the metabolism of a substance may be altered depending on the quantity the patient ingested, the first question to ask is whether using advanced therapies will lead to a higher or faster extraction rate than what the body may metabolize. If allowing the substance to be metabolized endogenously could be lethal or lead to significant or irreversible organ damage, ECT should be a consideration. Second, determine whether the substance could cause a progressive deterioration in clinical status or depression of midbrain function leading to hypoventilation, hypothermia, and hypotension, or if the patient is already demonstrating neurologic decline. If the patient has a pre-existing condition that may impair drug or substance excretion, ECT may be an added tool along with other medical therapies. In veterinary medicine, a challenge in determining the benefit of ECT includes often not knowing the exact timing of exposure and/or the exact dose ingested, and treatment recommendations are often based on the suspected maximal dose ingested or severity of signs (for example, neurological) at presentation.

Not all substances are amenable to clearance by each ECT modality. The choice of clearance therapy mode depends on the molecule's size, the degree of protein binding, and the volume of distribution. Typically, only low molecular weight substances (10,000 daltons) that are less than 80% protein bound with a low volume of distribution (< 2L/kg) can be cleared with diffusion-based HD. Machines capable of convection-based HD can clear slightly larger molecules, but their capacity is still low. Ethylene glycol, ivermectin, metaldehyde, phenobarbital, and baclofen ingestion have been successfully treated with hemodialysis in the veterinary literature.^{3,4,5,6}

Although more studies are needed to determine how large a molecule can be cleared with hemoperfusion (charcoal or the newer carbon-based

therapies), it is thought to be high.¹ Hemoperfusion filters can be used in tandem on hemodialysis platforms or as a stand-alone therapy using a blood pump. In addition, use with other therapies, such as intravenous lipids, may aid in the capture of some molecules. The majority of hemoperfusion reports are associated with non-steroidal anti-inflammatory ingestion (carprofen, ibuprofen, meloxicam).^{7,8,9} After ingestion of a massive dose (550mg/kg) of extended-release 5-hydroxytryptophan (5-HTP), a dog was treated with a combination of hemoperfusion and hemodiafiltration. Treatment was successful, and serial plasma samples indicated that the extraction of 5-HTP was 93.6% to 98.9%.¹⁰ Methotrexate treatment with charcoal HP in combination with HD has also been reported.¹¹ Manual HP may be considered to reduce the blood circuit volume for very small patients or when other specialty equipment is unavailable. This technique was used successfully in a feline patient after an accidental overdose of injectable meloxicam (0.48mg/kg SQ), and the meloxicam blood level was reduced by ~44% with a 60-minute treatment.⁹

Therapeutic plasma exchange is recommended for larger molecules that are highly protein-bound and is the most common therapy reported for ingestion of non-steroidal anti-inflammatory drug (NSAID) overdoses.¹²⁻²⁰ Reduction in plasma levels after one treatment, typically ~1.5 plasma volumes, is reported to be 50% to 85% for NSAIDs. Although TPE +/- additional therapies (intravenous lipids; ILE) may not completely eliminate the risk of acute kidney injury or gastrointestinal disturbances with NSAID overdoses, it may reduce the magnitude in instances where the total dose ingested is extremely high or the half-life is longer, such as with naproxen. Other reports of TPE use include treatment of bifenthrin (pyrethroid insecticide) ingestion²¹ and an inadvertent overdose of intravenous lipids.²² As with hemoperfusion, manual therapeutic plasma exchange has also been reported.^{20, 22}

> The canine patient received TPE for ingestion of an NSAID overdose.



An Update on Extracorporeal Therapies for Exogenous and Endogenous Toxins

CONTINUED FROM PAGE 3

Rarely, endogenous toxins may pose significant enough health risks to warrant removal via extracorporeal therapies. High serum bilirubin levels have been reported in humans and animals to cause neurologic signs. In addition, renal tubular injury may also occur from high bilirubin levels. TPE may be considered in cases with severe hyperbilirubinemia secondary to autoimmune hemolytic anemia leading to kernicterus, which has been reported in dogs.^{23,24} TPE has also been reported for treating one dog with hepatic encephalopathy prior to shunt correction. In this case, ammonia levels decreased by ~50%, and the patient improved clinically.²⁵ An 8-year-old British shorthair that developed neurologic signs and MRI findings consistent with post-attenuation neurologic signs (PANS) was also treated with TPE after failing medical management, including general anesthesia and mechanical ventilation. Seizure activity stopped immediately after the TPE treatment, and the patient showed progressive improvement.²⁶

Although specific guidelines for when to intervene with more advanced therapies should be developed, ECT is a growing field for toxicities. In most cases, the key to successful treatment is an early referral. However, initial decontamination (induction of emesis, activated charcoal), as dictated by a poison control center or patient stability, should be done before referral. Candidate cases are best treated within 24 hours or less of exposure, sooner if possible. While it is hard to conclude the prognosis for individual reports on toxin exposures, the prognosis with the treatment of NSAID overdoses is excellent.^{17, 18} Angell Animal Medical Center offers all three therapy platforms for its veterinary patients.

REFERENCES

- Ghannoum M, Roberts DM, Hoffman RS et al. Seminars in dialysis. Vol 27 (4) 2014 P 362-370
- King JD, Kern MH, Jaar BG. Extracorporeal removal of poisons and toxins. Clin J Am Soc Nephrol. 2019 Aug 22; 14 (9): 1408-1415
- Londono LA, Buckley GJ, Bolfer L et al. Clearance of plasma ivermectin with single pass lipid dialysis in 2 dogs. J Vet Emerg Crit Care 2017 Mar; 27 (2): 232-237.
- Teichmann-Knorm S, Doerfelt S, Doerfelt R. Retrospective evaluation of the use of hemodialysis in dogs with suspected metaldehyde poisoning (2012-2017): 11 cases. J Vet Emerg Crit Care. March 2020; 30 (2): 194-201.
- Basile JK and Vigani A. Treatment of phenobarbital intoxication using hemodialysis in two dogs. J Vet Emerg Crit Care. March 2020; 30 (2): 221-225.
- Hoffman L, Londono LA, Martinz J. Management of severe baclofen toxicosis using hemodialysis in conjunction with mechanical ventilation in a cat with chronic kidney disease. J Feline Med Surg. 2021. Open Rep Jul-Dec;7(2):20551169211033770
- Fick ME, Messenger KM, Vigani A. Efficacy of a single session in-series hemoperfusion and hemodialysis in the management of carprofen overdose in two dogs. J Vet Emerg Crit Care. March 2020; 30 (2): 226-231.
- Tauk BS and Foster JD. Treatment of ibuprofen toxicity with serial charcoal hemoperfusion and hemodialysis in a dog. J Vet Emerg Crit Care. Nov 2016; 26 (6): 787-792.
- Haire LE, Vitalo AD, Goncalves RP et al. Case Report: Manual carbon hemoperfusion for the treatment of meloxicam toxicity in a cat and suspected ibuprofen toxicity in a dog. Frontiers. Sept 2024. DOI 10.3389/fvets.2024.1395967
- Her J, Gordon D, Riggs A. Successful treatment of a severe 5-hydroxytryptophan intoxication using carbon hemoperfusion, hemodiafiltration, and mechanical ventilation in a dog. J Vet Emerg Crit Care. 2024 Mar-Apr; 34 (2): 186-192.
- Pardo M, Lanaux T, Davy R. Use of charcoal hemoperfusion and hemodialysis in the treatment of methotrexate toxicosis in a dog. J Vet Emerg Crit Care. May 2018; 28 (3): 269-273.
- Rosenthal MG, Labato MA. Use of therapeutic plasma exchange to treat nonsteroidal anti-inflammatory drug overdose in dogs. J Vet Intern Med 2019 Mar-Apr; 33 (2): 596-602
- Kicera-Temple K, Londono L, Lanaux TM et al. Treatment of massive naproxen overdose using therapeutic plasma exchange in a dog. Clin Case Rep. August 2019; 7 (8): 1529-1533
- Walton S, Ryan KA, Davis JL et al. Treatment of ibuprofen intoxication in a dog via therapeutic plasma exchange. J Vet Emerg Crit Care. July 2017; 27 (4): 451-457.
- Walton S, Ryan KA, Davis JL et al. Treatment of meloxicam overdose in a dog via therapeutic plasma exchange. J Vet Emerg Crit Care. July 2017; 27 (4): 444-450.
- Kjaergaard AB, Davis JL, Acierno MJ. Treatment of carprofen overdose with therapeutic plasma exchange in a dog. J Vet Emerg Crit Care. July 2018; 28 (4): 356-360.
- Chalifoux NV, Butty EM, Mauro KD et al. Outcomes of 434 dogs with non-steroidal anti-inflammatory drug toxicosis treated with fluid therapy, lipid emulsion, or therapeutic plasma exchange. J Vet Intern Med. 2023; 37: 161-172
- Groover J, Londono LA, Tapia-Ruano K et al. Extracorporeal blood purification in acutely intoxicated veterinary patients: a multicenter retrospective study (2011-2018): 54 cases. J Vet Emerg Crit Care. 2022 January; 32 (1): 34-41.
- Cambournac M, Moumadah Y, Berny P et al. Treatment of flurbiprofen overdose with therapeutic plasma exchange in a dog. Vet Rec Case Rep. 2023 March; 11 (1): e539.
- Buseman M, Blong AE, Walton RA. Successful management of severe carprofen toxicity with manual therapeutic plasma exchange in a dog. J Vet Emerg Crit Care. 2022 September; 32 (5): 675-679.
- Fitzgerald AH, Davies RK, Zhang Y et al. Successful treatment of bifenhrin toxicosis using therapeutic plasma exchange. J Vet Emerg Crit Care. 2024 May-Jun; 34 (3): 291-295.
- Epstein SE, Hopper K, Farrell KS. Manual plasma exchange to treat an accidental overdose of intravenous lipid emulsion in a dog with baclofen toxicosis. JAVMA. Mar 2022; 260 (6): 650-655.
- Heffner GG, Cavanagh A, Nolan B. Successful management of acute bilirubin encephalopathy in a dog with immune mediated hemolytic anemia. J Vet Emerg Crit Care. Sept 2019; 29 (5): 549-557.
- Tovar T, Deitschel S, Guenther C. The use of therapeutic plasma exchange to reduce serum bilirubin in a dog with kernicterus. J Vet Emerg Crit Care. July 2017; 27 (4): 458-464.
- Culler CA, Reinhardt A, Vigani A. Successful management of clinical signs associated with hepatic encephalopathy with manual therapeutic plasma exchange. J Vet Emerg Crit Care 2020 May 30 (3): 312-317
- Niemann L, Beckmann K, Iannucci C et al. Diagnosis of post-attenuation neurologic signs syndrome in a cat with refractory status epilepticus and clinical response to therapeutic plasma exchange. J Feline Med Surg. Open Rep. 2022 Jul-Dec;8(2):20551169221121919.



Color Atlas: Feline Gastrointestinal Tumors

Patty Ewing, DVM, MS, DACVP (Anatomic and Clinical Pathology)
angell.org/pathology | pathology@angell.org | 617-541-5014

Introduction

Cytologic evaluation of gastrointestinal (GI) neoplasia is commonly utilized, given the widespread use of abdominal ultrasound (AUS) in both general and specialty veterinary practices. AUS allows for identifying solitary or multiple masses, which can often be sampled via fine-needle aspiration (FNA) for cytologic evaluation. In a retrospective study that evaluated the diagnostic value of cytologic examination of GI tumors in dogs and cats, there was partial or complete agreement between cytologic and histologic diagnosis for 48 of 67 (71.6%) fine needle aspirates.¹ This degree of agreement warrants using FNA for obtaining a preliminary diagnosis, especially when the owner has financial concerns or an unstable patient condition, which makes surgically obtaining a biopsy sample less desirable. Cytologic evaluation may also be helpful for surgical planning. Although ultrasound-guided FNA is less invasive and does not carry the risk associated with general anesthesia required for endoscopic and surgical biopsy, the procedure is not entirely without risk. Local hemorrhage, risk of tumor cell seeding, and leakage of intestinal content are possible complications associated with needle penetration of the GI tract. FNA should be avoided in patients with $<50,000/\mu\text{l}$ platelet counts or other significant coagulopathy. Intestinal content leakage, a life-threatening complication, and tumor cell seeding can be minimized with careful selection of the needle insertion site, using a 22-gauge or smaller needle, and avoiding multiple needle insertions into the mass.^{2,3} This article will review the cytologic features of the three most common types of feline GI tumors seen at Angell Animal Medical Center.

Lymphoma

Lymphoma is the most common tumor type of the feline GI tract. GI lymphoma can occur as primary GI lymphoma or, less commonly, as a component of multicentric disease. An association with feline leukemia virus (FeLV) in GI lymphoma is typically not observed. Feline GI lymphoma occurs most commonly in the small intestine, followed by the stomach, and infrequently in the large intestine. T-cell lymphoma is more common in the small intestine, and B-cell lymphoma is more common in the stomach and large intestine.⁴ The three

distinct morphologic presentations of feline GI lymphoma include 1) mucosal T-cell lymphoma (enteropathy-associated T-cell lymphoma or EATL type II), which appears well-differentiated, small to intermediate size; 2) large cell lymphoma (gastric B cell lymphoma or transmural T-cell lymphoma also known as EATL type I); large, immature cells with visible nucleoli, and 3) large granular lymphocyte (LGL) lymphoma (aka

granulated lymphoma). Granular lymphoma is likely of cytotoxic T cell or natural killer cell origin. LGL lymphoma is the least common of the three morphologic types. It may exhibit an aggressive clinical course with concurrent involvement of mesenteric lymph nodes, peripheral blood, abdominal effusion, and liver. In one retrospective study, cats with LGL lymphoma who received chemotherapy had a median survival time

FIGURE 1

▶ **Cytologic appearance of large cell gastric lymphoma.** **Left:** Lymphoma cells (black arrows) are intermediate to large, singly occurring round cells with a high nuclear:cytoplasmic ratio (N:C). Nuclei have dispersed chromatin, one or more large, round nucleoli, and a small amount of basophilic cytoplasm. A perinuclear clear zone is evident in most cells. Yellow arrows identify non-neoplastic small lymphocytes for size comparison. (Diff-Quik, 600x magnification.) **Right:** Large lymphoma cells show typical nuclear features of lymphoma, but the addition of small cytoplasmic vacuoles (clear spaces) is found in some cases of feline large-cell lymphoma. (Diff-Quik, 1000x magnification.)

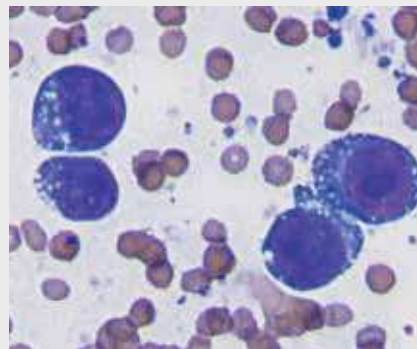
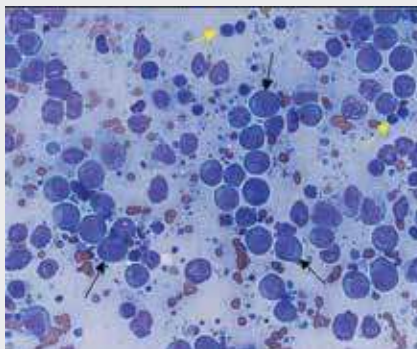
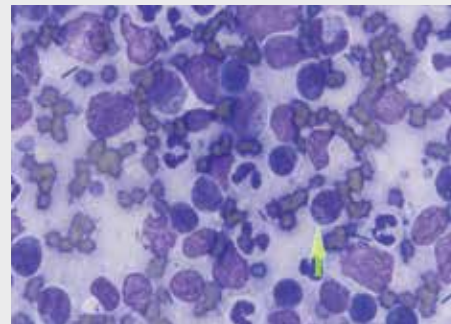
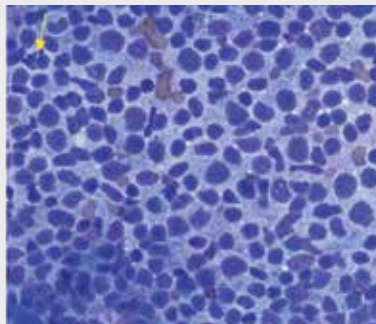


FIGURE 2

▶ **Cytologic appearance of intestinal granular lymphoma.** **Left:** Lymphoma cells are intermediate to large in size and have a high N:C, large round to oval nucleus, and smudged chromatin without distinct nucleoli. The basophilic cytoplasm has several magenta granules. The yellow arrow identifies a bizarre mitotic figure. (Diff-Quik, 600x magnification.) **Right:** Higher magnification shows a cluster of irregularly shaped magenta granules of variable size at one pole of the cytoplasm (yellow-green arrow). These round cells may be mistaken for mast cells if not carefully evaluated. The granular lymphoma cell magenta granules are round to irregular and of variable size compared to the fine metachromatic granules of uniform size evenly distributed throughout the cytoplasm of mast cells (Figure 5). Neutrophils and extracellular bacterial rods suggest ulceration of the overlying mucosa. (Diff-Quik, 750x magnification.)



PATHOLOGY

Color Atlas: Feline Gastrointestinal Tumors

CONTINUED FROM PAGE 5

of only 57 days.⁵ Cytologic appearances of large cell lymphoma and LGL lymphoma are shown in Figures 1 and 2, respectively. The author has found it nearly impossible to definitively differentiate EATL type II and lymphocyte-predominant inflammatory bowel disease (IBD) based on cytology alone. Thus, histopathology +/- immunohistochemistry and PCR for antigen receptor rearrangement (PARR) are recommended for diagnosis.

Carcinoma

Adenocarcinoma is the second most common tumor type of the feline GI tract. Carcinomas are found more commonly in the feline intestine than in the stomach.⁶ Intestinal adenocarcinomas may be intraluminal or intramural and present as plaque-like lesions or masses, often ulcerated. Annular or circumferential intramural lesions may lead to stenosis. Obtaining a diagnostic aspirate of carcinoma can be challenging due to necrosis, septic inflammation associated with ulceration, or the scirrhous connective tissue response that occurs with neoplastic infiltration of the intestinal wall. Cytologic findings include cohesive aggregates of epithelial cells exhibiting cytologic atypia (Figure 3, left panel), often with concurrent findings of mucus and inflammation that may be septic if the mass is ulcerated. Because benign epithelial tumors such as polyps/adenomas can exhibit considerable atypia, especially when inflamed. Likewise, some carcinomas may lack marked cytologic atypia (Figure 3, right panel), so the cytologic distinction between benign and malignant tumors must be made cautiously. For these reasons, histopathology may be required for definitive diagnosis. At the time of carcinoma diagnosis, metastasis to regional intra-abdominal lymph nodes or seeding of the peritoneum (carcinomatosis) has often already occurred.

FIGURE 4

› **Cytologic appearance of intestinal mast cell tumor. Left (aspirate of small intestinal mast cell tumor).** Note mast cells (black arrows) have a central or paracentral small oval dark nucleus with indistinct nucleoli and abundant pale cytoplasm with numerous minute vacuoles (location of non-staining granules). The yellow arrow identifies an eosinophil. (Diff-Quik, 600x magnification.) **Right (histopathology of small intestinal mast cell tumor stained with toluidine blue to highlight granules).** A special stain highlights fine purple granules in the cytoplasm of neoplastic mast cells. (Toluidine blue, 1000x magnification.)

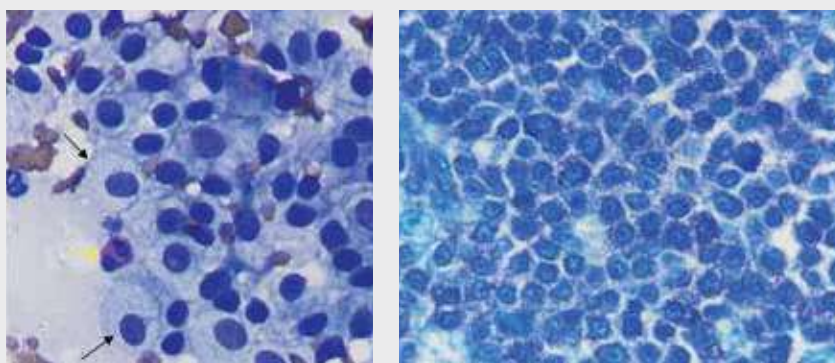


FIGURE 5

› **Cytologic appearance of visceral mast cell tumor in the spleen (left) and liver (right).** Note that on the left panel, neoplastic mast cells (black arrows) have more stainable granules than the distinctive intestinal mast cell tumor shown in Figure 3. The yellow arrows identify an aggregate of hepatocytes among the mast cells. (Diff-Quik, 600x magnification.)

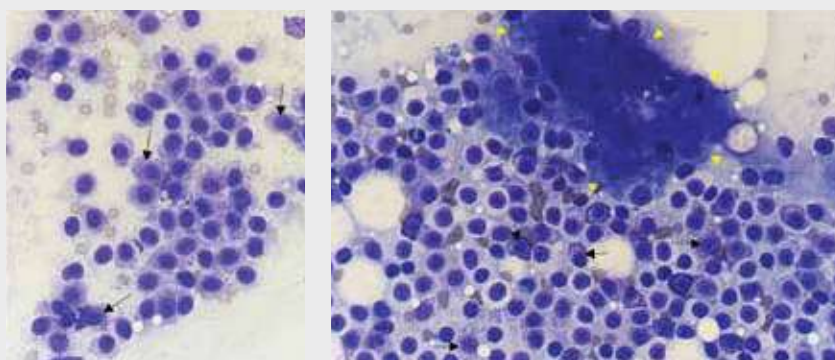
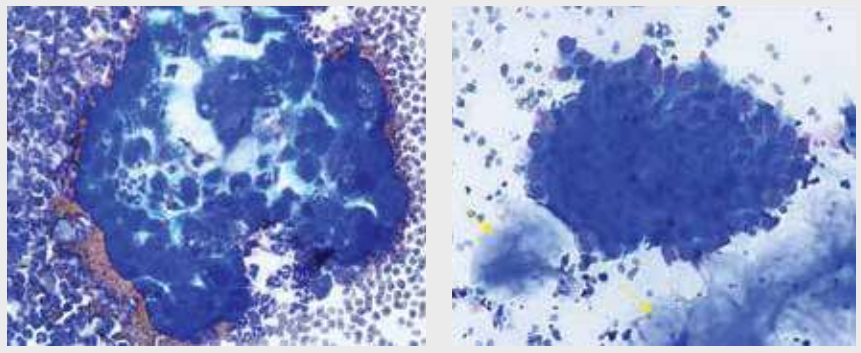


FIGURE 3

› **Cytologic appearance of intestinal carcinoma. Left (small intestine carcinoma).** Note a large cluster of carcinoma cells in a background of neutrophilic inflammation. Cells exhibit marked pleomorphism. They have large oval to irregular nuclei with large angular nucleoli and basophilic cytoplasm with cytoplasmic vacuoles. (Diff-Quik, 600x magnification.) **Right (colonic carcinoma).** Note the cohesive aggregate of neoplastic epithelial cells exhibiting less pleomorphism than the cells shown in the left panel. Smudgy pale blue mucus is present in the background (yellow arrows). Carcinoma diagnosis was confirmed via histopathology. (Diff-Quik, 500x magnification.)



Mast Cell Tumor

Gastrointestinal mast cell tumors generally occur as distinct extramural masses and are more common in the intestine than in cats' stomachs.⁷ They arise from mucosal mast cells rather than connective tissue mast cells and thus are typically less well-granulated (Figure 4). Cytologically, they appear different from other feline mast cell tumors, including visceral mast cell neoplasia (Figure 5). Special stains, including Giemsa and toluidine blue, may help identify metachromatic granules in some but not all feline intestinal mast cell tumors (Figure 4, right panel). Even immunohistochemistry with mast cell marker c-kit may not be positive in all feline intestinal mast cell tumors, which emphasizes the importance of cytopathology in identifying minimal granulation or the distinctive appearance. A variant of mast cell tumor in cats, referred to as feline sclerosing mast cell tumor, can have poorly granulated mast cells that appear

PATHOLOGY

Color Atlas: Feline Gastrointestinal Tumors

CONTINUED FROM PAGE 6

similar, with the addition of plump spindle cells, abundant collagen-type matrix, and numerous eosinophils. This variant of mast cell tumor can be challenging to differentiate from the entity known as feline eosinophilic sclerosing fibroplasia, and the distinction remains controversial.⁸

Summary

Cytologic evaluation of GI masses sampled via ultrasound-guided FNA may be useful in obtaining a presumptive or definitive diagnosis of neoplasia. This method is less expensive and invasive than obtaining samples surgically but does not always obtain sufficient numbers of cells for diagnosis. The risk of hemorrhage and intestinal content leakage can be minimized if appropriate precautions are taken. At the MSPCA-Angell, cytologic evaluation has helped obtain a presumptive or definitive diagnosis of the following common types of feline GI neoplasia if sufficient cellular samples are obtained: lymphoma, carcinoma, and mast cell tumor.

REFERENCES

- 1 Bonfanti U et al. Diagnostic value of cytologic examination of gastrointestinal tract tumors in dogs and cats: 83 cases (2001-2004). *J Am Vet Med Assoc* 2006 Oct 1;229(7):1130-3.
- 2 Penninck D and d'Anjou M. *Atlas of Small Animal Ultrasonography*, 2nd edition. Publisher: John Wiley and Sons, 2015, p. 306.
- 3 Shymalak K et al. Risk of tumor cell seeding through biopsy and aspiration cytology. *J Int Soc Prev Community Dent* 2014 Jan-Apr 4(1):5-11.
- 4 Pohlman LM, et al.: Immunophenotypic and histologic classification of 50 cases of feline gastrointestinal lymphoma. *Vet Pathol* 46:259, 2009.
- 5 Krick EL, et al.: Description of clinical and pathologic findings, treatment and outcome of feline large granular lymphocyte lymphoma (1996-2004), *Vet Comp Oncol* 6:102, 2008.
- 6 Risetto K, et al.: Recent trends in feline intestinal neoplasia: an epidemiologic study of 1,129 cases in the veterinary medical database from 1964-2004. *J Am Anim Hosp Assoc* 47:28-36, 2011.
- 7 Head KW, et al. *Histologic classification of the tumors of the alimentary system of domestic animals. Series 2*, Armed Forces Institute of Pathology, Washington, DC, 2003.
- 8 Halsey CHC, et al.: Feline intestinal sclerosing mast cell tumor: 50 cases (1997-2008). *Vet Comp Oncol* 8:72, 2010.



Marijuana Toxicosis

Katherine McKean, DVM
angell.org/emergency | emergency@angell.org | 617-522-7282

As the changing legal landscape has made marijuana increasingly accessible in the United States, accidental exposures have become increasingly common in our pets. THC toxicosis is a common presentation in both veterinary ERs and primary care clinics. A 2021 survey of veterinarians throughout the US and Canada found a significant increase in reported case numbers compared to just several years prior.

Most cases of marijuana toxicosis follow ingestion of either edible products, plant material, or medical THC preparations. Nearly two-thirds of exposures reported to the Pet Poison Helpline involved ingesting commercial or homemade edible products. These products are often intended to be split into many smaller doses and, when consumed in bolus, can contain very high concentrations of THC. Dogs are reported to have more cannabinoid receptors than humans, maybe even more sensitive than their human counterparts to THC's psychoactive effects.

Marijuana ingestion is not always witnessed, and owners are not always certain whether or where their pets may have gained access to THC-containing products. Unfortunately, no reliable bedside tests are available to quickly and definitively diagnose THC toxicity in veterinary patients. Some clinics rely on commercially available urine drug screening tests intended for human use. While a positive on these dipstick tests may prove diagnostically helpful — particularly when exposure status is unknown — false negatives are common. This may be due to differences between human and canine THC metabolism, error associated with sample handling (THC can bind to glass or rubber collection vials), or testing performed too soon after exposure.

The major toxin present in marijuana is THC. THC is rapidly absorbed and distributed into the tissues, crossing the blood-brain barrier. The onset of clinical signs usually occurs within 60 minutes of ingestion. Signs of toxicosis may include CNS depression, ataxia, hyperesthesia, vomiting or



hypersalivation, hypothermia, dribbling urine, bradycardia or tachycardia, mydriasis or miosis, hypotension, tremors, or in severe cases, seizure or coma. THC's minimum lethal dose is extremely high — greater than 3g/kg. For reference, the average 1g joint contains approximately 150mg of THC. However, with the rise of easily accessible edible preparations, exposure loads can be relatively high, increasing the likelihood that pets may present with severe CNS effects. Other considerations for pets who present with marijuana toxicosis — particularly pets who have ingested edibles — include potential exposure to other toxins (such as chocolate, xylitol, or raisins) and ingestion of foil or other packaging materials.

Often, patients with only mild symptoms can be discharged for care at home, where they should be monitored in a safe environment and kept away from stairs, pools, or other risks of falls or injury. Severely affected animals — those obtunded, respiratory depressed, experiencing seizures, or comatose — should be hospitalized for monitoring and supportive care. Though marijuana has anti-emetic properties, induction of emesis can be attempted in patients presenting within 60 minutes of exposure or when there is clinical suspicion that a significant amount of ingested material remains within the stomach. Care should be taken not to induce emesis in particularly sedated patients or present with CNS depression to reduce the risk of aspiration. Vomiting can be treated with anti-emetics. For patients with a low anticipated risk of aspiration, activated charcoal can be administered. As THC undergoes hepatic recirculation, one to two additional doses can be repeated every six to eight hours. Clinicians can provide fluid support (note that IV fluid therapy is not expected to enhance THC excretion significantly) and heat support as needed based on clinical status. Phenothiazines or benzodiazepines can be given as needed to manage agitation (avoid acepromazine in significantly hypotensive patients). Because THC is highly lipid soluble, clinicians may consider intravenous lipid emulsion (ILE) therapy for patients presenting with severe CNS signs. Intralipid therapy has been used with variable success in veterinary patients — ILE's mechanism of action is still not fully understood, and further research is needed to elucidate optimal dosing



EMERGENCY & CRITICAL CARE

Marijuana Toxicosis

CONTINUED FROM PAGE 8

protocols in dogs fully. A typical protocol is a 1.5ml/kg IV bolus given over one to two minutes, followed by a 0.25ml/kg/min CRI for an additional 30 to 60 minutes. If needed, this dose can be repeated once the patient's serum is no longer lipemic (typically four to six hours following the initial dose). When administering lipids, clinicians should collect blood samples before administration, as the hyperlipemia caused by treatment can interfere with lab assays, and consider that ILE may also impact other lipid-soluble drugs (such as sedatives or anticonvulsants).

Overall, the prognosis for THC toxicosis is generally very good for patients receiving appropriate supportive care. Most dogs will recover over 24 to 36 hours. For patients with high exposure loads (particularly those ingesting high concentrations of edible preparations), full resolution of clinical signs may take several days.

- 3 Fitzgerald et al. (2021). Detecting and quantifying marijuana metabolites in serum and urine of 19 dogs affected by marijuana toxicity. *Journal of Veterinary Diagnostic Investigation*, 33:1002–1007.
- 4 Gwaltney-Brant, S. & Meadows, I. (2018). Intravenous lipid emulsions in veterinary clinical toxicology. *Veterinary Clinics: Small Animal Practice*, 48, 933–942.
- 5 Meola et al. Evaluation of trends in marijuana toxicosis in dogs living in a state with legalized medical marijuana: 125 dogs (2005–2010). *JVECC* 2012; 22, 690–696
- 6 Robben, J.H., & Dijkman, M.A. (2017). Lipid therapy for intoxications. *Veterinary Clinics of North America: Small Animal Practice*; 47, 435-450.

REFERENCES

- 1 Amissah et al. (2022). Prevalence and characteristics of cannabis-induced toxicosis in pets: Results from a survey of veterinarians in North America. *PLOS ONE*, 17, 1–18
- 2 Brutlag, A., & Hommerding, H. (2018). Toxicology of marijuana, synthetic cannabinoids, and cannabidiol in dogs and cats. *Veterinary Clinics: Small Animal Practice*; 48, 1087–1102.

➤ MSPCA-Angell West, Waltham

24/7 Emergency Care

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

Referring veterinarians may alert staff to an incoming case by calling 781-902-8400.

For more information, please visit angell.org/emergency.

Same-Day Urgent Care Appointments

For non-emergent cases, the Urgent Care service at Angell West offers same-day appointments for dogs and cats. They are available Monday through Friday, 8am–6pm by calling **781-902-8400**. Urgent Care appointments are also available through the Angell West Avian and Exotic service by calling **617-989-1561**.

For more information, please visit angell.org/urgent.

Angell West Surgery

The MSPCA-Angell West Surgery Team is available to see **stable fracture cases** as emergency add-on appointments in the mornings on Monday-Friday each week. These patients can be booked in from the appointment for surgery the same day or the following day. If you would like to use this option rather than the Emergency service, please choose the Surgery selection and the Waltham location on the referral form at angell.org/referrals or call Angell West at **781-902-8400**.

Angell West has recently added a **Ziehm Solo FD c-arm**, which provides capability for intra-operative imaging and for the surgeons to perform minimally invasive approaches to fractures, improving outcome and recovery. This is a great option for patients and clients to be seen directly by the Surgery Team, rather than having care facilitated through the Emergency service.





Non-Neoplastic Anal Sac Disease: How to Help Patients Who Scoot, Lick, Leak, or Smell

Meagan R. Painter, DVM, DACVD
angell.org/dermatology | dermatology@angell.org | 617-524-5733

Non-neoplastic anal sac conditions are commonly seen in small animal practice. This article will help the general practitioner update their knowledge and approach to these cases.

Definitions

Anal sacs are paired cutaneous invaginations that are located between the smooth internal and striated external anal sphincter muscles at the anal-cutaneous junction. They are modified adnexal skin structures lined by keratinized, stratified epithelium. These structures serve as reservoirs for tubular apocrine glands opening into the epithelium's keratinized portion. Most of these glands are apocrine in the dog, but some sebaceous glands are also in the ductal region. You will recall that apocrine glands are a type of exocrine gland that bleb and release their material through a duct.

Often the term “anal glands” is used to refer to these structures. This is technically incorrect, and the term “anal sacs” is preferred. The anal sac contains glands.

The contents of the anal sac will typically be a mixture of secretions of both apocrine and sebaceous glands, desquamated epithelial cells, and bacteria. Consistency and color will vary significantly between individuals. All variations in color, consistency, and smell are considered variations of normal and should not be exclusively used to make a diagnosis of an anal gland disorder.

Cats, luckily, appear to have a more even distribution of sebaceous and apocrine glands within their anal sacs. They also have a more lateral location of their ductal opening. Together, these two findings could explain why there appears to be a lower incidence of feline anal sac disease and inflammation.

The anal sac duct is located near the inner cutaneous zone of the anus on the lateral anal margin. If considering a clock face, the anal sac ducts would be located around the 4 o'clock and 8 o'clock time points.



Pathophysiology

Non-neoplastic anal sac disease represents a continuum of conditions affecting the anal sacs. The differentiation between impaction, sacculitis, and abscessation is not clearly defined. Anal sac impaction refers to an overfilling of the anal sac whereby there is retention of anal sac contents. Inflammation is variably found, and pain and discomfort are likely. Anal sacculitis refers to inflammation of the anal sac and ductal lining. Abscessation occurs when the walls of the anal sac are compromised due to secondary infection, leading to localized cellulitis and/or draining tracts. It is estimated that between 4.4% to 15.7% of patients seeking primary veterinary care present with non-neoplastic anal sac disorders depending on the study referenced.

The pathophysiology of non-neoplastic anal sac disease is poorly understood. Many factors can influence normal anal sac function, including changes in stool consistency, decrease in activity level, dietary fiber levels change, pudendal nerve dysfunction, external anal sphincter dysfunction, obese body condition development, inflammatory bowel diseases, endoparasitism, foreign body, and atopic and seborrheic conditions.

What is most important to understand about non-neoplastic anal sac conditions is that they are nearly always secondary to an underlying condition. Only occasionally are these conditions idiopathic.

Making the Diagnosis and Differentials

The small animal practitioner needs to be able to recognize the clinical signs associated with non-neoplastic anal sac disease. Often, these clinical signs are associated with or caused by the pain/irritation from distention of the anal sacs and/or irritation secondary to inflammation and/or infection.

The signalment of your patient is not necessarily going to help you determine if your patient has non-neoplastic anal sac disease. It is possible that there is some breed predisposition toward German Shepherds, Labrador Retrievers,

DERMATOLOGY

Non-Neoplastic Anal Sac Disease: How to Help Patients Who Scoot, Lick, Leak, or Smell

CONTINUED FROM PAGE 10

brachycephalic dogs, and several small-breed dogs. Overweight dogs are not necessarily predisposed. Moreover, middle-aged dogs tend to be over-represented.

Common clinical signs seen by the clinician and/or described by pet owners of dogs (or cats) with non-neoplastic anal sac disease include scooting, discomfort when sitting down, licking or biting the perianal region, tail base involvement, tail chasing, tenesmus, perianal discharge, redness of the tail area, discoloration of the anus, lichenification of the anus, salivary staining of the tail area, moist dermatitis of the perianal region or tail base, and using their back to rub against objects.

Interestingly, dogs who do not have anal sac disease will likely not have a history of anal gland expression. If this is noted in your patient's history, it is important to consider that non-neoplastic anal sac disease could be at play. Anal sac expression typically relieves dogs of their clinical signs from anal sac disease for only three weeks. It is very common to hear this story in general practice. It is suggested that practitioners should pay greater attention to these patients to offer them a more effective long-term solution for their non-neoplastic anal sac disease over routine expression.

The value of cytology for diagnosis of non-neoplastic anal sac disease has been reviewed in various peer-reviewed studies. At this time, it is agreed upon that there are no clinically statistically significant cytological differences between normal dogs and those with non-neoplastic anal sac disease. Therefore, cytology is an ineffective tool for diagnosing impaction and/or sacculitis. For abscessation, the nature of secondary infection is often mixed given the location, and therefore, the value of cytology in helping guide antimicrobial selection is limited. Similarly, a culture of the anal sac contents is largely unnecessary except in cases of abscessation with secondarily infected cellulitis. No differences were noted in Pappalardo et al. (2002) in bacterial species isolated between normal dogs and dogs with pyoderma.

Dogs with non-neoplastic anal sac disease will often have a comorbidity that makes them more likely to develop sacculitis, impaction, and/or abscessation. The most common comorbidity associated with non-neoplastic anal sac disease is atopic disease (canine atopic dermatitis, adverse food reaction, etc). Perianal pruritus was seen in 52% of dogs with canine atopic dermatitis and 50% with adverse food reactions (Maina, 2014). Similarly, the frequency of perianal pruritus in dogs with atopic disease is higher than in dogs with any other diagnosis (Maina, 2014).



The patient's physical examination should thoroughly evaluate the perianal region for alopecia, erythema, excoriation, lichenification, and hyperpigmentation.

When observed, anal sac disease should be described as a separate clinical entity that deserves a specific intervention clinically, depending on the severity of clinical signs and disease. This will help ensure the patient is treated appropriately for all the clinical problems associated with their allergy.

Differential diagnoses for non-neoplastic anal sac disease include but are not limited to: vulvar dermatitis, vaginitis, urinary tract infection, flea allergy dermatitis, perianal tumor, perianal fistulae, tail fold pyoderma/intertrigo, endoparasitism, perianal hernia, perianal gland hyperplasia, rectal foreign body, stricture, prolapse, and trauma, etc. It is essential to rule out vulvar and/or urinary disorders in female dogs who are exhibiting typical clinical signs of anal sac disease (scooting, etc).

Perianal fistulae are an important differential for anal sac abscess/impaction/sacculitis, particularly when draining tracts are noted. You will recall that perianal fistulae development occurs due to an immune-mediated process directed at the perianal tissue. This leads to chronic, multifocal fistulae development within the anal tissue, which often extends to the perianal skin. A key distinction is the involvement of the actual anal tissue, not just the skin of the perineum. However, anal sac rupture, cellulitis, and draining tract can mimic the clinical signs associated with perianal fistulae. Being aware that breed predisposition is often seen, about 80% of cases are seen in German Shepherd dogs that are young to middle-aged. However, this should not be the sole determinant for this diagnosis, as you could see a case of sacculitis in a young German Shepherd that does not have true perianal fistulae. A fistulogram or CT scan may help determine whether or not a draining tract is associated with the anal sac or some other perineal location. Regardless, both diseases are associated with inflammation. It can be challenging and complicated to determine if the clinical signs seen are being caused by inflammation from a defective anal sac that is chronically inflamed or a formal disease process that is leading to fistulae and furunculosis. Care should be taken to avoid a swift diagnosis in these more complex cases. Referral to a dermatologist for care is recommended.

Treatment Options

Several treatment options are described, but most offer minimal, lasting improvement unless the underlying disease is well-controlled. It is important always to bear this in mind when proceeding with your treatment plan for non-neoplastic anal sac disease (i.e., treat the allergies, too!).

Anal gland expression can be helpful. However, it is important to remember that this often will not provide a lasting resolution of clinical signs. Most dogs will have remission of their clinical signs for up to three to four weeks, and then the cycle starts again. We should be open-minded to other treatment options for these patients.

Dietary fiber can help bulk stool, which could help improve glandular emptying during defecation. There are commercially available supplements available which claim to help dogs with anal sac disease by providing an anti-inflammatory dose of omega-3 fatty acids and fiber.

A diagnostic diet trial could be indicated for some patients if an adverse food reaction is suspected as the underlying primary cause of the patient's clinical signs. Remember that the diagnostic diet trial is the only diagnostic test to determine what percentage of your patient's allergic disease is caused by food. Serology, hair, and saliva tests for food allergens are inaccurate and should not be utilized.

DERMATOLOGY

Non-Neoplastic Anal Sac Disease: How to Help Patients Who Scoot, Lick, Leak, or Smell

CONTINUED FROM PAGE 11



Anal sac lavage and infusion is an effective treatment option for dogs with non-neoplastic anal sac disease. This procedure helps to reduce inflammation and secondary infection within the anal sac, thereby preventing recurrence of the clinical signs associated with non-neoplastic anal sac disease. Ideally, this is performed as a sedated procedure. However, some clinicians successfully perform this procedure without sedation in standing dogs.

Equipment needed will include:

- gloves
- lubricant
- 20g short IV catheters and/or a Tom Cat catheter (3.5 French x 5.5")
- syringes with sterile saline x 2
- syringe with 3mL of commercially available steroid/antibiotic/antifungal ointment.

1. The patient is placed in lateral recumbency. The dorsal duct should be worked on for ease of manipulation. The sac is first manually identified, and its contents are emptied. You can describe the contents and/or decide if you would like to perform cytology and/or culture (personal preference, not clinically necessary for treatment at this time).

2. The anal sac duct is identified, and a lubricated catheter tip is gently threaded into the ductal opening. This often requires gentle pressure, wiggles, forward motion, and patience. The author suggests envisioning the anatomy to help ensure the catheter follows the duct path. Never force the catheter tip through the duct. This could lead to accidental ductal rupture and further complications for your patient.

3. Next, you will attach a 3-6mL syringe with sterile saline to your catheter tip and gently pulse/flush the anal sac.

4. After this is clear, attach a 3-mL syringe with your antibiotic/steroid/antifungal ointment (Claro, Mometamax, etc) and gently infuse this into the sac. Most sacs will take about 1-2 mL total.

5. Stop infusion when the medication runs out of the duct into the anus. You can then gently appreciate the anal sac to be full.

6. Flip the patient and repeat! It is recommended that both sides be done, even if one side has been problematic historically.

Troubleshooting notes:

- If the patient is defecating, let them! Do not try to thread your catheter while they are defecating.
- Sedation really helps – if they are only lightly sedated, and resisting this, consider more profound sedation to help reduce anal tone and sphincter reactivity.
- This procedure requires patience! If you can't get one side, flip the patient and try the other.

This procedure can be repeated as needed. In some cases, it should be repeated every two weeks. (However, the author has not had to do this in her practice to date.)

Home care is minimal; some leakage is expected for 24 to 48 hours. However, most dogs are far more comfortable nearly immediately following the procedure. A reduction in clinical signs should be appreciated at home within one to two days.

The majority of non-neoplastic anal sac cases are not secondarily infected. This is an inflammatory problem. We should not routinely prescribe antibiotics for these cases unless there is evidence of abscessation. If you feel compelled to treat secondary infection, using an aerobic culture to guide your selection is best. However, the local treatment by infusion has improved the resolution of these cases and helped us achieve our antimicrobial stewardship goals.

Surgical intervention can be considered after infusion, and medical management for relevant comorbidities can be utilized. Before this, surgical intervention could be premature and unnecessary.

Conclusions

With improved knowledge and commitment, we can improve the lives of our patients who are suffering from non-neoplastic anal sac disease. Infusion procedures should be considered in cases where clinical signs are chronic. It is important to work up the comorbidity of allergic disease, which is frequently seen in these patients.



DERMATOLOGY

Non-Neoplastic Anal Sac Disease: How to Help Patients Who Scoot, Lick, Leak, or Smell

CONTINUED FROM PAGE 12

REFERENCES

- 1 Pappalardo, E, et al (2002). Macroscopic, cytological, and bacteriological evaluation of anal sac content in normal dogs and dogs with selected dermatological diseases. *VetDerm* 2002. 13: 315-322.
- 2 James, Danielle, et al (2010). Comparison of anal sac cytological findings and behavior in clinically normal dogs and those affected with anal sac disease. *VetDerm* 2010. 22: 80-87.
- 3 Maina, E, et al (2014). Perianal pruritus in dogs with skin disease. *VetDerm* 2014. 25: 204-e52.
- 4 Stetina, Kacie, et al (2015). Owner assessment of pruritus and gastrointestinal signs in apparently healthy dogs with no history of cutaneous or noncutaneous disease. *VetDerm* 2015. 26: 246-e54.
- 5 Lundberg, Annette, et al. (2022). Local treatment for canine anal sacculitis: A retrospective study of 33 dogs. *VetDerm* 2022. 33: 426-434.
- 6 Hvitman-Graflund, Katinka, et al (2023). A retrospective study of treatment, outcome, recurrence and concurrent diseases in 190 dogs with anal sacculitis. *VetDerm* 2023. 34: 576-585.
- 7 O'Neill, Dan, et al. (2020). Non-neoplastic anal sac disorders in UK dogs: epidemiology and management aspects of a research-neglected syndrome. *VetRecord* 2



➤ Referrals to Angell

24/7 access to your referred patients' records angell.org/vetportal

NEW Angell Referral Form with Medical Record/Image Upload Option

We are pleased to share that we have streamlined our Angell referral form for a faster and easier referral experience. This form is for both the Boston and Waltham locations. You can also now send medical records, X-ray images, and DICOM links to diagnostic images via our referral form. Visit angell.org/referrals to find a link to the new form and direct contact information for each Angell service.

Critical/Urgent Referrals

Our Referral Office is here to help if you have an urgent case that requires expedited specialty care, please contact our Referral Office at **617-522-5011** from 7am to 10pm or choose the Emergency option on the phone menu to reach our Emergency Desk 24/7.

Dedicated Line for Referring Veterinarians
617-522-5011 | referralservices@angell.org



Introduction to Dermatology in the Exotic Animal Patient

Patrick Sullivan, DVM, DABVP (Avian Practice)
angell.org/avianandexotic | avianandexotic@angell.org | 617-989-1561

Introduction

This article aims to give a general overview of common dermatologic conditions in exotic companion mammals. Additional resources are listed below and should be utilized for more in-depth cases.

Rabbits

Bacterial: Rabbits can develop a number of different types of bacterial infections, requiring veterinary care. Some, such as pyoderma, can be similar to the presentation in dogs and cats, while others are more species-specific. Pyodermas can develop in rabbits for many reasons, including obesity, renal disease, improper diet, improper flooring, and exposure to ectoparasites. Specific conditions such as rabbit syphilis, caused by *Treponema paraluis-cuniculi*, are species-specific and nonzoonotic. This is a venereal disease, spread by direct contact with infected skin or vertically from the dam to kits. Pododermatitis, also known as ulcerative pododermatitis or sore hocks, is a chronic condition involving the plantar surface of the foot. This typically leads to avascular necrosis, granulomatous, and ulcerative dermatitis. Unclean conditions and improper flooring, such as wire bottom cages and cement or hardwood flooring, can contribute to this. Obesity and keeping a doe intact can lead to the development of a large dewlap. This is a common site for moist dermatitis, especially for rabbits that drink from a bowl rather than a water bottle. An uncommon condition, occasionally associated with a large dewlap and excess salivation, is Necrobacillosis, or Schmorl's disease. This condition is caused by *Fusobacterium necrophorum*, an anaerobic gram-negative bacteria commonly found in the GI tract. Symptoms can include abscesses, ulcerations, and necrotic lesions. Surgical debridement and procaine penicillin G are often used to treat this condition.



▷ *Psoroptes cuniculi*, the rabbit ear mite with secondary scaling and crusting of pinna



Fungal: The most common fungal infection noted in rabbits is Dermatophytosis. While often asymptomatic, this condition can lead to alopecia and pruritus on the head, limbs, and nail beds. Unlike dogs, the causative organism in rabbits is typically *Trichophyton mentagrophytes*. Regarding diagnosis, it's important to know that, unlike *Microsporum spp.*, this organism will not fluoresce under a Wood's lamp. This condition may be zoonotic, so it's always important to ask owners if anyone in the household has developed any skin lesions.

Parasitic: Ectoparasitism can be seen in both indoor and outdoor rabbits, although outdoor hutches certainly increase the risk of exposure. *Psoroptes cuniculi*, the rabbit ear mite, causes intense pruritus of the ears and crusting, erythema, and head shaking. *Cheyletiella parasitovorax*, known as walking dandruff, is the rabbit fur mite. This mite is non-burrowing and visible to the naked eye. While most infections are not severe, advanced symptoms can include crusting, scaling, alopecia, and possibly mild pruritus. This parasite is considered zoonotic and can cause pruritic dermatitis in humans. Several types of fleas can affect rabbits, especially those kept outdoors. *Spilopsyllus cuniculi*, *Ctenocephalides felis*, and *Odontopsyllus multispinosus* are commonly identified in affected animals. Myiasis typically occurs in rabbits housed outdoors, although this is occasionally seen in indoor rabbits kept in suboptimal conditions. Flesh fly larvae, *Wohlfahrtia vigil*, hatch from eggs and colonize the area around wounds or soiled, moist skin folds. As the larvae molt, they become more destructive to the tissue,

AVIAN & EXOTIC MEDICINE

Introduction to Dermatology in the Exotic Animal Patient

CONTINUED FROM PAGE 14

› Advanced case of sebaceous adenitis in a rabbit



causing necrosis, secondary bacterial infection, and inflammation. Treatment typically involves physical removal of the maggots, medical management for the parasitism, and secondary infections. Advanced cases carry a guarded prognosis.

Viral: Shope Papillomavirus, also called Rabbit Papillomavirus, is a member of the Papovaviridae family. This virus is spread by arthropods, making outdoor rabbits a higher risk for infection. This virus typically affects wild cottontail rabbits but can also affect domestic rabbits. Symptoms typically include wart-like, keratinized lesions on the ears, eyelids, neck, and shoulders. After a few months, these lesions may resolve or advance to squamous cell carcinomas. This progression is much more common in domestic rabbits than in cottontails.

Cutaneous Neoplasia: While not overly common, several different types of cutaneous neoplasia have been reported in rabbits. Trichoblastomas are the most frequently identified non-viral associated cutaneous neoplasm, but others such as squamous cell carcinoma, trichoepithelioma, sebaceous cell carcinoma, apocrine carcinoma, and malignant melanoma are also seen. Surgical excision is often recommended to treat these conditions, although chemotherapy and radiation therapy may be indicated.

Sebaceous Adenitis: This condition has been identified in several rabbit breeds, although a cause has not been determined. Clinical symptoms begin as scaly dermatitis around the head and neck and can progress to diffuse flaking and alopecia on the entire body. Histopathology shows a replacement



of sebaceous glands with perifollicular lymphocytic infiltrate. Hyperkeratosis, follicular dystrophy, and perifollicular fibrosis are also commonly noted. While the cause of this condition is unknown, it may be linked to thymomas in rabbits, which are seen more commonly in this species than in other companion animals.

Rodents

Bacterial: While not common, primary bacterial pyoderma can develop in guinea pigs and chinchillas. Dental disease, causing excess salivation, can lead to a moist dermatitis and secondary infection as well. *Staphylococcus spp* are often isolated from these infections. Addressing the primary dental disease is crucial for treatment success. Rats seem somewhat resistant to Staph skin infections, but these can be brought on by intense pruritus secondary to ectoparasitism or abscess formation. Gerbils are unique because they seem to develop nasal dermatitis secondary to excess porphyrin secretions from the Harderian gland. This may be brought on by overcrowding or other stressful events. Similar to rabbits, guinea pigs, and chinchillas are also prone to pododermatitis. Soiled bedding, wire bottom enclosures, improper diet, and obesity may all contribute to the development of this condition. Mild cases may involve erythema and inflammation of the palmar and plantar surfaces, while more advanced cases will progress to ulcerations and granulomatous swellings. *Staphylococcus aureus* is the most common bacteria cultured from these lesions. Treatment and correcting potential husbandry issues often involve antibiotics, analgesics, nonsteroidal anti-inflammatory drugs, and potentially vitamin C supplementation for guinea pigs. The spread of the infection into the tendons, joints, and associated bones requires antibiotic therapy for several months and possibly surgery. This degree of infection carries a guarded to poor prognosis.

Fungal: Dermatophytosis is a condition that most often affects young or immunosuppressed rodents, although it can be seen in animals of any age. Group housing at pet stores, as well as the ability of the organism to survive in the environment, can contribute to the spread. Mild lesions can appear scaly, patchy areas on the head, feet, and around the ears. More advanced cases will have areas of alopecia, with crusting and inflammation. These lesions are typically pruritic. *Trichophyton mentagrophytes* are most often isolated in these species, although *Microsporum spp* can also be present. Diagnosis can be made using cytology, culture, or PCR testing. Treatment may involve topical miconazole or enilconazole for small lesions or oral antifungals for more disseminated cases. Cleaning the environment and isolating affected animals will reduce the risk of reinfection. This organism is potentially zoonotic, and the owner should be made aware of this when it's diagnosed.

Parasitic: Guinea pigs are susceptible to a number of ectoparasites, including mites, lice, and rarely fleas. The most common mites identified in guinea pigs are *Trixacarus caviae* and *Chirodiscoides caviae*. *Demodex caviae* and *Sarcoptes spp* are occasionally seen as well. *T. caviae* causes extreme pruritus that may result in self-trauma due to intense scratching. This can lead to secondary infections and scabbing. Louse infestations, typically caused by *Gliricola porcelli* or *Gyropus ovalis*, tend to cause less dramatic skin lesions and pruritus. Animals usually present with thinning fur or areas of alopecia, crusting, and an overall dull coat. While not common, flea infestations have been reported in guinea pigs. *C. felis* is the most common flea seen and is typically spread from another animal in the house. Treatment should be based on the parasite identified and any secondary infections or inflammation that may be present. Ectoparasitism appears rare in chinchillas and is thought to be impeded by their very dense coat.

AVIAN & EXOTIC MEDICINE

Introduction to Dermatology in the Exotic Animal Patient

CONTINUED FROM PAGE 15

Neoplasia: Trichofolliculomas are the most common skin tumors of guinea pigs. They are benign and tend to be located on or near the scent gland. These tumors can grow quite large and occasionally develop centrally located ulcerations, which give way to secondary infections. Surgery is typically curative. Other neoplasms, such as lipomas, liposarcomas, and cutaneous lymphoma, are also commonly seen. In the case of lymphoma, these are typically multicentric, and the prognosis is generally poor despite treatment.

Hedgehogs

Bacterial: Primary bacterial skin infections are not commonly reported in hedgehogs, but they can be seen secondary to ectoparasitism, unsanitary conditions, or possibly due to an allergic dermatitis, although this is considered rare. A single case of pemphigus foliaceus has also been reported, with spine loss, erythema, and epidermal collarettes on the ventrum. This case was treated with dexamethasone injections over 16 months.

Fungal: Like other exotic companion mammals, Dermatophytosis is the most common fungal infection in hedgehogs. *Trichophyton erinacei*, *T. mentagrophytes*, and *Microsporum spp* are routinely cultured. Culture and PCR testing are used for definitive diagnosis.

Parasitic: *Caparinia tripilis* and *Chorioptes spp* are typically implicated in hedgehog infestations, although *Notoedres cati* have also been identified. Symptoms of mite infestation typically include spine loss, flaking, hyperkeratosis, and seborrhea. Identification is made using a tape prep or direct mount with mineral oil. Fleas, *C. felis*, may be passed from a household dog or cat to the hedgehog.

Neoplasia: Neoplasia is very common in hedgehogs and can involve almost any system in the body. Some of the more common tumors associated with the skin are squamous cell carcinoma, cutaneous mast cell tumors, mammary gland tumors, and cutaneous hemangiosarcoma. Diagnosis of these would typically involve a fine needle or excisional biopsy.

REFERENCES

- 1 Hess, Laurie, and Kathy Tater. "18 - Dermatologic Conditions." Ferrets, Rabbits and Rodents Clinical Medicine and Surgery, 3rd ed., Elsevier, St Louis, MO, 2012, pp. 232–244.
- 2 Hawkins, Michelle, and Cynthia Bishop. "23- Disease Problems of Guinea Pigs." Ferrets, Rabbits and Rodents Clinical Medicine and Surgery, 3rd ed., Elsevier, St Louis, MO, 2012, pp. 303–305.
- 3 Paterson, Sue. Skin Diseases of Exotic Pets. Wiley InterScience, 2007.
- 4 Meredith, Anna. "17 - Dermatoses." BSAVA Manual of Rabbit Medicine, British Small Animal Veterinary Association, Gloucester, 2014, pp. 255–263.
- 5 Carpenter, James W., and Christopher J. Marion. Exotic Animal Formulary. 5th ed., Saunders, 2018.
- 6 Greenacre, Cheryl B. "Common Dermatologic Diseases of Exotic Companion Mammals." Western Veterinary Conference 2013, Las Vegas, NV.





Respiratory Emergencies

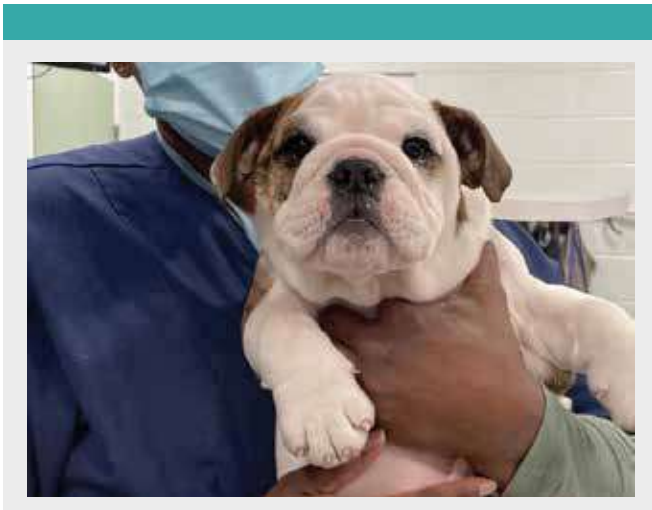
Audrey Koid, DVM, DACVECC
angell.org/emergency | emergency@angell.org | 617-522-7282

Respiratory distress is a common cause of an emergency visit, where prompt recognition and intervention could help save lives. Tools that can help with diagnosis include signalment, history, observation of breathing pattern, auscultation of the heart and lungs, and diagnostic imaging.

Respiratory Physiology

The respiratory system can be divided anatomically into the upper and lower airways, pulmonary parenchyma, pleural space, and thoracic wall/diaphragm. The two main functions of the respiratory system are ventilation and oxygenation. Ventilation is the process of moving air in and out of the lungs. Once at the level of the alveoli, gas is exchanged between blood and air. Hypoxemia can occur due to problems with ventilation and/or gas exchange.

Normal breathing is carbon dioxide-driven. When PaCO₂ rises, CO₂ diffuses into the cerebrospinal fluid, liberating hydrogen ions that stimulate the central chemoreceptors to increase ventilation. This response is magnified if PaO₂ is lowered. PaO₂ becomes the primary driver of respiration when PaO₂ drops below 50 mmHg at high altitudes and in some patients with lung disease (e.g., COPD).



Initial Assessment of a Patient in Respiratory Distress

The age and breed of the patient are great places to start to narrow down the possible causes of respiratory distress. Younger patients are more likely to have something infectious, trauma, or congenital-related, while neoplasia and degenerative conditions are much more likely in older animals. Most, if not all of us, automatically think that a loudly breathing brachycephalic breed has an upper airway obstruction, predisposing them to heat stress/stroke.



This “pattern recognition” helps allow for quick intervention. Returning to the example of the brachycephalic dog with likely upper airway obstruction, sedation, and potential intubation will make more of a difference than oxygen therapy alone.

Next, observe the patient. Is there loud or noisy breathing, usually indicating an upper airway issue? Are there any signs of trauma or abdominal distension? What type of breathing pattern is the patient showing? A restrictive breathing pattern characterized by rapid and shallow breaths in a cat is likely to have pleural effusion. In contrast, a cat with an expiratory effort is likelier to have asthma.

A heart murmur, gallop sounds, pulmonary crackles, or wheezes could narrow your differential further when auscultating patients. If the lung or heart sounds are muffled, there is a good chance of pleural space disease.

Temperature is sometimes overlooked as a tool to narrow down the differential list. A hyperthermic animal is more likely to have an infection (pneumonia or pyothorax) or an upper airway obstruction. In contrast, a hypothermic animal is usually in shock and is commonly seen in cats with congestive heart failure.

Initial Stabilization of a Patient in Respiratory Distress

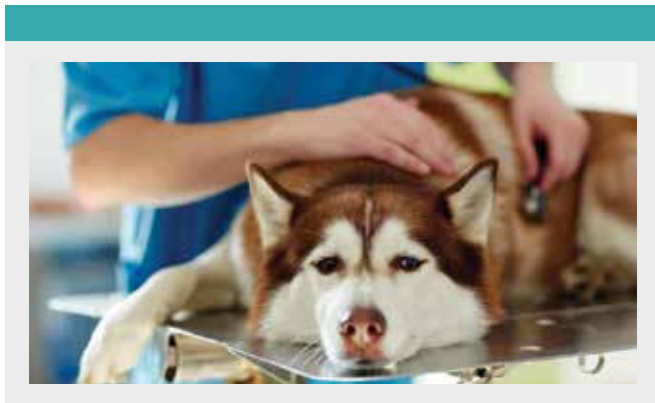
Oxygen therapy is helpful in all cases but more helpful in some than others, as mentioned with the brachycephalic dog with upper airway obstruction. Another situation where oxygen support provides less benefit is in patients with pleural space disease, where a therapeutic thoracocentesis to remove fluid or air allows for better expansion of the lungs with very noticeable improvement in the patient’s breathing.

Respiratory distress is reportedly panic-inducing in people, so sedation could help the patient calm down and decrease the work of breathing. On the flip side, the sedation may depress the patient’s respiratory drive, which could lead to worse hypoxia. Two general rules of thumb are to avoid sedating

EMERGENCY & CRITICAL CARE

Respiratory Emergencies

CONTINUED FROM PAGE 17



animals that appear very tired but catch themselves right before they fall asleep and be prepared to intubate any animal that has been given a sedative. It would also be ideal to have intravenous access for quicker administration of medications, especially if the patient requires intubation. Still, the placement of the IV catheter should be weighed against the stress of restraint.

More definitive treatment would depend on the cause of respiratory distress, but the following drugs are useful to have on hand and can be administered on triage:

- 1) Sedatives:
 - a. Butorphanol 0.1–0.4 mg/kg IV or IM
 - b. Acepromazine 0.005 – 0.02 mg/kg IV or IM
- 2) Furosemide 1–2 mg/kg IV or IM for patients with suspected congestive heart failure
- 3) Albuterol 1–2 puffs/cat if asthma is suspected
- 4) Terbutaline 0.01 mg/kg IM or SQ for asthma or chronic bronchitis

Next Steps

Once the patient is more stable, additional diagnostics such as radiographs, point-of-care ultrasound, lab work, and sedated oral exams can be performed to confirm the cause of respiratory distress. If the cause is unclear in a markedly dyspneic animal based on initial assessment, point-of-care ultrasound may help narrow down the options as it can be done quickly either through the window of the oxygen cage or with a helper administering flow-by oxygen to the patient.

The response to your initial stabilization, if your educated guess as to the cause of respiratory distress is correct, will generally result in a noticeable improvement in the patient fairly quickly. If you do not see this improvement, you must reassess your patient to determine whether they require an escalation of interventions for that particular disease process or whether a different or concurrent disease process is going on. For example, a brachycephalic animal with upper airway obstruction may require more sedatives than what was initially given on triage to calm them down and may also have concurrent aspiration pneumonia. Some brachycephalics may require heavy sedation and intubation for several hours (or longer) to decrease the inflammation in their upper airway enough to be safely extubated.

If any animal appears to be in imminent danger of respiratory fatigue, they should be intubated (and ideally mechanically ventilated) while a more definitive plan is made, even if the ultimate decision is euthanasia. Our goal is to always prevent suffering in the patients we treat.

Conclusions

As the name indicates, respiratory distress can be stressful for all parties involved, especially given its dynamic nature. However, using a systematic approach and the tools that are available at most, if not all, veterinary hospitals, from pattern recognition and situational awareness to good history taking and physical examination to more specific diagnostics such as point-of-care ultrasound and thoracic radiographs, acting quickly to stabilize these patients can make a big difference, and once definitive treatment is implemented, most animals do recover from their respiratory crisis.

REFERENCES

- 1 West J. B. & Luks A. (2016). *West's respiratory physiology: the essentials* (Tenth). Wolters Kluwer.
- 2 Drobatz, K. J., Hopper, K., Rozanski, E. A., & Silverstein, D. C. (Eds.). (2018). *Textbook of small animal emergency medicine*. John Wiley & Sons.
- 3 Silverstein D. C. & Hopper K. (2015). *Small animal critical care medicine* (Second). Elsevier/Saunders.
- 4 Tong CW, Gonzalez AL. Respiratory Emergencies. *Vet Clin North Am Small Anim Pract*. 2020 Nov;50(6):1237-1259. doi: 10.1016/j.cvsm.2020.07.002. Epub 2020 Sep 2. PMID: 32891440.
- 5 Sumner C, Rozanski E. Management of respiratory emergencies in small animals. *Vet Clin North Am Small Anim Pract*. 2013 Jul;43(4):799-815. doi: 10.1016/j.cvsm.2013.03.005. Epub 2013 Apr 20. PMID: 23747261.
- 6 Lisciandro GR, Lisciandro SC. Lung Ultrasound Fundamentals, “Wet Versus Dry” Lung, Signs of Consolidation in Dogs and Cats. *Vet Clin North Am Small Anim Pract*. 2021 Nov;51(6):1125-1140. doi: 10.1016/j.cvsm.2021.07.012. Epub 2021 Sep 14. PMID: 34535335.
- 7 Lemieux E, Rozanski E, Buckley G, Chalifoux N, Kennedy C, Lynch A, Rutter C, Tracy A, Silverstein DC. Indications and outcomes for puppies undergoing mechanical ventilation: 59 cases (2006 to 2020). *Can Vet J*. 2021 Aug;62(8):839-842. PMID: 34341595; PMCID: PMC8281947.

› Major Update: MSPCA-Angell Clinical Lab Renovation Now Complete

We're thrilled to share that construction on our newly renovated laboratory is complete! The final step before we can officially move in August 2025 is installing a critical component: the generator that powers the entire operation.

In the meantime, the Angell lab team is hard at work preparing for the next major phase — planning the complex and carefully choreographed move of lab equipment, supplies, and technology. Their goal is ambitious: to relocate everything within a 36-hour window, ensuring there is *no disruption* to our vital laboratory services.

This new space is bright, modern, and purpose-built for efficiency, accuracy, and safety. It will allow staff to deliver faster, more precise results that directly support the hospital in making timely, informed decisions.

Whether supporting wellness across 50+ animal species or helping law enforcement and adoption teams, the impact of this state-of-the-art lab will be felt in every corner of Angell. This is an exciting step forward for the lab, and we can't wait to see the positive changes it will bring!

angell.org/lab



Office area



Hematology section of the main lab



Chemistry/endocrinology testing area of the main lab

Nuclear Medicine at Angell

Precise Diagnoses | Highly Effective, Long-lasting Treatments

Our Nuclear Medicine service provides cutting-edge diagnostic imaging that offers structural and functional insights into your patient's health.

Services include I-131 therapy, Synovetin OA® injections, thyroid scintigraphy, GRF studies, and transsplenic portal scintigraphy (TSPS).

angell.org/nuclear

› Radioactive Iodine (I-131) Therapy

What distinguishes Angell's I-131 program to treat feline hyperthyroidism:

- Comprehensive thyroid imaging
- Fast access to treatment (consults in 1-2 weeks, treatment in 2-4)
- Individualized iodine dosing to improve effectiveness and reduce side effects
- High-dose treatment available for severe cases
- Extended post-treatment boarding (up to 2.5 weeks)
- Experienced, compassionate care, and sedation for anxious cats
- On-site advanced diagnostic services
- Seamless collaboration with referring veterinarians
- No automatic exclusions for pre-existing conditions

angell.org/i-131 | i131@angell.org



› Synovetin OA

Offered by Angell's Surgery service, Synovetin OA is a low-risk, minimally invasive treatment that relieves canine osteoarthritis pain and inflammation for up to a year. Angell is one of only two hospitals in Massachusetts with trained doctors authorized to deliver this targeted joint injection.

angell.org/synovetin | synovetin@angell.org



› Angell Fall 2025 Continuing Education

Save the Dates

Sunday, October 5, 2025

8:15am – 2:45pm

Live Webinar

5 CE Credits (*pending RACE approval*)

Speakers:

- Daniel Biro, DVM, DACVO (Ophthalmology)
 - Terri Bright, Ph.D., BCBA-D, CAAB (Behavior)
 - Kristine Burgess, DVM, MLA, DACVIM (Medical Oncology)
 - Terry Huh, DVM, DACVIM (Cardiology)
 - Alexander Quilty, DVM, DACVR (Diagnostic Imaging)
-

Wednesday, October 15, 2025

6:15pm – 8:45pm

Live Webinar

2 CE Credits (*pending RACE approval*)

Speakers:

- Shawna Han, DVM (Dentistry)
- Pamela Mouser, DVM, MS, DACVP (Pathology)

PLEASE VISIT [ANGELL.ORG/CE](https://www.angell.org/ce) FOR UPDATES AND REGISTRATION.



STAFF DOCTORS AND RESIDENTS

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

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

CHIEF OF STAFF

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

24-HOUR EMERGENCY & CRITICAL CARE MEDICINE, BOSTON

Alison Allukian, DVM
aallukian@angell.org

Matthew Araujo, DVM
maraujo@angell.org

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

David Carabetta, DVM, DACVECC
dcarabetta@angell.org

Zachary Carlson-Sypek, DVM
zcarlonsypek@angell.org

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

Elton Chan, DVM
echan@angell.org

Nicole D'Addezio, VMD
ndaddezio@angell.org

Michael Gay, DVM
mgay@angell.org

Ashley Goldacker, DVM
agoldacker@angell.org

Alanna Horton, DVM
ahorton@angell.org

Audrey Koid, DVM, DACVECC
akoid@angell.org

Li Lange, VMD
llange@angell.org

Katherine McKean, DMV
kmckean@angell.org

William Orrico, DVM
worrico@angell.org

Nathalie Suciu, VMD
nsuciu@angell.org

Megan Steinhilber, DVM
msteinhilber@angell.org

Sam Vitali, DVM
svitali@angell.org

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

24-HOUR EMERGENCY & CRITICAL CARE MEDICINE, WALTHAM

Jordana Fetto, DVM
jfetto@angell.org

Hilary Kinney, DVM
hkinney@angell.org

Ashley Lockwood, DVM, DACVECC
alockwood@angell.org

Aiden Masri, DVM
amasri@angell.org

Abbey Petronzio, DVM
apetronzio@angell.org

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

Jessica Seid, DVM
jseid@angell.org

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

ANESTHESIOLOGY

Stephanie Krein, DVM, DACVAA
skrein@angell.org

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

Declan Ryan, DVM
dryan@angell.org

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

AVIAN & EXOTIC MEDICINE (W/B)

Elena Buenrostro, DVM
ebuenrostro@angell.org

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

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

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

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

BEHAVIOR (W/B)

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

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

CARDIOLOGY

Alice Chirn, MA, BVetMed
achirn@angell.org

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

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

Barbara Linnehan, DVM, DACZM
blinnehan@angell.org

Sarah Rogg, DVM
srogg@angell.org

DENTISTRY

Shawna Han, DVM
shan@angell.org

Jessica Riehl, DVM, DAVDC
jriehl@angell.org

Joyce Tai, DVM, DAVDC
jtai@angell.org

DERMATOLOGY (W/B)

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

Brooke Simon, DVM
bsimon@angell.org

DIAGNOSTIC IMAGING (W/B)

Naomi Ford, DVM, DACVR
nford@angell.org

Alex Quilty, DVM, DACVR
aquilty@angell.org

Steven Tsai, DVM, DACVR
stsai@angell.org

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

INTERNAL MEDICINE (W/B)

Nyla Bent, DVM
nbent@angell.org

Douglas Brum, DVM
dbrum@angell.org

Maureen Carroll, DVM, DACVIM
mccarroll@angell.org

Anita Fothergill, VMD
afothergill@angell.org

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

Jessica Hayes, DVM
jhayes@angell.org

Antonia Ioannou, BVMS, DACVIM
aioannou@angell.org

Shawn Kearns, DVM, DACVIM
skearns@angell.org

Evan Mariotti, DVM, DACVIM
emariotti@angell.org

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

Ursula Ramalho, DVM
uramalho@angell.org

Vanessa Roza, DVM
vroza@angell.org

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

Lorena Sistig, DVM
lsistig@angell.org

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

Yu-An Wei, DVM
ywei@angell.org

STAFF DOCTORS AND RESIDENTS

CONTINUED FROM PAGE 18

NEUROLOGY

Rob Daniel, DVM, DACVIM
(Neurology)

rdaniel@angell.org

Michele James, DVM, DACVIM
(Neurology)*Service Director*

mjames@angell.org

Jennifer Michaels, DVM, DACVIM
(Neurology)

jmichaels@angell.org

ONCOLOGY

Kristine Burgess, DVM, MLA, DACVIM
(Medical Oncology)

kburgess@angell.org

Megan Cray, VMD, DACVS
(Surgical Oncology)

mcray@angell.org

Kendra Lyons, DVM (Medical Oncology)

klyons@angell.org

Gretchen McLinden, DVM, DACVIM
(Medical Oncology)

gmclinden@angell.org

Jillian Walz, DVM, DACVIM
(Medical Oncology), DACVR
(Radiation Oncology)

jwalz@angell.org

OPHTHALMOLOGY

Daniel Biro, DVM, DACVO

dbiros@angell.org

Martin Coster, DVM, MS, DACVO

mcoster@angell.org

PATHOLOGY

(CLINICAL & ANATOMIC)*

Patty Ewing, DVM, MS, DACVP

pewing@angell.org

Pamela Mouser, DVM, MS, DACVP

pmouser@angell.org

SURGERY (W/B)

Kristen Behrens, DVM

kbehrens@angell.org

Matthew Boules, DVM

mboules@angell.org

Sue Casale, DVM, DACVS

scasale@angell.org

Hadley Gleason, VMD, MS, DACVS
(Waltham)

hgleason@angell.org

Dilraj Goraya, DVM

dgoraya@angell.org

Michael Pavletic, DVM, DACVS
Service Director

mpavletic@angell.org

Jennifer Peterson-Levitt
DVM, MS, DACVS-SA

(Boston & Waltham)

jpetersonlevitt@angell.org

Sophia Topulos, DVM, DACVS
(Waltham)

stopulos@angell.org

Nicholas Trout

MA, VET MB, DACVS, ECVS

ntrout@angell.org

URGENT CARE

BY APPOINTMENT ONLY, WALTHAM

Tamara Kremer Mecabell, DVM

tmecabell@angell.org

Natasha Pakravan, DVM

npakravan@angell.org

Julie Shields, DVM

jshields@angell.org

ANGELL AT ESSEX

Heidi Broadley, DVM

hbroadley@angell.org

Sara Gardiner, DVM

sgardiner@angell.org

(W/B) Services available at both our Waltham and Boston locations

*Boston-based pathologists and radiologists serve both Boston and Waltham locations

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

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

MSPCA-ANGELL
 350 South Huntington Avenue
 Boston, MA 02130
 617-522-5011
angell.org

MSPCA-ANGELL WEST
 293 Second Avenue
 Waltham, MA 02451
 781-902-8400
angell.org/waltham

ANGELL AT ESSEX
 565 Maple Street
 Danvers, MA 01923
 978-304-4648
angell.org/essex

MSPCA-ANGELL CLINICS
 Boston | Cape Cod | Methuen
angell.org/clinics

Please consider adding Angell's Emergency service/617-522-7282 to your after-hours phone message.

Our Service Locations

BOSTON & WALTHAM

Avian & Exotic Medicine
 617-989-1561

Behavior & Training
 617-989-1520

Cardiology
 617-541-5038

Dermatology
 617-524-5733

Diagnostic Imaging
 617-541-5139

Internal Medicine
 B: 617-541-5186
 W: 781-902-8400

Surgery
 617-541-5048

Urgent Care*
 781-902-8400

BOSTON ONLY

Anesthesiology
 617-541-5048

Dentistry
 617-522-7282

I-131 Therapy
 617-522-7282

Neurology
 617-541-5140

Nuclear Medicine
 617-541-5139

Oncology
 617-541-5136

Ophthalmology
 617-541-5095

Pathology
 617-541-5014

*Available only in Waltham

