



EMERGENCY AND CRITICAL CARE

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ECC Survival Summary: How to Handle 5 Common Emergencies

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1 Obstipation is the inability to pass stool and it is a permanent condition. Constipation is difficult or infrequent passage of feces. Obstipation is quite uncommon in the dog and is usually a result of eating bones/bone fragments, colonic tumors, or pelvic fractures. Dogs do not get idiopathic megacolon like cats do. Obstipation is a fairly common problem in cats. It usually affects cats in middle to older ages. The most common cause of obstipation in the cat is idiopathic megacolon, but pelvic fractures and colonic tumors affecting the outflow of stool can also be seen with some frequency. The cause of feline idiopathic megacolon

is not entirely understood. Although it is suspected to be a problem of smooth muscle, both the colonic innervation and the myoneural junction have been proposed as the location of the pathology causing this issue.

There has been no identified cure or definitive treatment for feline idiopathic megacolon. Medical management has been reported to be “unrewarding,” which is likely because the problem is typically progressive regardless of treatment and management strategies are minimally effective at improving

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ONCOLOGY

Radiation Therapy Side Effects: Skin

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Radiation therapy is increasingly used in veterinary patients undergoing cancer treatment. In this setting, the most commonly employed radiation therapy technique is external beam radiation. Radiation is delivered using a beam of high-energy x-rays generated outside the patient (typically with a linear accelerator) and aimed at surface or internal targets. High-energy electrons may also be used to treat targets on the surface. With recent advancements in radiation therapy

planning technology, we are now able to aim the radiation beam much more precisely at our target using image-guided, computer-based treatment planning (i.e., intensity-modulated radiation therapy, or IMRT). This has resulted in decreased incidence and severity of skin side effects in many patients; however, in patients with tumors close to or even in the skin, radiation can result in skin changes and side effects.

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colonic motility. Medical management strategies include feeding a high-fiber diet – often canned food to improve water intake, laxatives, occasional enemas, and occasional deobstipation. The most effective laxatives are Lactulose (1-3ml with food 1-2 times daily to achieve desired stool consistency) and MiraLAX (polyethylene glycol), which is often given as 1/8-1/4 of a teaspoon mixed with food twice daily. MiraLAX tends to be very well tolerated by cats, and it is not messy or sticky – both disadvantages of Lactulose. Both of these medications are osmotic laxatives. The only medication that is really useful to improve colonic contractions is Cisapride. Cisapride is no longer available on the human market due to some cardiac arrhythmias that were noted in people. It can still be bought from some compounding pharmacies for veterinary use. It is typically given as 1mg/kg PO TID, but the range is 0.5-1.0mg/kg BID-TID depending on the patient. Erythromycin at 0.5-1.0mg/kg BID-TID (well below the antibiotic dose) and Ranitidine 1-2mg/kg BID have some mild promotility effects on the colon but do not seem to be as effective as Cisapride.

Once medical management fails, a sub-total colectomy can be performed where most of the colon is removed, leaving only a few centimeters of distal colon adjacent to the rectum. The ileocecolic junction is also sometimes removed in this surgery to allow adequate anastomosis of bowel. This procedure is met with very good results, with the vast majority of cats having some diarrhea for 1-12 weeks but then passing normal stool. Occasional constipation is reported, but it is usually easy to relieve by enemas, stool softeners, and digital manipulation of stool. Stricture formation at the anastomosis site is also rarely reported.

Manual deobstipation under anesthesia is often required in animals whose medical management is not adequate to keep the colon free of collecting stool. Once a cat has not defecated for more than 7 days, the stool is typically so hard and the colon so dilated that no amount of stool

softeners or promotility agents will allow passage of the stool with assistance. In fact, these patients effectively have a colonic obstruction and promotility agents and some strong laxatives may be contraindicated due to severe discomfort and the risk of colonic perforation.

Before deobstipating a patient, it is helpful to give a few enemas (40-60ml of warm water and K-Y Jelly) and some SQ fluid overnight. This will help to soften the stool, making the removal process easier – and sometimes to everyone's delight (!) the stool passes on its own after an enema or two.

Patients should be fully anesthetized and intubated in order to effectively deobstipate them. This is due to the discomfort associated with the procedure in awake patients, and the need for aggressive manipulation of the abdomen and the stool in order to break it apart and retrieve it from the rectum. It is helpful to have a water reservoir hanging above the table to use as an enema during the procedure. An enema bag from a pharmacy will work well for this purpose. An IV fluid bag with warm saline may work but will only flow very slowly. A large-diameter (12-14 Fr) red rubber catheter is then connected to the warm water reservoir via some flexible rubber tubing. Have plenty of K-Y Jelly available to coat the red rubber catheter, several towels to wipe stool off your hand, and a large garbage can to drop stool into. Wrapping the cat's tail in VetWrap will help prevent soiling of the tail during the procedure.

DEOBSTIPATION PROCEDURE:

1. Patient's legs pointing toward you.
2. Your dominant hand is your "dirty" hand. The other hand never touches stool.
3. Avoid using forceps for stool retrieval. It's dangerous.
4. Put K-Y on your finger every time before going into the rectum.
5. With your index finger, break apart and retrieve pieces of the most distal stool. Even chipping off small pieces from the edge is helpful. If your finger can be inserted into the middle of the mass of stool, then larger pieces can be broken off.
6. Use the enema set up to instill 60ml of warm water to "flow" the remaining stool toward the rectum. Putting the enema fluid high up into the colon is helpful for this purpose.
7. With your "clean" hand, gently push stool toward the rectum by palpation.
8. Go back to step 5 until the water coming out of the rectum is clear and no more stool is palpable.

9. Milk any residual water out of the colon by gently caudally directed abdominal palpation.
10. A post-procedure radiograph is rarely necessary if the patient is not obese.

This procedure usually takes about 15-30 minutes depending on experience. Once the patient is awake, they rarely pass additional fluid from the rectum if the stool was successfully removed. Patients can be sent home when fully awake, sometimes with an oral antibiotic or an analgesic if the procedure was particularly difficult or blood was noted to be coming out in the rectum during the procedure from mucosal damage. This is generally not necessary, though.

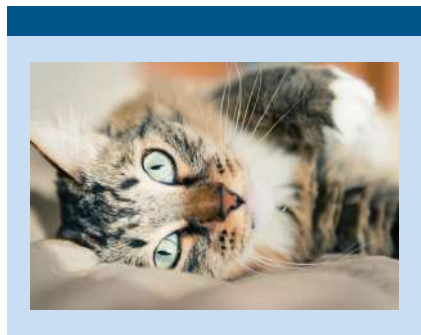
2. THYROID STORM

An acute thyrotoxic crisis is called a thyroid storm in human medicine. It is a life-threatening condition requiring emergency treatment. It is often triggered by severe physical or mental stress in thyrotoxic patients. Patients have multiple organ failure as a result of the breakdown of compensatory mechanisms. It is characterized by some or all of the symptoms of neurologic dysfunction (restlessness, delirium, psychosis, somnolence, or coma), fever, tachycardia, heart failure, GI signs, in addition to an elevation of thyroid hormone(s). Mortality rates range from 10% to 75%.

There is some debate about the use of the term thyroid storm as it is applied to cats. While a thyrotoxic crisis similar to that described above does occur in cats, it is less severe than that seen in humans, and for that reason may (eventually) need a different nomenclature to describe it. But for now the term thyroid storm is what is used.

There are no case reports, case series, or retrospectively published papers that describe the condition of feline thyroid storm in a refereed journal. Much if not all of the information about feline thyroid storm that is published in veterinary texts and a few journals is anecdotal, or taken from the human experience with this syndrome. For that reason, there is no clear definition of feline thyroid storm, nor are there any diagnostic criteria that are outlined for it. The diagnosis is predominantly based on clinical characteristics of thyroid storm that are seen in hyperthyroid patients.

Both in humans and in cats, there tend to be historical characteristics that suggest or portend a thyroid storm. In cats this is most often stopping the anti-thyroid medication (methimazole) several days or weeks prior, usually by accident. Other triggers include infections, or some stressor (psychological or physical). Aggressive thyroid gland palpation, thyroid gland surgery, and treatment with I-131 (disputed by some sources) have also been implicated.



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FIGURE 1

Hyperthyroid cat



The most commonly identified clinical characteristics of thyroid storm in cats are tachypnea (panting), tachycardia, and hyperactivity. Most cats also will have the typical hyperthyroid appearance of a thin face and body, and a poorly groomed hair coat. Less commonly, fever may be noted. A panting cat having the appropriate appearance should immediately create the suspicion of thyroid storm. Most panting cats with thyroid storm have no pulmonary dysfunction and are not truly dyspneic – but they look it! It can be very hard to determine if a thyrotoxic panting cat is truly dyspneic without further testing such as thoracic radiographs, thoracic ultrasound, or echocardiography.

Initial and most important diagnostic steps are a T4 and thoracic radiographs. Treatment is usually initiated in the absence of T4 results unless it can be measured quickly, as is the case with some in-house machines. Thoracic radiographs are usually normal despite the clinical appearance of dyspnea and will help to rule in thyroid storm and rule out causes of dyspnea. Some cats will have concurrent heart failure due to the thyrotoxic heart disease – but that is less common. Ideally, a CBC/chemistry/UA will also be performed to try to identify a triggering cause if none is clearly evident. Full labs can be done the next day when the immediate crisis is under control if additional sample collection is too stressful for the patient.

The goals of treatment are 1) reduce production/secretion of thyroid hormones, 2) counteract the effects of thyroid hormone, 3) provide systemic support, and 4) identify and correct the triggering factor.

The mainstay of therapy is oral methimazole and a β -blocker. Improvement is usually seen within hours of starting this treatment. Methimazole is usually given at 5mg/cat BID, but in small cats or if renal dysfunction is present 2.5mg BID can be used. Methimazole will stop the production of new thyroid hormone from the thyroid gland but will not prevent release of already-made thyroid hormone from the gland. In human medicine, the latter source of thyroid hormone is routinely curtailed by giving iodine compounds an hour after methimazole is given. This has also been advocated in cats but is rarely necessary given the rapid response to methimazole and a β -blocker. Iodine can be given as iopanoic acid and diatrizoate meglumine (both iodinated radiographic contrast media) at 100mg PO BID for this purpose. IV administration is also possible but needs to be done with care since they are hyperosmolar compounds. The β -blocker helps control the accelerated heart rate and calm the patient. A β -blocker is contraindicated, though, if the patient is in heart failure. Atenolol is widely available in many hospitals and is usually dosed at 1mg/kg or 6.25mg/cat PO SID-BID in the initial crisis. Propranolol 2.5-5mg/cat PO TID or 0.02mg/kg IV TID is also a reasonable option. The fast-acting Esmolol can be given by IV at 10-200mcg/kg/min. Despite the appearance of dyspnea, oxygen therapy is not needed if there is no pulmonary disease. Additionally, the sound of the oxygen compressor of an oxygen cage can sometimes create extra stress for these edgy cats. IV fluid may be needed in some cases, but that choice should be made based on hydration status and lab work. If a triggering illness is identified, that should also be treated, but that is not necessary to get the crisis under control in the emergent setting.

The prognosis for feline patients with thyroid storm is very good. I have not seen patients die from this problem who have been treated appropriately. Improvement is usually noted within a few hours of oral therapy, and most patients feel quite normal within 24 hours. The grave consequences that are identified in human medicine are not seen with cats in our experience.

3. ESOPHAGOSTOMY TUBE (E-TUBE)

When a patient is unable or unwilling to eat for a prolonged period, a temporary feeding tube is indicated to supply enteral nutrition until the patient heals and can resume eating. A number of tubes and tube locations can be used, and each one comes with pros and cons. We most commonly use naso-esophageal/gastric tubes in our CCU. These tubes are technically easy to place, require little or no sedation, and are

inexpensive. Unfortunately, they can only be managed easily in the hospitalized patient, require a liquid diet that always results in liquid stool, and can only practically be used for <5 days in most cases because of annoyance and irritation to the patient. Gastric tube (surgically placed or percutaneously placed endogastric tubes – PEG tube) are very durable and well tolerated, and a standard canned diet can be fed. But gastric tubes require anesthesia and a certain degree of expertise to place. They also create some risk of peritonitis if the tube is dislodged, becomes infected, or is removed before a stoma forms (7-10 days). Jejunostomy tubes are very specialized tubes that bypass the esophagus and stomach, which is ideal for upper-GI problems of the esophagus/stomach/pancreas, but they are quite challenging to place and to maintain and they do require a liquid diet. Although I could not say that an esophagostomy tube is the “best” tube since it depends on the patient and the problem, esophagostomy tubes do have some of the positive characteristics of the other tube types that make them fairly practical in many situations.

Esophagostomy tubes are useful for inappetence or structural disease of the oropharyngeal region (tumors/trauma/etc.) that limits eating. An E-tube requires no specialized equipment to place, and placement is technically straightforward. Although anesthesia and endotracheal intubation are required for placement, the procedure is fairly quick. E-tubes can remain in place for weeks or even months, are easy for owners to maintain, and are very well tolerated by most patients. There is no risk to the patient if the tube is immediately removed after placement. Any standard canned diet can be fed through the E-tube when mixed with water and blended. Liquid medications and water can also be easily given through the tube, making complicated at-home medication schemes much easier for an owner than giving all those medications directly orally. E-tubes should not be used for esophageal diseases like esophagitis, megaesophagus, or esophageal trauma. A larger recent study identified that about 35% of patients who had E-tubes placed experienced some sort of complication. Complications were most commonly premature dislodgement, followed by infection at the tube insertion site. Tubes were in place for a mean of 11 days (range 1-94 days).

Esophagostomy tubes can be placed in cats or dogs. They are even used in a number of exotic species (turtles, lizards, hedgehogs, etc.). The most common type of E-tube is a simple red rubber feeding tube – usually a 12-18 gauge. Many more specialized E-tubes are available from different manufacturers, though. Larger-

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FIGURE 2

Placement of E-tube



bore tubes are less likely to be vomited up than thinner, more flexible, smaller-diameter tubes. Larger tubes are also less apt to become clogged.

PLACEMENT TECHNIQUE:

1. The patient should be anesthetized and intubated, and placed in right lateral recumbency. The esophagus runs slightly to the left of midline at this level of the neck, making a left-sided placement a bit easier – but not essential.
2. The desired tube should be measured and marked from the likely insertion site to about the level of the heart (7th rib or so).
3. The left rostral cervical area just caudal to the angle of the jaw is clipped and surgically prepped.
4. A curved mosquito hemostat or Kelly clamp is inserted into the mouth and the point of the instrument is used to push the left wall of the esophagus laterally so the hemostat tip can be seen causing a bump when looking at the left neck of the patient. This bump should be dorsal to the jugular vein, which is readily visible in most cats and some dogs.
5. A scalpel blade is used to cut down onto the tip of the instrument. The incision in the skin should be just large enough to get the tip of the hemostat out of the skin.
6. Once the tip of the hemostat is protruding through the left side of the neck, the mouth of the instrument is opened and closed a few times to widen the hole. The hole should be made wide enough so that the tube can fit through the hole when it is grasped using the hemostat.
7. The hemostat's mouth is opened and the tip of the tube is grasped firmly with the hemostat. The tube is then drawn retrograde into the esophagus of the patient and pulled out of the mouth while still holding it fast within the mouth of the hemostat. About 2/3-3/4 of the length of the tube should be drawn out of the mouth.
8. The tip of the tube is then redirected down the esophagus and the finger is used to push

the bend in the tube down the esophagus as far as possible. Ideally, about 1/4-1/2 of the tube would be redirected normograde back down the esophagus. (The flanged end of the tube is still protruding from the insert site and is facing toward the back end of the patient.)

9. Gentle traction is then put on the flanged end of the tube and about 1-2" should be drawn out of the insert site. If enough of the tube is directed down the esophagus, this light traction will serve to straighten out the tube and eliminate the 180-degree bend of the tube in the rostral esophagus. It is easy to tell when the bend in the tube is gone and the tube is fully directed normograde since the flanged end of the tube will immediately "flip" toward the front of the patient, and the tip of the tube will be in the mid-esophagus.
10. The tube is then inserted into the esophagus to the pre-measured depth.
11. A finger trap pattern using a nylon suture is used to secure the tube in place. The purse string suture encircling the tube should not be tied too tightly or the pressure will result in necrosis.
12. A lateral radiograph of the tube should be taken to ensure that the tube is in the right location and inserted to the right depth. The tip of the tube should not go into the stomach. It should end roughly at the level of the heart.
13. The portion of the tube that is outside the esophagus can be wrapped in a light bandage, or a manufactured wrap can be bought from one of several manufacturers to protect the tube and keep it from dangling or getting in the way. These premade bandages are definitely worth stocking if you use this type of tube.
14. The open end of the tube should be capped with a catheter cap, 3-way stop cock, or a more specialized E-tube plug with a syringe port. Some tubes come with a plug incorporated on the end of the tube.

4. CHEST WOUND MANAGEMENT

Chest wounds most commonly occur from bite wounds and make up about 30% of all chest injuries. In a small retrospective paper evaluating 22 cats that were bitten in the chest by dogs, 11/18 cats had radiographic evidence of pneumothorax, 8/20 cats underwent exploratory thoracic surgery, and there was an overall mortality rate of 27%. The conclusions of this paper suggested that "thoracic exploratory surgery may be necessary, particularly in the presence of pseudo-flail chest, pneumothorax or >3 radiographic lesions."

The dogma of thoracic (and abdominal) penetrating wounds is that if penetration into the cavity has occurred, thoracic exploratory surgery is indicated. Our experience has been that radiographs can sometimes predict when surgery is indicated but can never give adequate information to decide that thoracic exploratory surgery is not indicated. A good digital exam under sedation or anesthesia is usually needed to help determine if seemingly superficial wounds may actually enter the thoracic cavity. Due to the crushing trauma that happens with bite wounds, a relatively mild superficial wound may belie the severity of the wounds to the deeper tissue layers, or even intrathoracically. Although other causes of penetrating thoracic trauma do occur (cuts or punctures from sharp objects), they tend to be less severe than bite wounds due to the lack of crushing trauma. The information gained from bite wound trauma does largely translate to other types of thoracic trauma.

If it is clear that thoracic penetration has occurred, but the patient is not dyspneic, some method of keeping the chest wound closed to prevent development of a pneumothorax should be taken before exploratory surgery can be pursued. This may mean simply applying a water-based jelly to the wound and bandaging the affected area to exert a bit of pressure on the damaged tissues. Sometimes, rough coaptation of skin and muscle with a staple gun can be a very efficient way to temporarily close a large defect before thoracic surgery or referral can be achieved. If it is clear that a clinically significant pneumothorax has developed, then a temporary chest tube should be placed prior to temporarily closing the defect. A simple red rubber tube of 8-14 Fr works well for this purpose and can be simply laid through the thoracic defect into the thorax prior to temporary closure. Once the tissues are opposed, any air in the chest should be suctioned out to re-expand the lungs. Frequent reaspiration of air from the thorax should be attempted since a temporary closure is likely not

FIGURE 3

Chest wound from stick. (Photo courtesy of Dr. Roxanna Khorzad)



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fully airtight. Remember that the goal is to improve the patient's ability to breathe and make them stable for anesthesia or transport. The goal is not to immediately fix the problem fully – that can be done in a more controlled setting later.

Thoracic impalement with sticks or other sharp objects is not common, but this situation does require some non-intuitive decision making in the initial stabilization phase. Most impaled patients are relatively stable. This likely is because those that are most severely compromised do not survive transport to a veterinarian. For sure, the knee-jerk response is to immediately remove the impaling object. This temptation should be resisted. Patient comfort to prevent struggling and facilitate handling or even immediate anesthesia are the most important first steps. Although not ubiquitously available, a CT scan of the affected area is the most effective way to identify the extent of damage and the location of the object prior to surgery. Thoracic or abdominal radiographs will only give a partial understanding of what is happening but are still worthwhile to pursue if CT is not immediately available. Once the patient is considered safe to handle and there is some understanding of what structures are involved with the impaling object, he/she should be taken to surgery. This is truly an exploratory surgery – and the surgeon needs to be ready for blood loss, ruptured viscera, and contamination. Positive pressure ventilation during anesthesia is likely necessary if thoracic penetration has occurred.

5. ABDOMINOCENTESIS

Abdominocentesis is a very simple and safe diagnostic test that yields a huge amount of information about our patients. The near-ubiquitous use of abdominal ultrasound has really made abdominocentesis so much easier and more available than it was even 10 years ago.

The first step is to identify or suspect the presence of free abdominal fluid. This can be done by palpating a fluid wave on physical exam, or by identifying fluid on a radiograph or abdominal ultrasound. Although any patient position may be suitable for sampling of abdominal fluid if enough is present (standing, sternal, or lateral recumbency), often left lateral recumbency is preferred since it allows for easy and comfortable restraint of the patient, and this position encourages the spleen to sink down and a bit out of the way of collecting a sample. If no ultrasound unit is available to identify a large and available collection of fluid, then a location near the umbilicus just to the left (below) of midline is selected if the patient is in left lateral recumbency. A small square of hair 1"-2" wide should be clipped and prepped. Mild sedation can be helpful in some patients who will not settle down, but a local lidocaine block is rarely needed since the injection of lidocaine is usually more uncomfortable than using a sharp needle to collect fluid. The sample can be collected using a 21g needle, a 3cc syringe with needle attached, or a fenestrated catheter if a therapeutic tap is being performed. The needle should be gently inserted perpendicular to the skin through the skin/fat/muscle and into the abdomen. Avoid inserting the needle too quickly, as this can result in puncturing abdominal organs if they are met by the needle. By inserting the needle slowly, the needle will only be inserted as deep as is necessary to collect fluid, and if internal organs are contacted, they will hopefully be pushed out of the way rather than punctured or lacerated. If just a needle and no syringe is used, once fluid starts to flow into the hub of the needle it can be sucked out of the hub by a syringe. This technique allows for greater control of the needle in placement and in cases where it needs to be redirected. For larger-volume effusions, a 21g needle on a 3ml or 6ml syringe is usually fine. If a therapeutic abdominocentesis is being performed, then using a fenestrated catheter makes the procedure a bit safer, especially if the patient is apt to move a bit during the procedure.

A sample of fluid should be put into a red and a purple top tube. This will allow for culture and biochemical analysis from the red top tube, and cytology and TP analysis from the purple top tube. K2EDTA, the anticoagulant in the purple top tube, does have some antibacterial activity, which makes culturing fluid from that tube problematic. A fair amount of evaluation and testing of the abdominal fluid can be done in-house – especially in the emergency situation, even before sending the sample to the lab for analysis by a pathologist.

SEPTIC EFFUSIONS

- Intracellular bacteria noted on a direct smear or spun sample (no further testing is needed)
- >13,000 nucleated cells
- Effusion glucose >20mg/dl less than blood glucose
- Effusion lactate >2.0 mmol/dl greater than blood lactate

UROABDOMEN

1. Effusion creat/serum creat >2:1 (100% specific, 86% sensitive)
2. Effusion K+/serum K+ >1.4:1 (100% specific, 100% sensitive)
3. Effusion creat >4x normal serum creat
(Very high likelihood of uroabdomen if 2 of 3 above criteria are fulfilled)

BILE PERITONITIS

- Effusion bili:serum bili >2:1

FIP

- Effusion total protein >4.5 g/dL (usually)
- Effusion alb:glob ratio <0.9
- FCoV antibodies >1:1,600
- Effusion gamma-glob concentration >1.0 g/dL
- FCoV PCR of effusion

CHYLE

- Effusion triglyceride > serum triglyceride
- Effusion triglyceride > effusion cholesterol
- Effusion triglyceride >100 mg/dL

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OVERVIEW OF RADIATION SIDE EFFECTS

There are two major categories of radiation side effects: acute effects and late effects.

Acute radiation side effects typically occur during or within a few weeks after treatment, due to death of rapidly dividing cells and concurrent inflammation. These side effects are transient and begin to heal within 2-3 weeks of RT completion. Complete healing may take up to 1-3 months in some cases. Acute radiation side effects are more likely to occur with full-course radiation (i.e., 10-20 daily treatments).¹

Late radiation side effects occur months or years after treatment as a result of fibrosis and vascular changes. These complications are often permanent and in some cases may be severe. Risk of late radiation side effects increases with larger doses per treatment, as in hypofractionated (palliative-intent) or stereotactic protocols.¹

ACUTE RADIATION DERMATITIS: PATHOPHYSIOLOGY

Within several hours of radiation treatment, temporary hyperemia and erythema may occur due to local capillary dilation. This is not typically accompanied by any discomfort.²

Under normal conditions, the stem cells in the basal layer of the epidermis continually divide to replace skin cells as they mature and eventually exfoliate. This process is interrupted by radiation-induced death of these rapidly dividing basal keratinocytes, resulting in breakdown of the skin barrier. About 2-4 weeks into treatment, this can manifest as dry desquamation (nonpainful flaking of upper skin layers) progressing to moist desquamation (moist dermatitis with exudate). Moist desquamation is generally painful, and patients are left susceptible to secondary infection of affected areas. Activation of local inflammatory pathways and mast cell degranulation further contribute to inflammation and discomfort.³

Hair follicles and adnexal glands are also radiosensitive at fairly low doses of radiation. Most patients receiving radiation to targets at or near the skin will experience hair loss, which may be temporary or permanent.

Considerable variability is observed between patients in regards to incidence and severity of radiation dermatitis. In humans, established risk factors such as skin tone, smoking, and certain comorbidities or genetic factors are useful to identify patients who may experience more florid acute dermatitis. No prognostic factors

FIGURE 1



A patient with moist desquamation following full course radiation therapy (19 treatments). Photos taken at 7, 13, 25, and 42 days following radiation therapy.

have been identified in veterinary patients. However, there are certain anatomic locations recognized as being at increased risk for severe dermatitis. This includes regions with skin folds (e.g., facial folds in brachycephalic breeds), footpads, periocular skin, and the perineum.⁴

ACUTE RADIATION DERMATITIS: TREATMENT

There is no agreed-upon standard of care for treatment of radiation dermatitis. A meta-analysis of 20 clinical trials using various topical treatments in human radiation patients failed to find evidence that any agent provided effective treatment or prevention of radiation dermatitis. Topical steroids are not recommended in human patients, as they can contribute to skin thinning and increase the risk of infection.⁵

Very few studies have addressed the management of radiation dermatitis in veterinary patients. A survey of veterinary radiation facilities found wide variability in management strategies between institutions.⁶ A placebo-controlled clinical trial evaluating the use of oral steroids (prednisone) in dogs undergoing RT showed no difference in the timing, incidence, or severity between dogs treated with steroids versus placebo.⁷ Another prospective trial evaluated prophylactic

antibiotic treatment in canine radiation patients. This study showed no improvement in incidence or severity of dermatitis, and also found an increase in multidrug-resistant infections in dogs treated with prophylactic antibiotics. The authors therefore concluded that prophylactic antibiotic therapy is not recommended in canine radiation patients.⁸

With little evidence-based medicine to guide treatment or prevention of acute radiation dermatitis, each institution typically develops its own protocols. Here at Angell, our approach is adapted to the severity and location of dermatitis in each individual patient:

- In patients experiencing mild dermatitis limited to hyperemia and dry desquamation, no therapy is recommended. Patients experiencing pruritus are often prescribed diphenhydramine.
- E-collars are recommended at the first sign of dermatitis to prevent self-trauma. Self-mutilation of radiation sites can result in devastating complications due to delayed healing and secondary infection.
- In patients with exudative/moist dermatitis, oral pain medications such as NSAIDs, gabapentin, and amantadine are proactively prescribed. Parenteral analgesia may be used in patients with severe discomfort, including fentanyl patch placement and local nerve blocks in amenable locations.

FIGURE 2



A patient with severe self-trauma after near-complete healing of radiation site. Photos taken at 42, 56, 118, and 138 days following radiation therapy.

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- Sites are shaved and cleaned at the first sign of moist dermatitis and SSD ointment applied daily during anesthesia to non-periocular/facial sites. In patients amenable to it, owners are instructed to apply SSD ointment at home 1-3 times daily.
- Patients with periocular or facial dermatitis are prescribed triple antibiotic or erythromycin ophthalmic ointment for daily application.
- Systemic antibiotics such as cefpodoxime or Clavamox are prescribed in patients with moderate to severe dermatitis to prevent or address secondary infection.
- Skin cytology may be performed in patients where secondary bacterial or fungal infection is suspected.
- Wound dressing is NOT typically recommended, as radiation dermatitis is often highly exudative.
- Avoiding sun exposure is recommended.

LATE RADIATION SIDE EFFECTS

In most patients, chronic changes following radiation are cosmetic only and do not result in discomfort. Most patients undergoing radiation to sites at or near the skin will have some degree of alopecia, which may be permanent. Underlying skin may remain thin and pigmentation changes are common (both hypo- and hyperpigmentation). Hair that does regrow is often white or grey (leukotrichia).

Chronic skin changes are poorly described in veterinary patients but can include fibrosis, ulceration, and lymphedema (including edema of the distal limb in patients undergoing circumferential/full thickness irradiation of the limb). The inflammatory cytokine transforming growth factor beta (TGF- β) is primarily implicated in late radiation side effects. Along with other inflammatory cytokines, increased expression of TGF- β results in fibrosis, endothelial damage, and – in severe cases – skin atrophy and necrosis.⁹

Oral pentoxifylline has shown promise in the treatment of radiation-induced fibrosis in humans, although prolonged treatment (years long) is often needed to elicit clinical effects.⁹ Pentoxifylline is generally well-tolerated in dogs and cats, with nausea and hyporexia being the most commonly reported side effects.¹⁰ Prophylactic use of pentoxifylline is not supported in the human literature.

In patients with chronic wound formation, surgical correction or even amputation may ultimately be required for wound management.

CONCLUSIONS

Acute radiation dermatitis varies between patients, and there is no agreed-upon standard of care in either veterinary or human patients. Clinically relevant chronic skin side effects are uncommon following radiation in veterinary patients.

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Anemia of Chronic Kidney Disease in Cats

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Chronic kidney disease (CKD) is one of the most common diseases of older cats, affecting 15-30% of cats over 12 years of age.¹ Kidney disease is also a major cause of mortality in cats, accounting for 13.6% of all deaths in cats 5 years or older in one study.² Although many cats with chronic kidney disease can remain stable and minimally symptomatic for long periods of time, several variables have been found to be associated with faster progression of disease, including hyperphosphatemia, proteinuria, and anemia. In fact, for cats with stage 2 CKD, a 1% increase in packed cell volume (PCV) was found to correlate with a 10% reduction in the risk of disease progression.³ Anemia is also associated with shortened survival times in patients with CKD. While the overall survival from time of diagnosis for cats with CKD was just over 2 years in one study, cats with CKD that developed anemia were found to have a median survival time of just over 3 months from the onset of anemia.⁴ Since 30-65% of CKD cats will develop anemia at some point in the course of their disease,¹ addressing anemia in these patients is an important consideration that can improve their length and quality of life.

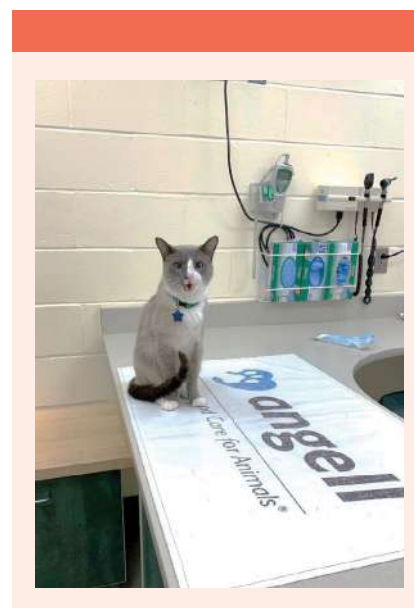
There are a number of factors that may contribute to the development of anemia in CKD patients, including an inflammatory state resulting in iron sequestration, mucosal bleeding, decreased red blood cell survival due to uremia, medication effects, and poor nutritional status. However, the primary cause of anemia in CKD patients is loss of erythropoietin-producing cells in the kidney. Normally, specialized cells in the kidney produce erythropoietin (EPO) in response to renal hypoxia, which then serves as the main stimulus for new red blood cell production in the bone marrow. However, in CKD these cells die off, leading to reduced EPO production and, in turn, reduced stimulation of erythropoiesis and subsequent non-regenerative anemia.¹ Reduced EPO production has been confirmed in anemic CKD cats, with these cats having significantly lower EPO levels than non-anemic CKD cats.⁵

Synthetic forms of EPO have long been used in human patients to stimulate red blood cell production and combat anemia of CKD. Feline

EPO shares approximately 83% homology with the human EPO molecule, which allows synthetic human EPO products to be used effectively in cats to stimulate red blood cell production.¹ However, cats may produce antibodies against synthetic human EPO drugs, which can then cross-react with their own native EPO. This may ultimately lead to pure red cell aplasia (PRCA), the most severe complication associated with use of synthetic forms of EPO. Although PRCA is rare in human patients receiving recombinant EPO products, it was reported in 25-30% of cats receiving epoetin, the earliest available form of recombinant human EPO.¹ Because patients with PRCA are entirely transfusion dependent, often for months, many of these patients will ultimately die or be euthanized due to this complication. The higher likelihood of PRCA associated with epoetin in cats made its use limited to cats with severe anemia (hematocrit <20%), as in those cases the risk of PRCA would be worth the potential gain. Luckily, darbepoetin (Aranesp) has largely supplanted the use of epoetin due to its lower rate of developing PRCA (<10%) and its longer half-life allowing for once-weekly dosing.¹

One study evaluating the use of darbepoetin in anemic dogs and cats with CKD has been published to date. In dogs, use of darbepoetin in anemic CKD patients led to 85% of patients achieving a PCV of $\geq 30\%$, and 67% of patients experiencing a gain of PCV of $\geq 10\%$, with an average time to response of about 4 weeks. A higher starting dose of 0.8 $\mu\text{g}/\text{kg}$ once weekly was associated with a better response than a dose of 0.5 $\mu\text{g}/\text{kg}$, which is closer to the starting dose typically used in people. Although increasing the dosing interval was attempted in most dogs, no dog was able to maintain their PCV at a dosing interval of 3 weeks or more, and many who went to an interval of 2 weeks required going back to a weekly interval to maintain PCV. Adverse events noted in dogs treated with darbepoetin included hypertension requiring medical treatment in 36% of patients, seizures in 15%, and possible PRCA in 6%.⁶

In a study of 25 anemic cats with CKD who were given darbepoetin and had at least 8 weeks of follow-up, 56% had a durable response in which



their PCV reached and stayed at $\geq 25\%$, with an average time to response of about 3 weeks. Ninety-three percent of responders were started at a dose of 1 $\mu\text{g}/\text{kg}$ once weekly, indicating that this dose appears more effective in cats than the lower starting dose of 0.5 $\mu\text{g}/\text{kg}$ once weekly used in some patients. Of the cats that did not respond, 64% had serious concurrent disease, and 45% initially responded but subsequently had their PCV drop below 25% despite continued darbepoetin therapy. This implies that as in human CKD patients, concurrent disease processes may drive anemia and contribute to a poor response to darbepoetin. Hypertension was noted at some point during treatment in 41% of patients, and acute neurologic signs or seizures were noted in 16%, most of which occurred in hypertensive cats. PRCA was considered as a differential for persistent anemia in 8% of cats, but was thought to be unlikely in both cases. Importantly, a significant survival advantage was noted in cats who responded to darbepoetin vs. non-responders, with median survival times of approximately 8 vs. 3 months, respectively.⁷ These results indicate that treatment with darbepoetin can correct anemia and prolong survival time in feline CKD patients.

INTERNAL MEDICINE

Anemia of Chronic Kidney Disease in Cats

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Although the potential benefit of treatment with darbepoetin is clear, the best time to start treatment and what PCV to have as an end goal are not universally agreed upon. Traditionally, synthetic EPO has been recommended in veterinary patients when their PCV drops below 20%, or when they develop significant clinical signs of anemia. The target PCV to reach prior to reducing the darbepoetin dose is typically within the low end of the reference range. However, there is some evidence that even mild anemia predisposes patients to progressive disease. In one study, the median PCV of cats with progressive CKD was 33%, vs. 36% in cats with stable CKD,³ suggesting that even very mild degrees of anemia may have important consequences. This raises the question of if we should consider treating anemic CKD patients sooner and target a higher PCV than was traditionally recommended. Regardless, the most commonly recommended dosing protocol is 1 µg/kg darbepoetin SQ once weekly, along with 50 mg/cat (or 10-20 mg/kg for dogs) iron dextran IM every 1-2 months.⁸ Iron is recommended because iron deficiency is a known complication of darbepoetin therapy and one of the most common reasons for treatment failure in people.¹ PCV and blood pressure should be rechecked weekly until the target PCV is reached. Once this has occurred, dosing can be reduced to every other week, and monitoring should continue to ensure an adequate response is sustained.⁸

Anemia is a common and potentially debilitating complication of CKD, leading to faster progression of disease and increased mortality. Treatment of anemia with darbepoetin is a viable option for these patients and can lead to improved survival and quality of life.

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Avian Bornavirus in Clinical Practice

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Avian Bornavirus (ABV) is an enveloped single-strand RNA virus with worldwide distribution that is responsible for the condition we now know as Proventricular Dilatation Disease (PDD). This condition has been identified in psittacines since the 1970s, and it was originally described as “Macaw wasting disease.” The causative agent, ABV, was only recently identified in 2008, although many other questions regarding this condition still remain. While this disease has been identified in numerous types of birds, this article will be focusing on psittacines, or parrot species, as they are the most commonly seen birds in clinical practice.

PROVENTRICULAR DILATATION DISEASE

The description given to the disease process that is caused by ABV is itself somewhat controversial and contested. The pathology of the disease involves nonpurulent inflammation of peripheral nerves. While the autonomic nerves of the gastrointestinal (GI) tract are commonly affected, this inflammatory process can also affect the central nervous system, as well as other organs entirely, leading to symptoms that may not be related to the GI tract at all. The classic presentation of PDD is a bird that is weak and lethargic, often with a normal appetite. If handled, the owner may notice a decrease in weight, but often this is not noted until there is a significant change. The plumage may appear dull or tattered, due to chronic malnutrition. Feces may contain undigested seeds or other food material, and vomiting or regurgitation is often noted. Most of these symptoms are identified in the more advanced stages of the disease. Symptoms not related to the GI tract may include ataxia, lameness, blindness, or even seizures. These may be seen concurrent with, or independent of, GI changes. Sudden death has been reported, in the absence of clinical symptoms. This may be due to changes or irregularities in cardiac conductivity, although this has not been proven.

Signalment for PDD is quite varied and can include any psittacine, although African grey parrots, Amazons, cockatoos, macaws, and cockatiels appear overrepresented. No age predilection has been identified, although a study in the early 1990s found the average age of affected birds to be 3.8 years old. Latent periods are suspected to be possible, with lengths being quite variable. Single-housed birds have become symptomatic after years of no known contact with, or exposure to, infected individuals.

Differential diagnoses for confirmed proventricular dilatation are numerous and should be ruled out prior to making a diagnosis of PDD. A partial list should include bacterial or mycotic infections, particularly *Macrorhabdus ornithogaster*, neoplasia, parasitism, heavy metal toxicity, or a gastrointestinal outflow obstruction. While proventricular dilatation may be seen with these conditions, they do not typically cause thinning of the proventricular wall as PDD does. If changes to the wall are noted, they usually involve a thickening secondary to chronic inflammation.

TRANSMISSION

Viral RNA has been identified in several bodily fluids and materials, including feces, urine, tears, and oral secretions. Due to these findings, transmission has been assumed to be fecal-oral route. Several studies have found varying degrees of success with this hypothesis. Infected birds introduced into “clean” flocks resulted in spread of the disease to some, but not all, birds. Other studies have shown that horizontal transmission through direct contact was not a sufficient route in immunocompetent birds. Often these patients will test positive for the virus on feathers, or skin biopsies, but will never seroconvert. Due to these findings,

Piepenbring et al. concluded that exposure most likely did not achieve persistent infection. This hypothesis has been strengthened by the fact that multiple reports have identified infected and non-infected birds living together for years, with no transmission.

At the time of this publication, transmission is not fully understood. Both horizontal and vertical transmission appear possible, but several variables are thought to affect each of these, including individual health of the patient, genotype of the virus, and any possible co-infections.

DIAGNOSIS

Diagnosis of a clinical ABV infection is, in a word, challenging. Initial detection of the virus in the living patient is typically done through reverse-transcription polymerase chain reaction (RT-PCR) testing. Samples submitted include cloacal/choanal swabs, tissue, and whole blood. Submitting a swab from only one site, or blood without accompanying swabs, dramatically reduced the likelihood of a positive sample. It is recommended to combine swabs and blood into one tube, although



AVIAN AND EXOTIC

Avian Bornavirus in Clinical Practice

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individual labs may have different submission requirements. Less commonly run tests to identify ABV include immunohistologic staining and viral isolation. ABV-specific antibody detection (ELISA, Western blot) may provide valuable information for diagnosis, although at the present time it is unclear when antibodies are produced, or if they can potentially be correlated to clinical signs.

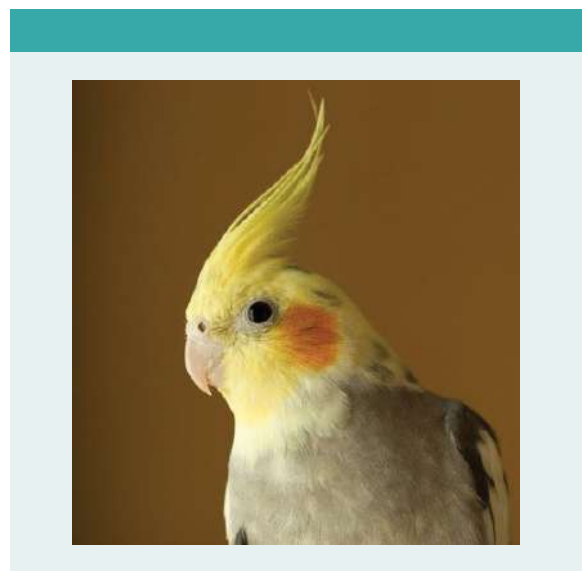
Another factor that adds even more confusion is the fact that some birds become infected with ABV, shed the virus, but never seroconvert. Several theories for why this may happen have been introduced, although none have been proven. Based on this information, the following protocols have been proposed to help identify positive and negative birds. A bird that is found to be positive for ABV using both PCR and serology should be considered a positive infection. A bird that is positive on PCR, but negative on serology, should be retested in 4-6 weeks to check for a false positive PCR and also to monitor for seroconversion. Repeated positive PCR tests, and/or seroconversion, should be considered a positive bird. Negative PCR paired with a positive serology are thought to be carriers, although this has not been proven. Persistent elevated titers are indicative of infection in mammals infected with bornavirus and should be considered the same for birds. Some birds appear to be able to clear the infection, which would explain the positive serology and negative PCR, although this too has yet to be proven.

TREATMENT

Several therapies have been proposed for treatment of both the symptoms of ABV, as well as for the virus itself. To date, there are no successful antiviral therapies reported. Previous reports of nonsteroidal anti-inflammatory drugs have failed to show significant, repeatable success. One study using meloxicam in infected cockatiels actually showed more severe lesions, and increased mortality, when compared with the control group. Immunosuppressive treatments, including the use of glucocorticosteroids, have been proposed at recent conferences. No published data has been made available as of yet, and clinicians should be cautious of these currently unproven methods. Symptomatic treatment, including diet modification, analgesia, and control of secondary infections, should be the basis of treatment for these cases.

CONCLUSION

Despite the recent advances in identifying and diagnosing this condition, there are still many questions left unanswered. While diagnostics have improved considerably, interpretation of these tests can still be challenging. A positive ABV diagnosis should not result in euthanasia, especially since many patients who test positive will never show symptoms. That being said, once patients become symptomatic, the chances of long-term survival are very low, and quality of life should be assessed. Along with the discovery of additional symptoms, not related to the GI tract, some have proposed a name change from proventricular dilatation disease to avian or neuropathic ganglioneuritis. While this more accurately describes the condition, a name change has not been uniformly agreed upon at this time.



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Keeping the Geriatric Canine Mobilizing!

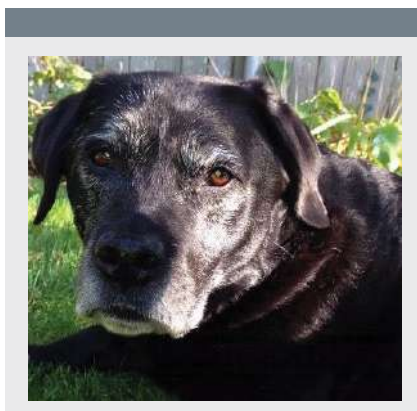
Amy Straut, DVM, CCRT
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Just like a human's, the canine body changes with age. Both species get a little grayer and often experience hearing loss and eyesight impairment. And, just like us, geriatric dogs typically experience a decrease of muscular strength and endurance and often encounter a slower metabolism.

There is much benefit to keeping an older dog moving safely. Dogs are typically social creatures, and it is extremely important to assist them in maintaining their socialization for best quality of life. In addition to keeping a dog mentally engaged, safe exercise encourages weight management, musculoskeletal maintenance, and proper joint function, as well as cardiovascular and respiratory conditioning.

More and more, at Angell-West Physical Rehabilitation, we are being asked to design strength and conditioning programs for senior dogs. In most cases, these dogs have either encountered a physical injury, illness, or recent surgery that resulted in a degradation of their physical mobility and/or stamina. In addition to an acute injury, these older patients often present with comorbidities, including an underlying element of chronic osteoarthritis.

Because one size does NOT fit all when developing a physical rehabilitation program for these patients, we implement an individualized multimodal plan of care that encompasses:



- Pain evaluation and management
- Lifestyle considerations
- Nutritional requirements
- Proper rehabilitation techniques

Elderly patients are often referred to our services with an established, adequate pain management protocol already in place. In cases where a dog either needs increased or decreased pain management, we encourage patient owners to discuss further options with their primary care veterinarian. In these cases, we do our best to contact the primary care veterinarian to offer assistance where helpful.

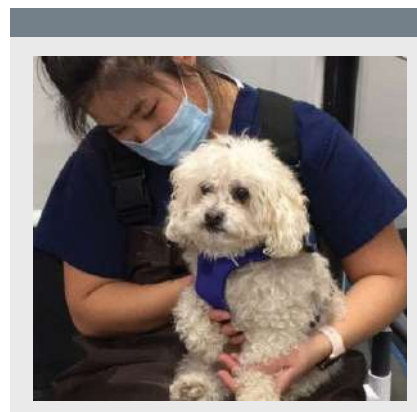
We review each patient's lifestyle with their owner to make sure that we address all considerations:

- Harness and shoe requirements
- Secure floor surfaces
- Safety around access to furniture and stair navigation
- Interaction with other animals and children
- Assistance into and out of car, as well as car riding
- Sleeping arrangements
- Access to food and water

Proper nutrition is critical for overall well-being, healing assistance, and weight management. We discuss diet including appropriate form, balance of nutrients, specialized ingredients, and food frequency and volume offered. We also discuss the use of supplemental anti-inflammatory agents (omega 3s, CBD) and gastrointestinal support (fibers, probiotics) where appropriate.

It is imperative to obtain a thorough knowledge of all comorbidities inherent in the patient. As a result, when assigning rehabilitation techniques, it is extremely important to keep the following in mind:

- Preparation of environment (flooring, equipment, additional human resources, temperature)
- Appropriate warm-up & cool-down requirements



- Proper pace and intensity level of each movement
- Required rest post-exercise

We use all of the same rehabilitation equipment with our geriatric clientele as we do with our younger patients.

- Land therapeutic exercise
- Manual therapies: passive range of motion (PROM), massage, stretching
- Supplemental therapies: low-level laser, acupuncture, chiropractic care
- Hydrotherapy: water treadmill, therapeutic pool swimming

It is wonderful to witness the joy in finding the appropriate level of safe exercise for an elderly dog who wants to be active! In addition to gold standard pain management methodology as well as proven strength, conditioning, and weight-management protocols...creativity is the backbone of designing a proper physical rehabilitation program for each elderly dog we work with!

Dogs are honest creatures. They typically work hard and rise to the occasion when gently challenged to find fun ways to enhance or maintain their physical well-being. Let's collectively offer our elderly dogs their best quality of life!

Physical Rehabilitation at MSPCA-Angell West

Watch patients enjoy land-based and hydro-therapy treatment in our Physical Rehabilitation facility at angell.org/rehab

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

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

Currently physical rehabilitation services include:

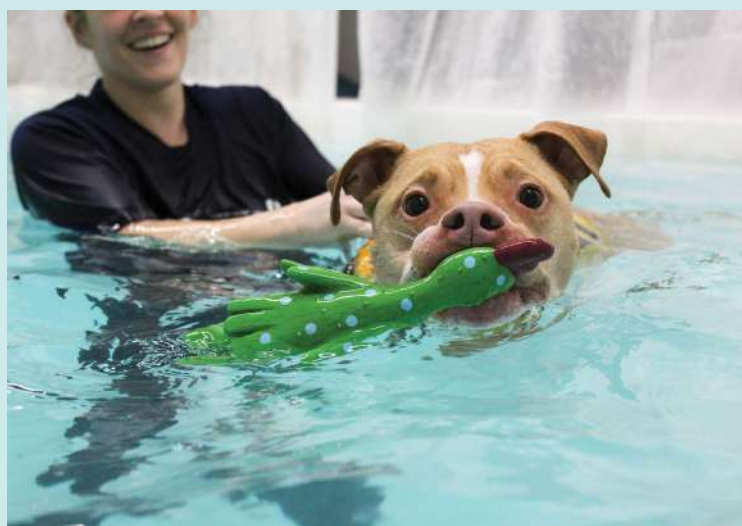
- Hydrotherapy
- Land-based exercise
- Manual therapy
- Therapeutic laser treatment
- Massage
- Consultation and fitting of assistive devices
- Chiropractic



Land-based Exercise Area



Hydrotherapy – AquaPaws Water Treadmills



Hydrotherapy – Indoor Pool



Treating Albuterol Toxicity

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Toxin ingestion is a common presenting complaint among pets in the emergency room, and of all those incidents, according to the ASPCA Animal Poison Control Center, the most commonly reported is ingestion of human medications. Albuterol ingestion and resulting toxicity is somewhat uncommon, and unlike with the more frequently occurring chocolate or grape toxicity, a veterinarian faced with this situation may not immediately know how to proceed. A retrospective study on this subject was recently published in the *Journal of Veterinary Emergency and Critical Care* by veterinarians from the Angell Emergency and Critical Care service. This study outlined the pathophysiology, treatments, and outcomes for 36 dogs who presented through the emergency service for albuterol toxicosis. The fortunate news for our patients and their owners is that most dogs tend to do very well with prompt supportive care and for a relatively short duration of hospitalization.

ALBUTEROL PHARMACOKINETICS

Albuterol is used as a human (as well as a veterinary) treatment for asthma. It acts as a selective β_2 -adrenergic receptor agonist to provide bronchodilation by relaxing smooth muscles in the bronchi. It is short-acting and considered effective for quick relief of symptoms of bronchoconstriction, although it does not relieve long-term inflammation of the airways due to the underlying disease processes. Albuterol and other β_2 agonists also cause transient hypokalemia and hypophosphatemia via an unknown mechanism, although both of these are due to intracellular shifts of these ions rather than overall depletion. One hypothesis is that stimulation of the Na-K-ATPase pumps causes intracellular influx of potassium. In addition to its primary purpose of bronchodilation, albuterol also causes peripheral vasodilation and cardiac stimulation. Paradoxical bronchoconstriction has also been reported, as well as exacerbation of asthma symptoms in patients taking large quantities of β_2 agonists. The shifts in potassium can also cause cardiac arrhythmias, including ventricular arrhythmias, due to delayed repolarization of the myocardium.

These mechanisms can, of course, be life-threatening if not identified and addressed promptly.

PRESENTATION

The route of exposure for the veterinary patient depends on the prescribed form of the albuterol. In all species, including humans, it is most commonly prescribed as an aerosol in a canister, which, when punctured, immediately delivers a dose via inhalation or exposure through the mucus membranes. Asthma inhalers often contain up to 200 intended doses, depending on brand and number of previous uses. If a canister is chewed and punctured, it can potentially administer a severe overdose in a very short period of time. Ingestion of an oral form of the medication is another possible route of exposure. The clinical signs most commonly seen include sinus tachycardia, tachypnea, lethargy, and vomiting (see Table 1). Signs such as collapse and death have also been reported, but they are very uncommon. Patients often appear agitated or, conversely, lethargic. On intake to the hospital, they may also be noted to have hypertension or hypotension. Often, exposure to albuterol is documented by the owners, but in the absence of this a full history of potential toxins in the household should be taken.

It is not recommended to induce emesis unless the patient ingested albuterol pills. With the more common method of inhalation or solution ingestion, the focus is instead placed on cardiovascular and electrolyte support for the duration of clinical signs (usually up to 12 hours). In the case of ingestion of pills, activated charcoal decontamination can also be implemented. On intake, a minimum database that includes serum potassium, phosphorous, and glucose should be performed. Some publications also advise measurement of cardiac troponins to assess for myocyte damage.

If admission to a hospital is possible, intravenous fluid therapy should be implemented for cardiovascular support. Potassium supplementation should be started if hypokalemia is noted. Propranolol, which acts as a non-selective β -blocker, can be used as a

specific antagonist to treat the tachycardia and should be administered intravenously at 0.02-0.06mg/kg every 8 hours to effect. To control hypertension, esmolol (loading dose of 50-200mcg/kg IV slowly to effect over 1-2 minutes, then 50-200mcg/kg/min as needed) or metoprolol (0.5mg/kg every 12 hours) can be considered as a more specific β_1 -blocker. While ventricular arrhythmias were seen in only one patient during the retrospective study mentioned earlier, they have been reported throughout the literature and lidocaine can be administered to control these should they arise. Electrocardiogram monitoring for heart rate and rhythm should be strongly considered, and monitoring of serum potassium and phosphorus should be performed every 4-6 hours for 12 hours.

If resources permit, at least a brief EKG and basic electrolyte screening panel should be performed. Subcutaneous fluids should be administered for support, and if potassium on intake is found to be low, oral supplementation can be implemented for three days' duration. Tachycardia can be controlled with oral administration of the β -blocker atenolol (0.2-1.0mg/kg every 12-24 hours) for the same three days' duration. Ideally, blood work would be rechecked the next day to ensure that electrolyte normalcy has been achieved.

FIGURE 1

Retrospective evaluation of albuterol inhalant exposure in dogs: 36 cases (2007-2017). Meroni, Emiliana R. et al. *Journal of Veterinary Emergency and Critical Care*. 2020; 1-8.

TABLE 1 Clinical signs at hospital presentation in 36 dogs after albuterol exposure

| Clinical sign | Number of dogs | Percentage (%) |
|---------------------|----------------|----------------|
| Tachycardia | 34 | 94 |
| Tachypnea | 24 | 67 |
| Lethargy | 13 | 36 |
| Vomiting | 11 | 31 |
| Polyuria/polydipsia | 7 | 19 |
| Hyperthermia | 5 | 14 |
| Hypotension | 3 | 8 |
| Ataxia | 3 | 8 |
| Tremoring | 3 | 8 |
| Mydriasis | 2 | 6 |

EMERGENCY AND CRITICAL CARE

Treating Albuterol Toxicity

CONTINUED FROM PAGE 14

PROGNOSIS

The recent retrospective study showed a good outcome for all 36 dogs included, both those who underwent inpatient and outpatient management. While the average hospital stay for those who were managed as inpatients was around 20 hours, most patients who can be supported through the first 12-18 hours will not have residual signs. However, patients reported to develop arrhythmias or who may have already had underlying cardiac disease are at a higher risk and the prognosis may be more guarded in these cases. By taking a complete history, conduct basic diagnostics, and providing symptomatic care, a positive outcome is usually achieved.

FIGURE 2

↪ <https://www.petpoisonhelpline.com/poison/asthma-inhaler/>



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↪ Courtesy Shuttle for Patients Needing Further Specialized Care

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

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

▾ Angell Fall Continuing Education — **Registration is Open!**

Practical Techniques in Veterinary Medicine

Sunday, October 3, 2021

8:15am – 2:45pm

5 Interactive CE Credits (pending RACE approval)

TOPICS:

- **Top 10 Tips for Better Pathology Submissions;** *Patty Ewing, DVM, MS, DACVP*
- **The Neurologic Exam;** *Michele James, DVM, DACVIM (Neurology)*
- **Emergency Preparedness;** *Ashley Lockwood, DVM, DACVECC*
- **Treating GI Stasis in the Herbivore Patient and Appropriate Antibiotic Use in Rabbits;** *Patrick Sullivan, DVM, DABVP (Avian Practice)*
- **Practical Approach to CPR;** *Catherine Sumner, DVM, DACVECC*

Making the Cut: 1) A Practical Approach to Surgical Upper Airway Disease in Dogs and 2) Treating Hip Luxation

Wednesday, October 20, 2021

6:15pm – 8:45pm

2 Interactive CE Credits (pending RACE approval)

TOPICS:

- **A Practical Approach to Surgical Upper Airway Disease in Dogs;** *Nicholas Trout, MA, VET MB, DACVS, ECVS*
- **Hip Luxation;** *Emily Ulfelder, BVetMed, DACVS*



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The clinics do not provide overnight care, specialty service care, or 24/7 emergency care. Appropriate cases will be referred to Angell's Boston or Waltham facilities or a surrounding specialty veterinary referral hospital.

To see if your clients qualify for financial assistance, please visit angell.org/essex or angell.org/Nashoba



angell at nashoba

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

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



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

➤ MSPCA-Angell Clinics now in Boston, Methuen, and Cape Cod

Offering subsidized veterinary care to help keep pets and families together.

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angell clinic

Boston | Cape Cod | Nevins Farm

MSPCA-Angell Cape and Boston clinics
Monday – Friday 8am-4pm

MSPCA-Angell Nevins Farm in Methuen
Monday – Saturday 8am-4pm

To refer low income clients, please visit angell.org/referrals

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

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

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

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

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