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Winter 2018 ■ Volume 12:1 ■ angell.org ■ facebook.com/AngellReferringVeterinarians

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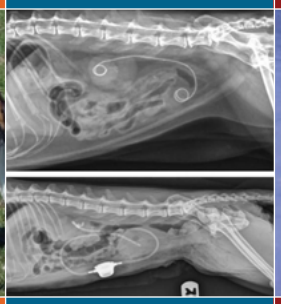

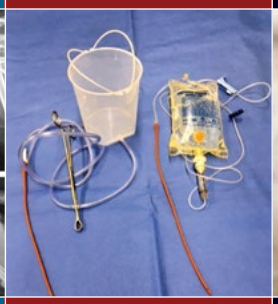

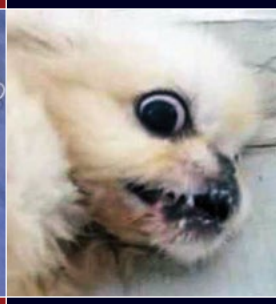

■ TECH TIP: POC COAGULATION TESTING ON PAGE 13 ■

SURGERY

3D Printing in Veterinary Medicine

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The technology of 3D printing has made tremendous strides in regard to materials and techniques used by medical and veterinary professionals, allowing new applications in everyday procedures. Additive 3D printing works by laying down material in thin layers on top of each other that, when cured, form a full model. Imagine a simple desktop printer: if it printed a single square on a page, it would be a 2D printer. If it were able to print numerous squares on top of each other, these squares would stack up into a cube. This is the basic premise of additive 3D printing.

There is a wide variety of materials available including rubbers, plastics, ceramics, metals, and glass. The plastics can range from soft and flexible nylon that allows for dynamic functionality to plastics with glass fibers inside for increased rigidity and structural strength. Adding color and fillers allows for specific blends and unique applications such as training models, functional prosthetics, and lab fixtures and equipment.

In order to make a 3D model, the 3D data is acquired (such as from MRI or CT), processed, numerically modeled, and printed. One study found that 3D models of bone were more accurately represented when data was acquired using CT versus MRI.[1] The medical information from the CT or MRI is exported as a DICOM (digital imaging and communications in

(CONTINUED ON NEXT PAGE)

EMERGENCY

Polycythemia/Erythrocytosis

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When a patient is found to have a significantly elevated red blood cell (RBC) count, we usually report that they have polycythemia. However, there is often confusion between the terms polycythemia and erythrocytosis. Polycythemia can be used more appropriately to describe situations in which all blood cell lines are increased (RBCs, leukocytes, and thrombocytes/platelets). Erythrocytosis is defined as an increase in solely RBCs. This increase can be determined by measuring packed cell volume (PCV), hematocrit (Hct), hemoglobin (Hb) or RBCs, all of which would be increased. In our patients, increases in leukocytes and platelets are not usually noted concurrent with erythrocytosis. For the purposes of this article, the terms polycythemia and erythrocytosis will be used interchangeably, as this is how they are generally used clinically.

The first question to consider is at what PCV the presence of erythrocytosis is described. In dogs, the upper limit for PCV is considered to be 55%. In cats, the upper limit for PCV is lower and is stated to be 45-48%. At PCVs above these cut-offs, erythrocytosis is reported. Clinical signs of erythrocytosis do not usually appear until the PCV reaches 60%, and PCVs described in the literature can be upwards of 85%. Some breeds (Greyhounds, some other sight hounds, and some Dachshunds) normally have a higher PCV.

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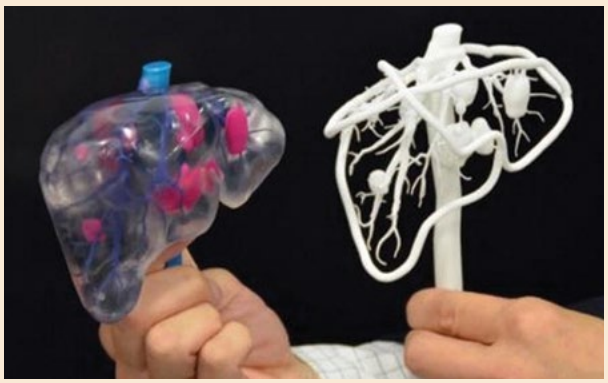
SURGERY

Continued from page 1

FIGURE 1

↘ Image of 3D printed hearts with color coding for different working attributes used in teaching.



FIGURES 2 & 3

↘ Derby, a dog with 3D printed prosthetics. Photo source: https://tripawds.com/2015/10/21/pet-prosthetics/.

medicine) file to be used in a software that is compatible with the printer that helps to create the 3D model. The cost of imaging for CT or MRI can be quite high, but the cost of making the 3D model can be low in comparison; often the materials are less than $50 depending on the materials used.

In human medicine, 3D models have been reported to be used in spinal surgery; maxillofacial surgery; oncologic, cardiovascular and visceral surgery; and pelvic surgery. The models have been shown to improve the diagnosis and treatment of pathology. They also help surgeons to prepare for complex and risky operations, and allow them to have a chance to practice on a patient-specific model that is to scale. It can help the surgeon be prepared for the potential risks and complications prior to the actual live surgery.[2] Several papers have shown that 3D printers/models may reduce operation times, complications, and the cost of surgery rooms overall.[1] In Japan, the cost of a 3D printed model used for surgery is covered by insurance.[2]

Having a model in advance can help to customize instruments and devices such as plates that will be used for that specific surgery. Models can also be used in bone replacement or prosthetic devices. They have also been shown to help train veterinary students/medical students to allow training prior to working on live patients.[3] 3D printing can also aid in the position of radiation for cancer treatment and to create implants to be used for jaw fractures and prostheses. Bioprinting is the printing of live cells and tissues that are then implanted into the body.

One of the most common surgical applications for 3D printing in veterinary medicine is for angular limb deformities. CT is useful for angular limb deformities but does not give the full picture of complex multiplanar deformities. 3D printers can help the surgeon physically examine the angular deformity and make an accurate treatment plan. Once an angular limb deformity model is created, surgeons can practice the correction in advance. This will help to ensure the best surgical approach and fixation, facilitate shorter anesthesia time, and potentially reduce infection rates. Once the model is created, the angular deformity is measured and a wedge is cut from the bone and a bone plate and screws, or an external fixator is placed to allow stability for the bone to heal. For example, DICOM data from CTs were used by the surgery team at Angell to create 3D models for angular limb deformities. The model is then used to practice surgery and plan for the correction in advance.

FIGURE 4

↘ The below picture is a 3D printed model of a canine forelimb. It was printed at Angell Animal Medical Center in preparation for angular limb correction surgery.

On occasion, a patient will present with a more complex disease process causing lameness such as a malunion from an old trauma. In this situation, often radiographs are not sufficient to completely evaluate the severity of the injury and abnormal conformation. A 3D printed model in these cases might help to decide if there is a surgical correction option and the prognosis. Another example of a challenging case is a young, year-old Labrador who presented for lysis of the patella from infection that caused severe destruction and irreversible damage of the patella. A 3D printer was used to create an implantable patella prosthetic for the dog using a PC-ISO based polycarbonate polymer. After aggressive physical therapy, the patient has been walking increasingly well since surgery. (See Figures 5 and 6).[4]

3D models can also be performed to help assist with neurologic surgery. For example, a model was created on a two pound Yorkshire-Terrier with an atlantoaxial subluxation.[1] This could also be used for a vertebral fracture to assess the fracture configuration and complexity and to properly plan the type of repair needed for stabilization. Practicing complex brain surgery in advance can also be facilitated by having a 3D printed model of the particular brain tumor prior to the actual surgery.

Printing a 3D printed skull in advance can help in numerous ways for maxillofacial surgery. It can help to accurately determine the extent and location of a cancerous mass or trauma/injury. It can determine resectability or see how close lesions are to the patient's brain or eyes in order

SURGERY

Continued from page 2

FIGURES 5 & 6

↘ The picture on the left is of a lateral stifle radiograph, which shows aggressive lysis of the patella and also of the distal femur. The picture on the right shows the 3D printed bone model of the same case to prepare for surgical implantation of the patella into the patient. Case and pictures courtesy of Dr. David Hummel at Skylos Sports Medicine, MD.

FIGURE 7

↘ The below picture is a 3D printed model of a canine skull. It was printed at Angell Animal Medical Center.

to plan the best possible approach/repair. Similarly to orthopedic surgery, it can also help to choose implant or plate sizes prior to the procedure. 3D printing is also extremely helpful for maxillofacial surgery, such as a horse with a comminuted orbital fracture and TMJ that was assisted by evaluating a 3D model preoperatively.[5] Printing 3D models helped in one study of three dogs with orbital and peri-orbital masses. The authors found that this helped to improve surgical planning and provided another modality to visualize the full extent of the disease.[6]

3D models have also been evaluated for assisting veterinary students' education. One study compared the efficacy of textbooks, computerized models, and 3D printed models in teaching anatomy in MRI images of the distal equine limb. The results found that 3D models help students to perform better and have a better learning experience.[1] In human medicine, a study found that surgeons were able to practice procedures on 3D models in a standardized format that may help to minimize the need for cadavers.[7]

Some of the main limiting factors of 3D printing are the size limitations and time limitations for creating a 3D model. Most often, the largest size that is able to be printed is a 6" cube. For example, Angell's current 3D printer can print a model that is about the size of a canine skull. But in order to print and practice for an angular limb deformity, the limb would be printed in two sections (if it is a large patient) and then placed together to create one specimen. The biggest limitation for 3D printing currently is the amount of time required to print the model, which can be almost 24 hours depending on the printer. For this reason, the main application of 3D printing to veterinary medicine is for elective planned surgeries to study in advance. It will not be useful for emergent cases that do not allow for enough time to print the sample model. 3D printing is a technological tool that can help doctors to fully assess pathologic disease in order to plan surgical cases, create or prepare implants, study and practice surgical technique, and many other goals to help our animal patients.

With clinical help from David Elentukh and Megan Kudzma.

1 Hespel A-M, Wilhite R, Hudson J et al. Invited Review—applications for 3D printers in veterinary medicine. Vet Radiol Ultrasound Vol 55. No 4, 2014, 347-358.

2 http://www.3ders.org/articles/20160127-japanese-medical-insurance-to-cover-cost-of-3d-printed-organ-models.html)

3 Waran V, Narayanan V, Karyppiah R et al. Injecting Realism in Surgical Training—Initial Simulation Experience with Custom 3D Models. Journal of Surgical Education, 2014, 193-197.

4 Case in progress—case and pictures courtesy of Dr. David Hummel at Skylos Sports Medicine, MD.

5 McMaster M, Caldwell F, Gillen A et al. Reconstruction of a complicated orbital depression fracture with medial wall and globe repositioning in a horse: a collaboration across disciplines and specialties. Veterinary Surgery, 2016, 45; 529-535.

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EMERGENCY

Continued from page 1

Once erythrocytosis has been identified, the next question is whether it is a relative erythrocytosis (far more common) or an absolute erythrocytosis. This can usually be determined based on the patient's history and physical exam.

Relative erythrocytosis occurs when there is a decrease in the blood plasma volume, rather than a true increase in the number of RBCs present. Causes include a decrease in fluid intake, a rapid loss of fluid, such as would occur with gastrointestinal signs or burns, or fluid shifts. This is a situation of hemoconcentration and can also be termed a spurious polycythemia. A transient relative erythrocytosis can occur in dogs with splenic contraction, but typically this will not result in an increase in PCV over 60%. Patients with a relative erythrocytosis should show concurrent signs of dehydration (tachycardia, prolonged CRT, hypotension, concentrated urine, pre-renal azotemia, etc.), typically do not show clinical signs attributed to erythrocytosis, and have a history supporting the development of dehydration. Additionally, there should be an increase in total protein along with the increased PCV. However, one exception to this could be a dog with hemorrhagic gastroenteritis. In these cases we can see a very high PCV but with a normal or even low total protein level due to gastrointestinal losses. In cases of relative erythrocytosis, the PCV typically does not exceed 65% and will return to normal with appropriate fluid therapy, which is the mainstay of therapy for these cases.

It is also important to consider iatrogenic causes for erythrocytosis. Aggressive blood transfusions could result in erythrocytosis. Additionally, patients receiving erythropoietin (EPO) supplementation could develop a high PCV if EPO therapy is excessive.

Absolute erythrocytosis occurs when there is a true increase in the number of red blood cells present. Typically, this is due to increased erythropoiesis (production of RBCs), rather than a longer survival of existing RBCs. Erythropoiesis is controlled by oxygen delivery to the kidney and EPO, a hormone. EPO synthesis is increased as a result of renal hypoxia; acceptable or over supply of oxygen to the kidneys will result in decreased EPO synthesis. Absolute erythrocytosis cases can be further classified into primary and secondary, and testing for suspected cases should include labwork (CBC with reticulocyte count, chemistry panel, blood gas analysis, urinalysis), imaging (thoracic radiographs, abdominal radiographs or ultrasound, echocardiogram), and EPO level. Unfortunately, examination of the bone marrow is not helpful in distinguishing the cause of erythrocytosis; typically, the bone marrow will exhibit erythroid hyperplasia regardless of the cause. Measurement of EPO levels can be difficult, as human assays are often the only available method and there can be overlap in measured values between affected patients and normal animals.

Primary erythrocytosis is defined as cases in which increased erythropoiesis is not due to an increase in EPO. These cases can be either congenital or acquired, and EPO levels should measure low in these patients. In primary congenital cases, there are EPO receptor mutations or EPO receptor hypersensitivity. These patients are usually diagnosed with erythrocytosis at a young age, would not be expected to have concurrent thrombocytosis or leukocytosis, and their disease will not progress. In acquired cases, a myeloproliferative disease of the bone marrow results in clonal expansion of a single hematopoietic stem cell, and this can be termed polycythemia vera. In these cases, one could theoretically see increases in all three blood cell lines (RBCs, WBCs, platelets). These patients can progress to having leukemia or myelofibrosis. Polycythemia vera is a diagnosis of exclusion; there is no test in animals to differentiate polycythemia vera and congenital erythrocytosis, other than considering the history and time of diagnosis.

Secondary erythrocytosis is defined as cases in which increased erythropoiesis is due to an increase in EPO. These cases can be considered either appropriate or inappropriate—this classification is based on the presence of systemic hypoxia causing an increase in EPO (appropriate) or the absence of systemic hypoxia but with an increased EPO level (inappropriate). Measurement of EPO levels can be helpful in diagnosis of secondary polycythemia, as an elevated level is diagnostic; levels can be increased up to 50 times normal. However, on occasion, EPO levels can measure as normal or low in these cases, so a low level does not rule out secondary erythrocytosis.

Secondary appropriate erythrocytosis is diagnosed in patients with concurrent systemic hypoxia. These patients will have an arterial oxygen saturation <92%. The vast majority of these cases are due to situations that result in right to left shunting of blood. This results in circulating blood that is less oxygenated than normal, as some of it is not passing through the lungs. Many congenital heart defects can result in right to left blood shunting, including ventricular septal defects (VSD), tetralogy of Fallot, and reversed patent ductus arteriosus

FIGURE 1

↘ Some breeds, like Greyhounds, normally have higher PCV.

EMERGENCY

Continued from page 4

(rPDA). Other causes of secondary appropriate erythrocytosis could include chronic pulmonary disease or chronic upper airway obstruction (i.e. bulldogs), but usually an extreme increase in RBC count is not noted in these cases. Rarely, patients may have defective hemoglobin that is not able to carry oxygen as effectively; examples include patients with methemoglobinemia. Also, animals living at high altitude may have a higher PCV due to chronic hypoxia. Carbon monoxide toxicity can cause erythrocytosis when there is recurrent exposure. Clinically, these cases can be divided based on the presence or absence of cyanosis: patients with heart defects, airway disease, or methemoglobinemia will exhibit cyanosis, whereas those at a high altitude or with carbon monoxide exposure will not. Patients with heart defects usually have a concurrent heart murmur, except for rPDAs, which often do not.

Secondary inappropriate erythrocytosis is the term used to describe patients that have an increase in EPO but are not suffering from systemic hypoxia. Conditions of the kidney can cause this, including neoplasia (carcinoma, lymphoma, nephroblastoma), amyloidosis, infection, or inflammation. Tumors that secrete EPO can also be located elsewhere in the body other than the kidneys; they have been reported in dogs in the cecum (cecal leiomyosarcoma) and liver (hepatoma). These neoplasias can be either benign or malignant.

Of all of the causes of erythrocytosis discussed above, it appears that the most common cause for absolute erythrocytosis is neoplasia—classified as a secondary inappropriate erythrocytosis. In these cases, there is usually a neoplastic process in a kidney.

Most clinical signs of absolute erythrocytosis are related to an increase in blood viscosity. As viscosity increases, there is a decrease in blood flow in capillaries, resulting in tissue hypoxia, sludging, and thrombosis. This impairment of the microcirculation occurs in the brain and is responsible for neurologic clinical signs including behavior changes, ataxia, blindness, and tremors that can progress to seizures. These neurologic signs are what prompt most owners to present their pet for evaluation. Patients may also develop hemorrhage due to increased viscosity; epistaxis, hyphema, and GI bleeding have been noted.

It is crucial to distinguish relative erythrocytosis from absolute erythrocytosis, as the treatment varies significantly. In cases of relative erythrocytosis, the treatment is aggressive IV fluid, as discussed above. With absolute erythrocytosis, initial treatment is removal of whole blood. This is most often performed via phlebotomy, but leeches could also be considered. Recommendations are for removal of 10-20 mL/kg of blood at a time consecutively until clinical signs are improved. The removal of 20 mL/kg of whole blood should decrease the PCV by roughly 15%. The goal PCV varies somewhat based on the cause of erythrocytosis. For cases with secondary appropriate erythrocytosis, a higher PCV (60%) is acceptable after phlebotomy, whereas for cases of primary erythrocytosis, the goal is PCV <55% (dogs) or 50% (cats). Typically, blood removal is performed via jugular phlebotomy. If repeated removal of blood is expected in a short time period, a jugular catheter that bleeds back may be helpful. If leeches are used, each leech is reported to remove between 5-10 mL of blood. As blood is removed, it is important to consider the patient's volume status and provide IV fluid therapy as needed to preserve blood volume. In patients exhibiting seizures, traditional anti-seizure medications alone are unsuccessful.

If the underlying cause for erythrocytosis can be corrected, it should be treated (i.e. removal of an EPO secreting tumor, prevention of carbon monoxide exposure). Prior to surgery, the patient should be stabilized via phlebotomy and IV fluids, as indicated. However, most patients diagnosed with absolute erythrocytosis do not have a curable condition and need to be treated throughout their lifetime. Often, signs can be controlled with intermittent phlebotomy, every 1-2 months based on sequential PCV/TS monitoring. Chronic phlebotomy can result in hypoproteinemia and iron deficiency; administration of plasma and iron supplementation can be considered in these cases. If the PCV and clinical signs cannot be controlled with phlebotomy, myelosuppressive drug therapy with hydroxyurea can be considered. Hydroxyurea therapy should be initiated following phlebotomy to reach the goal PCV for the patient, and it has been used successfully in veterinary patients with primary and secondary erythrocytosis. CBCs must be carefully monitored during therapy to determine the appropriate dose as well as to monitor for myelosuppressive complications. Radioactive phosphorus has also been used as a myelosuppressive agent. Antithrombotic therapy can also be considered in patients with absolute erythrocytosis.

As erythrocytosis is a rarely diagnosed condition, there is a scarcity of data regarding survival. In general, the prognosis for patients with primary erythrocytosis is guarded, whereas the prognosis for patients with secondary erythrocytosis depends on the underlying cause. Patients with suspected erythrocytosis can benefit from referral to Angell. In patients with acute neurologic signs, the emergency and critical care service can stabilize these patients and help to decrease the PCV rapidly to resolve signs. Our specialists can then perform advanced diagnostic tests to look for the underlying cause for erythrocytosis. While a rare condition, erythrocytosis should be considered in patients with an elevated PCV without other signs and in particular in patients with an elevated PCV and concurrent neurologic signs.

For more information about Angell's Emergency/CriticalCare service, please visit **angell.org/emergency** or call MSPCA-Angell West in Waltham at **781-902-8400**.

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Summary of the ACVIM Consensus Recommendations on the Treatment and Prevention of Uroliths in Dogs and Cats

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In an age of advancing technologies, the management of urolithiasis poses a unique opportunity to advance veterinary care. In 2016, a panel of veterinary internists with special interest in urology published a consensus statement in the *Journal of Veterinary Internal Medicine* to offer suitable contemporary strategies in the management of urolithiasis.[1] The panelists recognized that the availability of minimally invasive technology or willingness to travel to centers of expertise may be a limitation, but the recommendations were selected in the patient's best interest as if all options were available. These recommendations apply to stable patients.

TREATMENT OF LOWER URINARY TRACT (BLADDER AND URETHRA) UROLITHS

1. Struvite uroliths (Figure 1) should be medically dissolved while treating associated UTI, as this is a highly effective approach, unless dissolution foods are not tolerated, or the uroliths cannot be adequately bathed in urine (extremely large uroliths, or due to urinary obstruction). Struvites are usually produced by urease-producing bacteria, however, sterile struvites are also amenable to dissolution (usually in 2-5 weeks). Urethral obstruction as a potential risk of dissolution has not been reported in the veterinary literature.

FIGURE 1

2. Urocystoliths associated with clinical signs should be removed by minimally invasive procedures including medical dissolution, voiding urohydropropulsion, or basket retrieval, if they are small enough to pass through the urethra. Otherwise, intracorporeal laser lithotripsy or percutaneous cystolithotomy can be used. Authors recommend avoiding cystotomy to decrease the risk of suture-induced uroliths, length of hospitalization and anesthesia time, and the number residual stones due to improved visualization. Dissolution of urate uroliths should be attempted before removal and can usually be accomplished within 4 weeks by feeding a purine-restricted, alkalinizing, diuretic diet, along with allopurinol therapy (15 mg/kg PO q12h). Dissolution has not been possible in dogs and cats with uncorrected liver disease. Similarly, dissolution of cystine uroliths can be achieved by feeding an alkalinizing, protein-restricted diet with the addition of 2-mercaptopropionylglycine (Thiola) at 15-20mg/kg PO q12h.

3. Nonclinical nondissolvable urocystoliths unlikely to cause urinary obstruction (due to their size) do not require removal but monitoring and appropriate client education about the signs of urinary obstruction.

4. Nonclinical urocystoliths likely to cause obstruction (diameter similar to the urethra) should be removed by minimally invasive procedures.

5. Urethroliths should be managed by intracorporeal lithotripsy and basket retrieval, which is a safe, quick, and highly effective procedure.

6. Urethral surgery is discouraged and considered a salvage procedure. Urethroliths should be repositioned by retrograde urohydropropulsion into the bladder and removed by minimally invasive procedures.

TREATMENT OF UPPER URINARY TRACT (KIDNEY AND URETER) UROLITHS

1. Nephrolithiasis: Only problematic nephroliths (causing outflow obstruction, renal parenchymal compression, recurrent infections, or pain) require treatment. Nephroliths were not found to significantly affect progression of CKD in cats and the same is likely true for dogs. Suspected struvite nephroliths, which represent about 20-30% of upper urinary tract uroliths, should be dissolved. Suspicion is based on alkaline urine, UTI caused by urease-producing bacteria and moderate radiopacity. If ureters are obstructed, they should be stented to allow medicated urine to reach the calculus and for evacuation of bacteria and inflammatory debris. Over 90% of nephroliths and ureteroliths in cats are calcium oxalates; therefore, dissolution is not recommended. Problematic nephroliths should be removed by endoscopic nephrolithotomy or extracorporeal shockwave lithotripsy (applicable only in dogs with nephroliths < 1.5cm). Larger nephroliths require ureteral stenting.

2. Hydronephrosis and hydroureter proximal to an obstructive lesion visualized by ultrasound are sufficient to diagnose ureteral obstruction. Partial and complete ureteral obstructions should be managed as an emergency as they can result in a rapid and lasting decrease in renal function. Decompression by subcutaneous ureteral bypass, ureteral stent, or traditional surgery is necessary when medical management fails or is contraindicated in severely ill patients (Figure 2). Interventional procedures have lower morbidity and mortality rates than traditional surgical options; therefore, referral is advised. Renal function can be maximized by relieving obstruction even after a long time (>8 weeks); therefore, the duration of obstruction should not be a deciding factor.

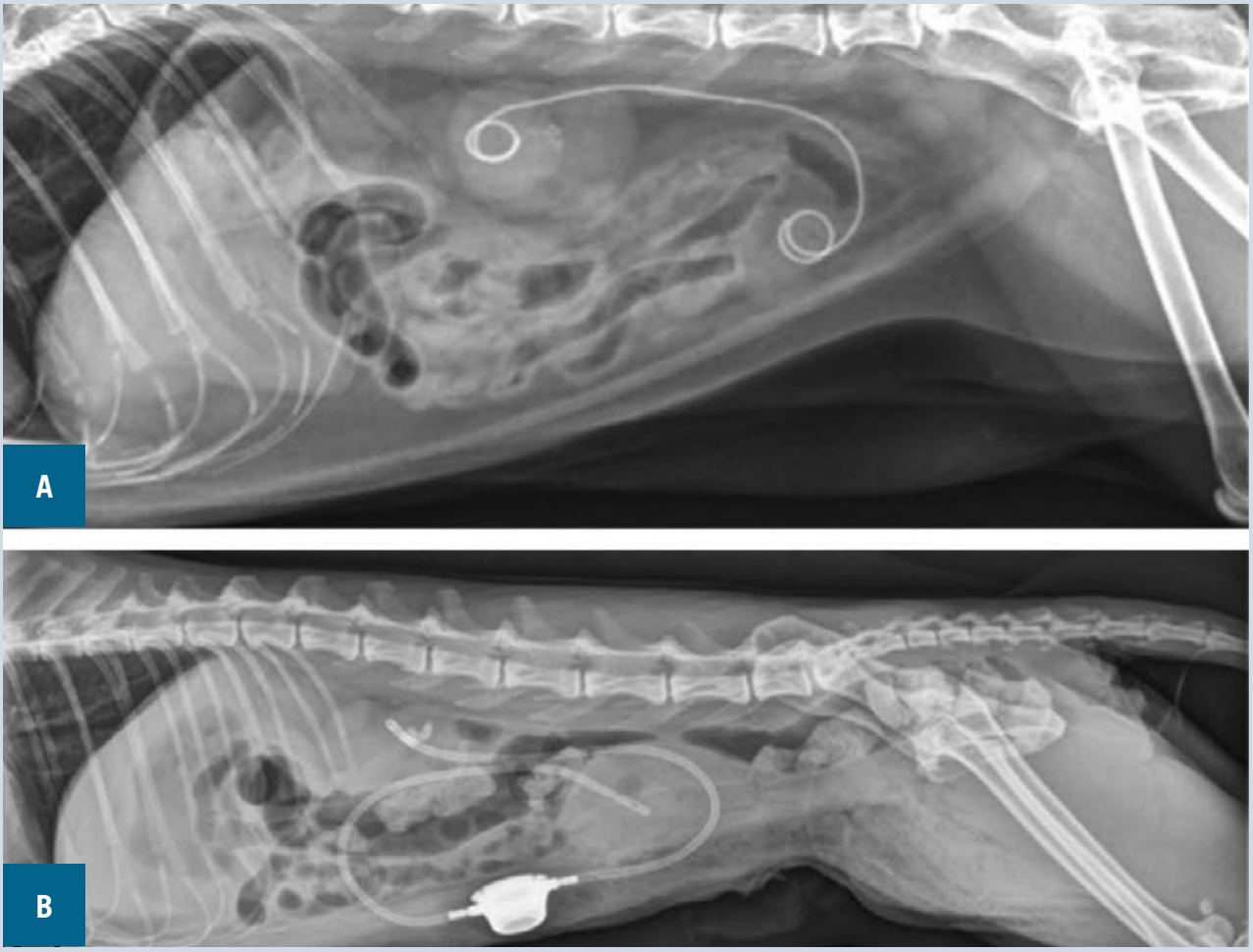
3. Medical treatment for obstructive ureteroliths can be considered for 24-72 hours including fluid diuresis, mannitol, alpha adrenergic antagonists, and tricyclic antidepressants. Antibiotics should be administered in dogs because of the high incidence of concurrent UTI and pyelonephritis. Medical management is effective in only 8-13% of feline cases.

4. Subcutaneous ureteral bypass or ureteral stenting in cats and ureteral stenting in dogs are considered the treatment of choice for ureteral obstruction.

Continued from page 6

FIGURE 2

↘ Lateral radiographs of cats treated with a ureteral stent (a) and subcutaneous ureteral bypass device (b).



UROLITH PREVENTION

Additional therapeutic strategies are needed to prevent urolith recurrence or stent encrustation, based on quantitative urolith analysis.

Dogs, and especially cats with sterile struvites, should be fed acidifying therapeutic diets with low magnesium and phosphorus (goal: pH<6.5). The most important step in prevention of infection-induced struvites is elimination of UTI based on urine culture, as urinalysis is an insensitive marker for UTI. In patients with recurrent UTI, structural and functional risk factors should be investigated.

To minimize calcium oxalate urolith recurrence, high-moisture (>75% water) diets are recommended to achieve USG < 1.020 in dogs and < 1.030 in cats. High sodium diets are not a substitute for high-moisture diets, as their effect is short-lived. Acidifying diets with excessive protein content should be avoided. Potassium citrate can be added in patients with persistently acidic urine. Thiazide diuretics can be considered in patients with a high recurrence rate of these calculi.

Prevention of urate uroliths consists of decreasing urine concentration, limiting purine intake and alkalinizing urine pH. In dogs with genetic mutation in the urate transporter, allopurinol therapy can decrease the high rate of urate recurrence.

Prevention strategies for cystine uroliths consist of decreasing USG, limiting animal protein and sodium intake, increasing urine pH > 7.5, and neutering.

For more information on this topic or Angell-West's Internal Medicine Service, please contact **Dr. Vrabelova Ackley** at **dvrabelova@angell.org** or **781-902-8400**.

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Feline Megacolon and Deobstipation

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Obstipation is the term applied when constipation (incomplete defecating) is so severe that the stool can no longer be passed. Stool then accumulates in the colon, becoming more and more voluminous as the patient eats, and getting harder and harder as the large bowel absorbs more and more water from the mass of stool. Feline idiopathic megacolon is the most common cause of obstipation in the cat. Some other causes of obstipation include pelvic deformity from trauma, neoplasia, strictures, foreign bodies, and cauda equina syndrome. Feline megacolon is most commonly seen in middle-aged male cats, and any breed can be affected. It is the result of dysfunction of the colonic smooth muscle. Most obstipated cats strain to defecate and produce little or no stool. If obstipation is severe, it can result in vomiting, and even diarrhea due the irritation that the hard stool causes against the colonic wall. This is unfortunately a slowly progressive disease, and the ability to defecate usually worsens over months and years.[1]

MEDICAL MANAGEMENT

In the development of feline megacolon, medical and dietary management can be successful in delaying the need for deobstipation. The colon still maintains some ability to contract and expel stool, but defecation may be infrequent or incomplete (constipation). If management efforts lapse, obstipation may result.

Dietary management is often a first line choice to manage developing feline megacolon. Higher fiber diets can be useful at this stage of management. Soluble fiber is preferable to insoluble fiber foods, since insoluble fiber tends to be bulk forming and further distends the colon. Soluble fiber is highly fermentable, attracts water into the stool, and aids in gel formation within the colon. The soluble versus insoluble fiber fraction usually does not appear on pet food labels; listed instead is crude fiber, which is a measure of both soluble and insoluble fiber.[2] As colon function declines and obstipation develops, low-residue diets can help to decrease stool volume.

Prokinetic medications may be successful in early management. Cisapride is often the prokinetic of choice. It has been shown to improve smooth muscle cell contraction in vitro, but the in vivo benefits of this medication for feline idiopathic megacolon are unproven. Nonetheless, it remains a rational and anecdotally supported medication to use. The starting dose is 2.5 mg BID, but can be titrated upwards as needed to 5-7.5 mg TID. Cisapride is no longer available for human use due to its arrhythmogenicity, but it can be procured from compounding pharmacies for veterinary use. Other prokinetics that may be considered include ranitidine, misoprostol, and metoclopramide, but they are likely not as effective as cisapride.[1]

Stool softeners are also a common first line choice for any cat that is having difficulty defecating. Lactulose (0.5 ml/kg BID-TID)3 has historically been the laxative of choice. The dose of this medication may be adjusted by the owner to achieve a desired stool consistency. It is an indigestible sugar that osmotically pulls water in to the bowel. It can sometimes be difficult to give to cats due to the flavor and mouth feel, limiting client compliance. MiraLAX™ (polyethylene glycol 3350) may be replacing lactulose as the stool softener of choice for cats with megacolon and obstipation. It has virtually no flavor and can be easily sprinkled on or mixed with wet food. The starting dose is 1/8 to 1/4 teaspoon BID, and this dose is titrated upwards depending on desired stool consistency. Polyethylene glycol (PEG) 3350 is not absorbed from the intestinal tract and binds to as many as 100 water molecules, resulting in a softer stool. It was shown to mildly increase potassium levels and may result in some dehydration if a patient does not drink adequately. High doses can result in diarrhea—like any laxative.[4]

Almost invariably, medical management will eventually fail as the colon loses more and more contractibility. If the stool becomes too hard and too wide to pass through the pelvic outlet (see Figure 1), some method of deobstipation may be necessary.

FIGURE 1

↘ Example of pelvic outlet obstruction.

EMERGENCY

Continued from page 8

ENEMAS

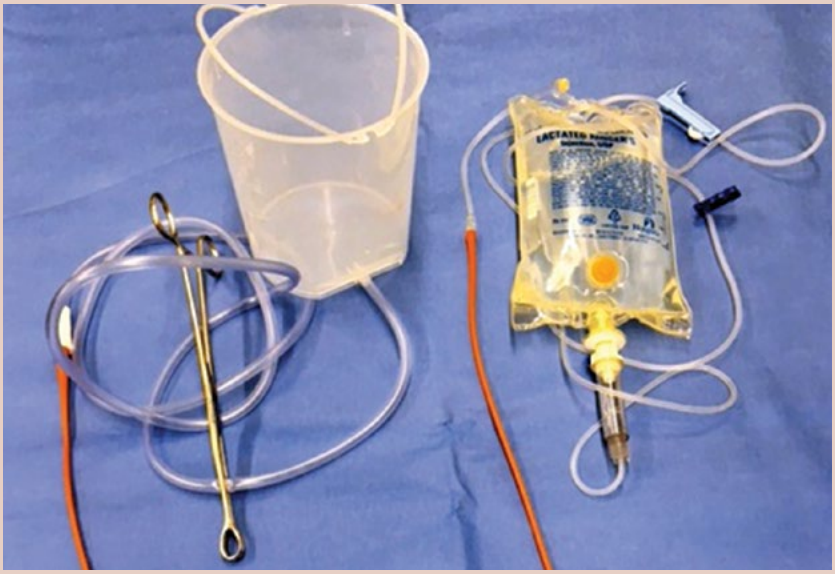
Enemas can be successful if the stool is not yet too firm or too voluminous. Enemas of about 60 ml per cat can be used and may need to be repeated every 8 hours. Enema solutions of warm water, saline, or water mixed with a water-based lubricant or lactulose have been used. I find the mixture of 50 ml of warm water with 5-10 ml of water-based lubricant well tolerated and much easier to clean up than an enema containing lactulose or other lubricants.

MANUAL DEOBSTIPATION

If the stool volume is simply too much to mobilize with enemas, then manual deobstipation is an option. The patient should be anesthetized and intubated. A pre-procedure radiograph of the abdomen to show stool volume and pelvic conformation may be helpful but is not necessary. Pelvic narrowing can usually be detected by rectal palpation of the pelvis. It is helpful to have a 1 liter bucket of warm water hung above the patient, which is connected to an 8 or 12 French red rubber catheter via a segment of suction tubing (see Figure 2), or a bag of warmed IV fluid with tubing can be used. The tube can then be clamped off using a Kelly hemostat or Carmalt to control flow. Using a gloved finger covered with ample water-based lubricant, small pieces of the stool can be chiseled off the distal end of the stool mass per rectum. Those pieces of stool need to be small enough so that they can be removed via the rectum, often pressed between the gloved finger and the pelvic brim. Using forceps of any sort for this procedure should generally be avoided due to the risk of colonic damage and perforation. Once the stool mass is no longer reachable per rectum, the red rubber catheter (covered in water-based lubricant) should be inserted rectally; about 50 ml of warm water can be infused. It helps to insert the red rubber catheter quite deeply so the tip is positioned oral to the stool mass. As the water then flows out of the rectum, the stool mass is brought aborally. Some gentle pressure on the cranial abdomen pushing the stool mass caudally is helpful to bring the stool back into reach. This process is repeated about 10-20 times until no more stool is palpable in the abdomen. Once all the stool is successfully removed, the water that is flushed into the rectum will come out clear without any feces dissolved in it. This is generally a reliable indicator that no more stool remains. A post-procedure lateral radiograph may be helpful to confirm complete removal of the stool if this cannot be done by palpation. It is important to remember that anytime a gloved finger or red rubber catheter is inserted into the rectum that it be covered in water-based lubricant to prevent abrasion and trauma to the rectal mucosa.

FIGURE 2

↘ Equipment used for manual deobstipation.



MEDICAL DEOBSTIPATION

Medical deobstipation using a continuous infusion of polyethylene glycol (PEG) 3350 (Colyte™ or Golytely™) solution via a nasogastric tube has largely supplanted the need for manual deobstipation. Even cats with very large volumes of stool can be successfully evacuated using this protocol with a 12-24 hour infusion. A nasogastric or nasoesophageal tube should be placed. Light sedation or topical analgesia is sometimes needed for tube placement. A post-placement radiograph should be taken to confirm appropriate placement and location of the tube. Via the nasogastric tube, a solution of PEG 3350 diluted according to the bottle instructions is infused at 6-10 ml/kg/hr. This dilution of the PEG 3350 does not result in fluid shifts either into or out of the cat, so neither overhydration, dehydration, nor significant electrolyte derangements are expected. In a small pilot study of 9 constipated cats by Dr. Anthony Carr, the median time to significant defecation was 8 hours (range 5 to 24 hours), and the median total dose given was 80 ml/kg (range 40-156 ml/kg).[6] It is admittedly hard to imagine that this method can successfully evacuate such large volumes of stool like in Figure 1, but we have been pleasantly surprised with both the effectiveness and rapidity of this method. It seems well tolerated by the patient and avoids general anesthesia.

For more information, please contact Angell's Emergency Service at **617-522-7282** or **emergency@angell.org**.

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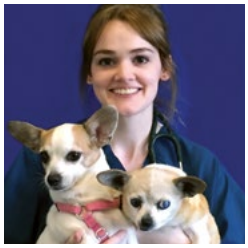

Seizures vs. Syncope

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A seizure is the most common clinical complaint involving the nervous system that most veterinary professionals encounter. However, the term "seizure" is often used by clients to describe other types of neurologic episodes and even non-neurologic paroxysmal events. Therefore, veterinary professionals must ascertain whether a given episode is truly a seizure before proceeding with further diagnostics and treatment.

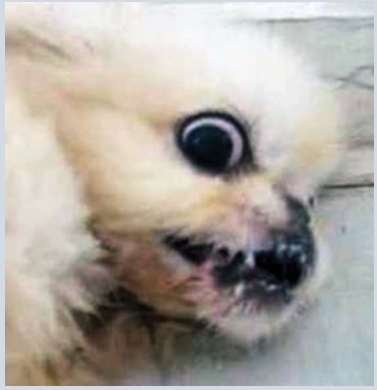
A seizure is the clinical manifestation of intermittent, spontaneous, abnormal neuronal activity in the cerebral cortex. This clinical manifestation is highly variable; in theory, any unusual, involuntary phenomenon that is episodic and recurrent may represent an underlying seizure disorder. A generalized seizure (previously referred to as a "grand mal" seizure), according to the International League Against Epilepsy, includes bilateral symmetric tonic-clonic contractions of muscles, usually associated with autonomic phenomena (e.g. pupillary dilation, salivation, urination, defecation) and loss of consciousness. Afterward, during the post-ictal period, abnormalities such as disorientation, pacing, apparent blindness, and/or excessive eating/drinking often occur. A partial seizure involves focal or asymmetric motor activity affecting any part of the body. Common examples of partial/focal seizures are facial twitching and fly-biting behavior. For veterinarians and technicians, it is important to realize that both types of seizures may easily be confused with syncope, vestibular disease, movement disorders, obsessive/compulsive behavior, and even narcolepsy/cataplexy and sleep disorders.

As with a generalized seizure, a syncopal event is episodic and will begin and end abruptly. A syncopal animal will experience sudden loss of consciousness and muscle tone, secondary to a transient cerebral deficiency of oxygen. As with seizures, vocalizing and involuntary urination/defecation may occur. However, spontaneous recovery will occur in seconds-minutes (unless the event is fatal), and post-ictal disturbances should not occur, aside from possibly a brief period of confusion. Syncope may be caused by underlying cardiopulmonary dysfunction (e.g. arrhythmia, pulmonary hypertension, congestive heart failure). It can also occur due to non-cardiac causes; tussive syncope ("cough drop") can occur when coughing increases intrathoracic pressure and diminishes venous return to the heart, thereby lowering cardiac output. Vasovagal syncope can be seen after straining to defecate or after experiencing sudden fright or excitement.

Vestibular disease, especially in acute cases, is sometimes mistaken for seizure activity by clients. Vestibular disease is a category of disorders affecting the peripheral (inner ear, cranial nerve VIII) and central (brainstem, cerebellum) vestibular systems. The most common clinical signs are head tilt, nystagmus, and loss of balance (e.g. ataxia, circling, and leaning/falling in one direction). Vomiting, recumbency, and "gator-rolling" may be seen in more severely affected patients. In most cases, vestibular disease should be identifiable by veterinary professionals based on clinical signs alone.

Muscle tremors, or myoclonus, may also be difficult to differentiate from seizure activity. There are many breed-specific diseases that can cause muscle tremors. Muscle tremors may also be seen in patients with toxicities (e.g. pyrethrins, tremorgenic mycotoxins, organophosphates, numerous illicit drugs), metabolic derangements (e.g. hypocalcemia, hypoglycemia, hepatic/uremic encephalopathy, hyperthyroidism in cats), and infection (e.g. canine distemper virus). Practicing veterinarians will occasionally see an English bulldog or Doberman pinscher for idiopathic head tremors or head-bobbing. These tremors may be mistaken for focal seizures and manifest as repetitive, episodic movements of the head (in either a horizontal or vertical direction), during which mentation and gait remain normal. In general, loss of consciousness and post-ictal signs are not seen with movement disorders.

When obtaining a history from an owner reporting seizure activity in his/her pet, it is important to take note of the usual details (e.g. signalment, diet, and vaccine status). It is also important to consider the following:



1. The circumstances/setting. When did the activity occur with respect to the pet's last meal? Was the pet sleeping or exercising immediately prior? Was there any observed odd behavior, coughing, vomiting, or straining to defecate before the episode?
2. What was the pet physically doing during the episode? Did it seem to be aware or conscious of its surroundings?
3. How long did the episode last? Did the pet recover quickly, or was there a subsequent period of disorientation?
4. Has the pet had any similar episodes previously? Has it had any recent confusion, weakness, or changes in behavior?

A thorough history is sometimes enough to determine if a seizure has occurred. However, physical examination is also important when evaluating a pet at your clinic for possible seizure activity:

1. Obtain a full set of vital parameters, including blood pressure. The temperature may be increased following prolonged seizure activity or with sustained muscle tremors. Active cooling may be indicated for hyperthermia.

Continued from page 10

2. Are there any cardiac abnormalities (arrhythmia, murmur) identified on auscultation? In a pet with obvious cardiac disease, syncope may be more likely than seizure. An arrhythmia may need classification (via ECG) and immediate treatment.

3. How is the pet breathing? What is its mucus membrane color? Cyanosis and increased respiratory rate/effort may be present in syncopal patients with underlying cardiopulmonary disease. However, post-ictal patients may also present with tachypnea or dyspnea due to neurogenic/non-cardiogenic pulmonary edema. Supplemental oxygen should be provided as needed. Thoracic x-rays may be useful but should only be performed if the patient is stable enough to be imaged.

4. Is there any external evidence of trauma? Head trauma is a potential cause of seizures. Also, animals may inadvertently injure themselves during seizures, syncopal events, and vestibular episodes.

5. Bloodwork may be useful in determining the underlying cause(s) of seizure. However, regardless of the underlying cause, point-of-care bloodwork may be used to evaluate for metabolic effects of seizure activity, such as hypoglycemia, hyperlactatemia, acidosis, coagulopathy, etc. This is especially important in cases of prolonged seizure activity.

Ultimately, the most thorough history and physical examination may not yield a definitive diagnosis. The episode—whether it is a seizure or not—may never be witnessed directly by veterinary staff. If the pet is stable and does not need immediate care, instruct your client to obtain a video recording of any future events (this is relatively easy today with the aid of an iPhone or other smart phone).

For more information, please contact Angell's Emergency/Critical Care Service at **617-522-7282** or **emergency@angell.org**.

↘ PHYSICAL THERAPY NOW AVAILABLE AT MSPCA-ANGELL WEST

The MSPCA-Angell is pleased to announce the addition of Physical Therapy (PT) to our Waltham specialty services. In July of 2017, Angell acquired FlowDog, a canine aquatic fitness & rehabilitation center. This acquisition is instrumental to the comprehensive, personalized care Angell strives to provide to each and every patient.

The facility will continue to operate during its regular business hours at 96 Clematis Ave.in Waltham until February 2018, when the official location will shift to the MSPCA-Angell West building at 293 Second Avenue in Waltham.

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

Current PT services include:

- HYDROTHERAPY
- LAND-BASED EXERCISE
- MANUAL THERAPY
- THERAPEUTIC LASER
- MASSAGE
- CONSULTATION AND FITTING OF ASSISTIVE DEVICES
- UNDERWATER TREADMILL*
- ACUPUNCTURE*

*Available as of February 2018 after relocation from 96 Clematis Ave. to 293 Second Ave. in Waltham. For more information, visit **angell.org/PT**.

↘ ANGELL AT NASHOBA: LOW COST CARE FOR FINANCIALLY QUALIFIED CLIENTS

In 2016, Angell Animal Medical Center and Nashoba Valley Technical High School partnered to create **Angell at Nashoba**, a veterinary clinic for low income pet owners that also serves as a rigorous academic and experiential training program for students enrolled at Nashoba Valley Technical High School.

The clinic provides discounted:

- Spay/neuter services
- Vaccinations
- Primary veterinary care

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.

↘ Medical Director Dr. Laurence Sawyer provides routine care to two Ragdoll cats.

↘ ANGELL'S REFERRING VET PORTAL

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We are pleased to offer the Angell Referring Veterinarian Portal to our referring partners. The Portal provides 24/7, secure, mobile-friendly access to your referred patients' records through angell.org/vetportal. The system automatically updates throughout the day and provides 24/7 access to:

- Online Medical Records
- Check-in Status
- SOAPs
- Diagnostic Images
- Lab Results
- Referral Reports
- Discharge Instructions
- Prescriptions

Settings can be customized within the Portal to receive notices by email or fax, and you may list multiple emails to receive check-in, discharge, deceased, and update notices.

Visit **angell.org/vetportal** or call **Eleanor Cousino** at **617-522-5011** to gain access to your account.

POINT OF CARE COAGULATION TESTING FOR THE VETERINARY TECHNICIAN

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Medical decision making is becoming more and more time-sensitive and laboratory information crucial to patient care can often be needed within minutes. Because of this, many tests have become point of care (POC). Point of care refers to the performance of a diagnostic test at or near the patient. Tests are performed by portable instruments using small amounts of blood. The primary advantage is the decreased turnaround time. Examples of common POC tests used these days are glucometers and lactometers. These handheld devices are affordable, convenient, fast and efficient. Recently new to the market is a hand held POC coagulation analyzer that is gaining favor in many practices that see urgent cases or perform surgery. Other POC coagulation analyzers are also available. Regardless of the device, coagulation testing is becoming an important tool to help diagnose conditions that put our patients at risk for hemorrhage.

The definition of coagulopathy is a condition in which the blood's ability to clot is impaired. The process of forming a blood clot first occurs by primary hemostasis, when the primary platelet plug is formed. Disorders that affect the number of platelets or the function of platelets will disrupt this first step in coagulation. Secondary hemostasis occurs at the same time as primary hemostasis. This is the formation of the fibrin clot which is produced by activation of multiple coagulation factors. Lack of adequate coagulation factors will affect the formation of this fibrin clot. Patients with clotting disorders typically present with signs of unexplained bleeding. These disorders can be congenital as in the case of hemophilia and von Willebrand's disease, but may also be acquired in situations of trauma and anticoagulant rodenticide toxicity. Veterinary patients may be asymptomatic during a routine exam or may present urgently with signs associated with excessive or unexplained spontaneous hemorrhage (cavity bleeding, bleeding into joints or muscles, and "rebleeding" after initial clot formation).

Whether congenital or acquired, the most common bleeding disorders arise from lack of functional platelets or coagulation factors. POC in house diagnostics are quick and easy to perform to help figure out what may be going on. Only a drop of blood is needed to make a blood smear to perform a platelet estimate and a small amount more for coagulation screening tests. In the presence of an adequate platelet count, a prolonged PT and/or aPTT will alert the veterinarian that a specific coagulation factor is missing, or too low. This could be due to a toxin such as anticoagulant rodenticide ingestion or a congenital clotting factor deficiency.

In house coagulation testing is handy for preoperative screening or to help diagnose conditions for at-risk animals such as those that may have ingested a toxin or when a hereditary bleeding disorder is suspected. Additionally, the easy-to-perform diagnostics are good for patients with known specific conditions or abnormalities which allows for more focused therapy and clinical decision making. A number of assays have been validated for use in the dog. Most are simple to use and results are available within minutes.

One such machine, the Abaxis VSPro coagulation analyzer, is a table top analyzer which uses a single cartridge to provide both PT and aPTT results and requires less than 100uL of citrated whole blood to run. Cartridges are stored in the refrigerator and must be warmed to room temperature prior to use.[1] Another analyzer is the Idexx CoagDx. This is a hand-held device that uses separate cartridges for both PT and aPTT. Citrated or fresh whole blood can be used, with specific cartridges available dependent on the type of sample. These cartridges also require refrigeration but can be stored at room temperature for a period of time if they remain unopened. A third analyzer available, the InSight qLabs Veterinary Coagulation Analyzer, is a handheld device. This machine uses test strips, either combined PT/aPTT or PT only. A single drop of non-anticoagulated whole blood (~10uL) is required. These strips do not need to be refrigerated.

There are many benefits to these POC tests. They are easy to perform, minimizing user error. A small amount of blood is required. Test results are available quickly, allowing for faster patient diagnosis, treatment, and management of their disorders.[2] The quality of the sample is also improved as it is tested immediately.

Although these tests do provide many advantages, compared with a reference lab, these machines do not have as regular quality control which could lead to errors amongst testing.[2] Even with systems in place to decrease user error, there is still the potential for variation of results based on the users experience with the machine or in the sample provided.[2] While the cost of running these POC tests may be comparable to a reference lab, there is still the cost of the machine, supplies, and maintenance which is a consideration. There is also not much research available to help with interpretation of results. As the methods these machines use to determine clot formation varies, this can often make comparing results challenging both amongst POC analyzers and with reference lab equipment.[1] POC machines will provide information on hypocoagulation but do not provide information on hypercoagulation or disorders of fibrinolysis.[2]

Common indications for using POC coagulation testing include patients which present with unexplained bleeding, high risk patients which require emergent surgery or procedures such as fine needle aspirates, or patients with possible anti-coagulant rodenticide exposure. It is always important to interpret these results in light of the patient's clinical signs and risks/benefits of therapy.

FOR MORE INFORMATION, PLEASE CONTACT **ANGELL'S EMERGENCY SERVICE** AT 617-522-7282 OR EMERGENCY@ANGELL.ORG.

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ANGELL AT NASHOBA

Laurence Sawyer, DVM
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↘ OUR SERVICE LOCATIONS

BOSTON & WALTHAM

Avian & Exotic Medicine
617-989-1561

Behavior
617-989-1520

Cardiology
617-541-5038

Internal Medicine
617-541-5186

Neurology
617-541-5140

Physical Therapy**
781-209-5720

BOSTON ONLY

Anesthesiology
617-541-5048

Dentistry
617-522-7282

Dermatology
617-524-5733

Diagnostic Imaging*
617-541-5139

Nutrition
617-522-5011

Oncology
617-541-5136

Ophthalmology
617-541-5095

Pain & Palliative Care
617-541-5140

Pathology*
614-541-5014

Surgery*
617-541-5048

(W/B) Services also available at our Waltham location
**Available only in Waltham

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

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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.
Winter 2018 ■ Volume 12:1 ■ angell.org ■ facebook.com/AngellReferringVeterinarians

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 NASHOBA
100 Littleton Road
Westford, MA 01886
978-577-5992
angell.org/nashoba

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

■ angell.org/directions (free parking) ■ angell.org/hours ■ angell.org/ce

↘ ANGELL ANIMAL MEDICAL CENTER IS A FEAR-FREE CERTIFIED FACILITY

As a fear-free certified medical facility, our goal is to prevent and alleviate fear, anxiety, and stress in our patients.

ANGELL IS PROUD TO HAVE FEAR FREE PROFESSIONALS ON OUR TEAM

We've always worked hard to provide the best care to our patients, and are committed to looking after our patients' physical and emotional well-being. Angell's "Kitty Kruiser" carts and feline friendly exam rooms are additional steps we have taken to ease the stress of our patients.

Cat Friendly Practice Gold
Angell has earned gold accreditation by the AAFP.

FEAR FREE CERTIFIED Professional

THE FEAR FREE VETERINARY VISIT

During a typical Fear Free veterinary visit, the practice team members might use the following strategies:

- **Initially, avoid eye contact with pets and focus on clients instead**
- **Use carrier covers and elevated kitty kruisers**
- **Use mobile check in away from the busy front desk**
- **Provide non-slip surfaces for patients to stand/rest on to improve balance and stability**
- **Use gentle pressure to soothe the pet using a towel or compression garment**
- **Create a calming environment with pheromone diffusers**
- **Actively work to reduce stressful noises**
- **Prescribe anti-anxiety or other calming medications & supplements**
- **If a patient is showing excessive signs of fear, anxiety, or stress, the team may delay or postpone the exam/procedure until a time when the pet is calmer and more relaxed**

ANGELL.ORG/FEARFREE