

COMMON MISTAKES TO AVOID IN YOUR POISONED PATIENTS

Justine A. Lee, DVM, DACVECC, DABT

CEO, VETgirl, LLC.

justine@vetgirlontherun.com

<http://www.vetgirlontherun.com>

In the veterinary poisoned patient, the goal of decontamination is to “inhibit or minimize further toxicant absorption and to promote excretion or elimination of the toxicant from the body.”^{1,2} When treating the poisoned patient, the clinician should have an understanding of the toxic dose (if available), the pharmacokinetics (including absorption, distribution, metabolism, and excretion), the underlying mechanism of action, and the potential clinical signs that can be observed with the toxicant.² This will help determine appropriate decontamination and therapy for the patient. If this information is not readily available, the reader is advised to contact the [ASPCA Animal Poison Control Center](http://www.aspcapoisoncontrol.com) (888-426-4435) for life saving, 24/7 advice as needed. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

Appropriate decontamination and therapy is indicated to improve the overall prognosis and outcome of the small animal poisoned patient. The use of decontamination, if and when appropriate, should be implemented to help prevent further toxicant absorption. Gastrointestinal (GI) decontamination (including emesis induction and/or administration of activated charcoal with a cathartic) is considered the best method of limiting absorption and preventing continued exposure to potential toxicosis in veterinary medicine. This is particularly beneficial with potentially harmful or life-threatening ingestions. It is imperative, however, to consider whether decontamination is appropriate, as it may be too late or contraindicated (resulting in potentially further harm). Evaluation of the potential risk associated with induction of emesis needs to be considered. Five key mistakes to avoid in the poisoned patient include:

1. Not obtaining an appropriate toxicology history
2. Not triaging the poisoned patient appropriately
3. Not knowing the indications or contraindications for emesis induction
4. Using the wrong emetic agent to induce emesis with
5. Not knowing more about activated charcoal

NOT OBTAINING AN APPROPRIATE TOXICOLOGY HISTORY

One of the first mistakes made in the field of veterinary toxicology is not taking the time to obtain an appropriate toxicology history. Some key questions to ask prior to consideration for emesis induction include:

- What was the product ingested? Do you know the active ingredient?
- Can you bring me the original box/container/pill vial?
- How many total tablets could have been ingested? What was the minimum and maximum amount that your pet could have been exposed to?
- Was this an extended- or sustained-release product? Was there an extra “letter” behind the brand name (e.g., Claritin vs. Claritin-D)?
- When did your pet get into this?
- Has your pet shown any clinical signs yet?
- Did you give your pet anything at home (e.g., hydrogen peroxide, salt, milk) when you found out he was poisoned?

NOT TRIAGING THE POISONED PATIENT APPROPRIATELY

The second important consideration is to make sure that pet owners are instructed to do the following:

- Safely remove their pet from the area of poisoning so additional ingestion does not occur
- Do *not* give any home remedies found circulating on the Internet (e.g., milk, peanut butter, oil, grease, salt)
- Do not induce emesis without consulting a veterinarian or the ASPCA animal poison control center first.
- Bring the pill vial, bait station, or container in to the veterinarian so they can assess the bottle for verification of the product name and/or active ingredient.
- Have the pet owner call the original pharmacy to find out how many total pills were prescribed, and attempt to back-count how many were taken/ingested.
- Seek immediate veterinary attention.
- Provide adequate ventilation (e.g., rolling down the windows, turning on the air conditioner) if emesis occurs with zinc phosphide toxicosis as the phosphine gas is also poisonous to humans.

Once the poisoned patient is presented to the clinic, veterinarians should do the following:

- Re-verify the spelling of the product and confirm the active ingredient (AI).
- Evaluate if the product is a sustained-release (SR), extended-release (XR), or long-acting (LA) product. These initials will follow the name of the drug on the vial.
- Evaluate whether the patient should have emesis induced (see “Not knowing the indicators or contraindications for emesis induction”).
- Stabilize the patient based on triage and physical examination findings (e.g., temperature, heart rate, pulse rate, pulse quality).
- Call for medical assistance and toxicology advice if needed.

NOT KNOWING THE INDICATIONS OR CONTRAINDICATIONS FOR EMESIS INDUCTION

The goal of decontamination is to inhibit or minimize further toxicant absorption and to promote excretion or elimination of the toxicant from the body. Decontamination can only be performed within a narrow window of time for most substances; therefore, it is important to obtain a thorough history and time since exposure to identify whether decontamination is safe for the patient or if it will actually be beneficial for the patient. Decontamination categories may include ocular, dermal, inhalation, injection, GI, forced diuresis, and surgical removal to prevent absorption or enhance elimination of the toxicant.

One of the primary ways of decontaminating veterinary patients is via emesis induction. While gastric lavage is often more effective at removing gastric contents, it is less often performed in veterinary medicine as it requires intravenous (IV) catheter placement, sedation, intubation with an appropriately inflated endotracheal tube (ETT), and appropriate gavage technique. Veterinarians should be aware of which circumstances are appropriate for emesis induction versus gastric lavage, and be aware of contraindications for emesis induction.

Inappropriate Timing of Decontamination (See Table 1)

Emesis induction should only be performed with recent ingestion of a toxicant or unknown time of ingestion in an *asymptomatic* patient. The more rapidly emesis is induced post ingestion, the greater yield of recovery of gastric contents. Studies have shown that gastric recovery within 1 hour after toxin ingestion was approximately 17% to 62%. When emesis was induced within an even shorter time span (within 30 minutes), mean recovery of gastric contents was approximately 49% (range 9–75%). If several hours have elapsed since ingestion, the contents have likely moved out of the stomach and emesis will no longer be of benefit.^{1,2} While delayed emesis may still sometimes be successful, the amount of gastric

recovery significantly decreases as time passes. That said, induction of emesis can be performed in *asymptomatic* patients up to 4 hours post ingestion, particularly with certain toxicants.^{1,2}

In certain circumstances, delayed emesis induction can be performed within 4 to 6 hours of ingestion provided the patient remains *asymptomatic* with the following circumstances: when certain toxins that delay gastric emptying are ingested (e.g., salicylates, opioids, anticholinergics, tricyclic antidepressants) or if the toxin is known to physically stay in the stomach for a longer duration of time or form a large bezoar or concretion (e.g., iron tablets, a large amount of chewable multivitamins, bone or blood meal). Additional examples include:

- Large wads of xylitol gum
- Large amounts of chocolate
- Grapes and raisins
- Foreign material (e.g., sawdust/wax, kitty litter, bone meal)

Not Knowing the Contraindications for Emesis Induction

Certain animals with underlying medical concerns should not have emesis induced (particularly at home by the pet owner) due to a higher risk of aspiration pneumonia or secondary complications. Examples include a prior history of laryngeal paralysis, megaesophagus, aspiration pneumonia, and upper airway disease. Likewise, certain species and breeds may limit our ability to perform emesis induction. Most dogs, cats, ferrets, and potbelly pigs can be safely induced to vomit.³ Certain breeds (e.g., pug, English bulldog, Shih-Tzu) with brachycephalic syndrome (e.g., elongated soft palate, stenotic nares, everted sacculles, and a hypoplastic trachea) may be better candidates for sedation and gastric lavage rather than emesis induction due to the risks of aspiration pneumonia.¹ Rabbits, ruminants (e.g., sheep, cattle, llamas, and goats), horses, birds, and rodents (e.g., chinchillas, rats, gerbils) cannot safely have emesis induced or may not anatomically be able to vomit.³

Likewise, there are certain toxic ingestions where emesis should never be induced. Emesis should not be performed when agents such as caustic or corrosive substances (e.g., undiluted drain cleaners, toilet bowl cleaners, hydrochloric acid, concentrated sodium hypochlorite, lye products) are ingested. These agents can result in further burns and corrosive injury to the stomach, esophagus, and mouth when vomiting occurs after ingestion. In addition, if hydrocarbons and petroleum distillates (e.g., gasoline, mineral spirits, fuel, kerosene, furniture polish oils) are ingested, emesis should never be induced. These low viscosity liquids are very easy to aspirate when the patient vomits; therefore, emesis is contraindicated due to the high risk of aspiration.

Induction of Emesis in the Symptomatic Patient

Patients that are already symptomatic for the toxicosis should never have emesis induced. Certain toxicoses may result in severe sedation, a decreased gag reflex, or reduce the seizure threshold, increasing the risk for aspiration pneumonia during emesis induction. Patients with a lowered seizure threshold have the potential to develop seizures during emesis induction. As the patient is already symptomatic, the toxin has likely been already absorbed, and emesis induction is typically unrewarding.

USING THE WRONG EMETIC AGENT TO INDUCE EMESIS WITH

Emetic agents work by causing local gastric irritation, stimulating the central nervous system (CNS) chemoreceptor trigger zone (CRTZ), or a combination of gastric irritation and CNS stimulation.^{1,2} Considerations in choosing an emetic agent are broad and varied. Many home or Internet remedies are used without success and have the potential of causing further harm. Emetic agents are not effective if

an antiemetic such as ondansetron or maropitant has been previously administered. Currently, the only home recommendation for dog owners is hydrogen peroxide, while veterinary-prescribed emetic agents include apomorphine hydrochloride (dog) and xylazine hydrochloride (cat).^{1,2}

Hydrogen peroxide (H₂O₂) works by local irritation of the oropharynx and gastric lining, which results in a gag reflex. It is usually recommended for oral administration by the dog owner when transportation to a veterinary clinic is delayed. Only a 3% hydrogen peroxide solution should be used, as higher concentrations can potentially be corrosive to the GI mucosa. Adverse effects associated with use of H₂O₂ as an emetic agent include irritation to the GI tract, gastroduodenal lesions, gastric dilatation and/or volvulus (dogs), and potential for aspiration pneumonia.^{1,2,4} When using hydrogen peroxide as an emetic agent in dogs, the administration of sucralfate and antacids (e.g., proton-pump inhibitors or H₂ blockers) should be considered. Hydrogen peroxide is not a reliable emetic in cats and its use generally is NOT recommended in this species. In addition, cats can develop profound clinical signs from the administration of H₂O₂, including profuse foaming from the mouth and severe hemorrhagic gastritis.

Apomorphine hydrochloride is a centrally acting emetic agent. Administration results in stimulation of the CRTZ, quickly followed by emesis. Adverse effects associated with apomorphine administration are prolonged emesis and ocular irritation when administered subconjunctivally.^{1,2} Apomorphine should not be used in cats, as it is not considered to be effective. Apomorphine should not be used when there has been ingestion of medications that result in compounding of symptoms (e.g., respiratory or CNS depression) or with antidopaminergic drugs (e.g., metoclopramide) that prevent emesis from occurring.

Dexmedetomidine and xylazine hydrochloride, alpha adrenergic agonists, are centrally-acting emetic agents that are used as emetic agents in cats. The use of apomorphine and hydrogen peroxide are *not* recommended for cats, as they are ineffective or can result in severe adverse effects (e.g., hemorrhagic gastritis), respectively. Xylazine does not reliably produce an emetic response in dogs, and thus is not recommended in dogs as an emetic agent. Adverse effects associated with alpha-adrenergic drugs include bradycardia, sedation, tremors, and respiratory depression.^{1,2} Thawley and Drobotz found that dexmedetomidine (7 mcg/kg, IM) resulted in emesis approximately 80% of the time in cats, as compared to only about 44% of the time in cats with xylazine.⁵ A similar study by Willey et al supported this.⁶ Alpha adrenergic agonists should not be used in cats that have ingested medications (e.g., other alpha-adrenergic agonist drugs) or products that may result in compounding of bradycardia, respiratory depression, sedation, or CNS depression symptoms.^{1,2}

Methods that are not recommended for emesis induction include digital induction of emesis, syrup of ipecac, liquid soaps, dry mustard powders, and salt. Digital induction of emesis often results in physical injury to the pet owner (dog bite), or injury to the pet's throat and soft palate. Syrup of ipecac has historically been recommended to induce emesis, but is no longer the standard of care. Its cardiotoxic potential and tendency to result in prolonged vomiting, lethargy, and diarrhea have caused it to fall out of favor in both human and veterinary medicine.^{1,2} Soaps, mustard powders, and table salt are not reliable as induction agents and may be detrimental (e.g., resulting in further complications such as hypernatremia of the patient).

NOT KNOWING MORE ABOUT ACTIVATED CHARCOAL

After an appropriate history, triage, physical exam, and initial decontamination procedures have been performed in the poisoned pet, the next step is the administration of activated charcoal (AC), if appropriate. Activated charcoal should not be given to the poisoned patient when the toxicant does not reliably bind to AC (see below) or when it is contraindicated to administer AC (e.g., salt toxicity, poor gag

reflex). In addition, symptomatic patients who are at risk for aspiration pneumonia should not be administered AC orally. Finally, the administration of AC with a cathartic should be cautiously used in dehydrated patients due to the potential (albeit rare) risks for hypernatremia secondary to free water loss in the GI tract.

When administering AC, it should ideally be given within ≤ 5 minutes of ingestion to be most effective. In veterinary medicine, this is almost impossible due to driving time (to the clinic), lapsed time since ingestion, time to triage, and the amount of time it takes to physically deliver AC (e.g., syringe feeding, orogastric tube). As a result, administration of AC is often delayed for up to an hour or more. As time since ingestion is often unknown (e.g., pet owner coming home from work to find their pet poisoned), decontamination (including emesis and administration of AC) is often a relatively benign course of action, provided the patient is not already symptomatic. As always, when administering *any* drug, it is important that benefits outweigh the risks, and that complications be prevented when possible. In veterinary medicine, administration of AC with a cathartic as long as 6 hours out may still be beneficial with certain types of toxicosis, particularly if the product has delayed release [e.g., extended release (XR) or sustained release (SR)] or undergoes enterohepatic recirculation (see multi-dose AC below). While human medicine has moved away from administration of AC with poisoned patients, the aggressive use of AC in veterinary medicine is still warranted, as this is often our last line of defense when it comes to adequately decontaminating our patients. Certain modalities of therapy—e.g., antidotes [such as fomepizole, pralidoxime chloride (2-PAM), digoxin-specific antibody fragments], plasmapheresis, hemodialysis, mechanical ventilation—along with financial limitations of pet owners, limit our ability to treat poisoned pets aggressively as compared to human medicine. As a result, the continued use of AC in veterinary medicine is still warranted as a first line of defense therapy. Current recommended dosing for single dose AC is 1–5 g of AC/kg with a cathartic (e.g., sorbitol) to promote transit time through the GI tract.

Administration of Activated Charcoal When the Toxicant May Not Bind Appropriately

Before administering AC and a cathartic, it is imperative to consider whether or not the patient has a contraindication for its administration. Contraindications for AC administration include severe sedation, decreased gag reflex, or intestinal obstruction. Likewise, if the toxicant does not physically bind to AC, it is contraindicated to administer AC. Examples of toxicants that do not absorb reliably to AC include ethylene glycol, alcohol, xylitol, and heavy metals. Contraindications for cathartic administration include hypernatremia, dehydration, and salt toxicosis (e.g., salt, ice melters, homemade play dough), as fluid loss through the intestinal tract can result in excessive free water loss and severe, secondary hypernatremia.

Multi-dose Activated Charcoal

Human studies have found that multi-dose AC significantly decreases the serum half-life of certain drugs, including antidepressants, theophylline, digitoxin, and phenobarbital. While veterinary studies are lacking, there is likely an added benefit from using multi-dose AC, provided the patient is well hydrated and monitored appropriately. Certain situations or toxicities, including drugs that undergo enterohepatic recirculation; drugs that diffuse from the systemic circulation back into the intestinal tract down the concentration gradient; or ingestion of SR, XR, or long-acting (LA) release products will require multi-dose administration of AC. Keep in mind that when administering multiple doses of AC to a patient, the additional doses ideally should *not* contain a cathartic (e.g., sorbitol), due to increased risks for dehydration and secondary hypernatremia. Current recommended dosing for multiple doses of AC is 1–2 g of AC *without a cathartic* /kg of body weight, PO q 4–6 hours for 24 hours.

Contraindications of Activated Charcoal

Contraindications for AC include endoscopy (which would obscure visualization), abdominal surgery of the GI tract, gastric or intestinal obstruction, gastrointestinal hemorrhage or perforation (due to pathology, caustic injury, etc.), recent surgery, late-stage presentation with clinical signs already present, dehydration, lack of bowel motility, ileus, hypernatremia, hypovolemic shock, compromised airway (risk for aspiration pneumonia), and ingestion of a caustic substance or hydrocarbon (due to increased risk for aspiration pneumonia). In patients that have an unprotected airway that are at risk for aspiration pneumonia (e.g., a depressed state of consciousness, excessive sedation), the use of AC is contraindicated without ETT intubation (to protect the airway during gastric lavage and AC administration).

CONCLUSION

The appropriate and careful use of decontamination of the poisoned patient should be considered. Thorough history taking and physical examination of the patient is imperative prior to emesis induction. Recognizing contraindications for emesis induction, or which emetic to use for emesis induction, is imperative. With careful and thorough evaluation of the poisoned patient, proper decontamination can be performed confidently with safety and efficacy to aid in ensuring a positive outcome.

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NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb's Veterinary Drug Handbook*.

Table 1. Emesis Induction: Indications and Contraindications[1,2]*

WHEN EMESIS SHOULD BE PERFORMED	WHEN EMESIS SHOULD <u>NOT</u> BE PERFORMED:
With recent ingestion (<1-2 hours) in an asymptomatic patient	With caustic or corrosive toxicant ingestion (e.g., batteries, ultra-bleach, lye, oven cleaning chemicals), where emesis induction may result in further injury to the oropharynx, esophagus, and GIT when these agents are expelled.
With unknown time of ingestion in an asymptomatic patient	When petroleum distillates or hydrocarbons are ingested (e.g., kerosene, gasoline, motor oil, transmission fluid, etc.); these toxicants can be easily aspirated into the respiratory system and result in severe aspiration pneumonitis.
When ingestion of a product known to stay in the stomach for a long time is ingested in an asymptomatic patient (e.g., bezoar, massive ingestions, grapes/raisins, chocolate, wads of xylitol gum, FBO, etc.)	In symptomatic patients that have a decreased gag reflex (e.g., sedation, coma, hypoglycemia, etc.) or a lowered seizure threshold (e.g., tremoring, seizing, etc.) that may be unable to protect their airway, resulting in aspiration pneumonitis.
	In patients with underlying medical conditions that may predispose them towards aspiration pneumonitis or complications associated with emesis induction (e.g., megaesophagus, history of aspiration pneumonia, upper airway disease, laryngeal paralysis). Brachycephalic breeds (e.g., English bulldog, pug, Shih-Tzu) with an elongated soft palate, everted sacculles, a hypoplastic trachea, or stenotic nares may be better candidates for sedation, intubation, and gastric lavage rather than emesis induction due to the risks of aspiration.
	Species that anatomically cannot vomit or cannot safely have emesis induced such as birds, rabbits, ruminants (e.g., sheep, cattle, llamas, and goats), horses, and rodents (e.g., chinchillas, rats, gerbils)

* From Lee JA, Decontamination of the poisoned patient, in the *Textbook of Small Animal Emergency Medicine* (Eds: Drobatz et al).

COMMON MISTAKES TO AVOID IN THE ER

Justine A. Lee, DVM, DACVECC, DABT

CEO, VETgirl

Justine@vetgirlontherun.com

www.vetgirlontherun.com

Saint Paul, MN

Don't see emergency cases every day? Have a dog presenting to you with pale mucous membranes, a weak pulse, a heart rate of 190 bpm, and you're not sure what to do next? Have a dyspneic cat fish-mouth breathing in front of you? This article discusses how to avoid 10 common errors in emergency patients that will save your patient's life, including when to tap that dyspneic cat's chest, when to reach for that "FAST" ultrasound, or the best time to do chest radiographs. Having practiced in the trenches of a busy inner-city emergency room to the ivory tower of academia, I've seen these mistakes made, and I've made them myself. Here, some common mistakes to avoid in the emergency room.

NOT DOING CHEST RADIOGRAPHS

One of the most common mistakes in the emergency room is not performing chest radiographs (a "met check") as part of routine geriatric diagnostics. Geriatric patients (defined as a dog > 6–7 years of age [size-dependent] or a cat > 12 years of age) with, for example, hepatosplenomegaly, icterus, hemoabdomen, immune-mediated disease, or fever of unknown origin should have chest radiographs done at the same time as abdominal radiographs. Typically, a three-view chest set is the method of choice; however, this may be difficult in emergency patients with dyspnea. That said, a right- and left-lateral chest radiograph is also an effective way to screen for metastasis. While a met check is often a "low-yield test" (i.e., the likelihood of identifying chest metastasis is relatively low), it is an important screening tool that can help veterinarians counsel pet owners on end-of-life decision-making and overall prognosis.

USING THE SHOCK DOSE OF FLUIDS

The "shock dose" of fluids is extrapolated from the blood volume (60–90 ml/kg for dogs; 60 ml/kg for cats). More recently, emergency critical care specialists have moved away from using the entire shock dose when trying to stabilize hypovolemic patients—smaller aliquots (e.g., one-quarter to one-third of a shock dose) of intravenous (IV) crystalloids are preferred. A patient rarely requires replacement of the whole blood volume with crystalloid fluids.

USING THE WRONG DOSE OF STEROIDS

Traditionally, "shock doses" of steroids have been listed in emergency books (e.g., dexamethasone sodium phosphate [DexSP] 4–6 mg/kg). However, criticalists have moved away from giving steroids with trauma because of potential deleterious effects (including gastric ulceration in a poorly perfused "shock gut" in the dog, exacerbation of hyperglycemia, and delayed wound healing). More recently, we have moved to different *doses* of DexSP. Antiinflammatory doses of DexSP are generally considered 0.1 mg/kg, whereas immunosuppressive doses are as low as 0.25 mg/kg IV q 12 to 24 hours. For that reason, the 4–6 mg/kg dose for shock is no longer indicated. Remember that DexSP is approximately 8 to 15 times stronger than prednisone, and one is unlikely to need 40 mg/kg of prednisone in trauma cases.

Clinical application

- For cases warranting immunosuppression (e.g., immune-mediated hemolytic anemia), consider using lower doses of DexSP (0.25 mg/kg IV q 12–24 hours).

- Avoid the “shock doses” of steroids—if you are giving more than a few milliliters, it’s probably too much.
- Concurrent use of nonsteroidal antiinflammatory drugs and steroids should still be avoided to minimize GI effects.

GIVING STEROIDS TO HEAD TRAUMA PATIENTS

Recently, the use of steroids in both human and veterinary head trauma has been widely debated. Although research in this topic is voluminous, there are no experimental or clinical studies demonstrating a *clear* benefit of steroids in head trauma. In one human study, a meta-analysis of randomized, controlled trials did not show a beneficial response from steroid therapy (1). Unfortunately, steroids have been associated with the following deleterious side effects: gastrointestinal (GI) bleeding, hyperglycemia, immunosuppression, delayed wound healing, and perpetuation of a catabolic state. Currently, the brain trauma foundation guidelines state that glucocorticoids are “not recommended for improving outcome or reducing intracranial pressure in head-injured patients” (2). The “CRASH” (Corticosteroid Randomisation After Significant Head injury) study demonstrated that overall mortality was statistically higher in patients who were treated with steroids (3).

Recent studies have shown that human patients with head trauma and hyperglycemia have a *poorer* return to cognitive function than do euglycemic patients. Why is hyperglycemia dangerous in head trauma, or in any case of brain ischemia? Unfortunately, elevated glucose concentrations provide a substrate for anaerobic metabolism and glycolysis in the brain, worsening brain perfusion via the accumulation of the by-product, lactic acid. Hyperglycemia is also associated with proconvulsant effects, which are due to increased neuronal excitability. In a veterinary study by Syring and coworkers, 52 dogs and 70 cats with head trauma were compared with 122 age- and species-matched control dogs and cats (4). Severity of head trauma was classified as mild, moderate, or severe, and blood glucose concentrations were recorded within 1 hour after admission (4). The study found that the blood glucose concentrations were significantly associated with *severity* of head trauma in dogs and cats and were significantly higher in dogs and cats with head trauma than in the control animals.⁴ However, blood glucose concentration was not associated with outcome, which is divergent from human studies. This veterinary study may also differ from human medicine in that overall cognitive function varies between humans and dogs/cats. These studies reiterate that iatrogenic hyperglycemia must be avoided in patients with head trauma or cerebral ischemia and that severe hyperglycemia in head trauma should potentially be treated with regular insulin therapy if warranted and persistent. When in doubt, withhold steroid therapy in head trauma patients to prevent hyperglycemia and other detrimental effects. Instead, osmotic agents such as mannitol have been found to be helpful in decreasing intracranial pressure (ICP).

If IV fluid resuscitation alone does not reduce glucose levels in hyperglycemic patients with head trauma, a low dose of regular insulin (0.2 U/kg, intramuscular) may be given every 3 to 4 hours for the first few hours to help lower blood glucose. Blood glucose levels should be monitored frequently to ensure improvement and to preclude hypoglycemia, which would further complicate neurologic monitoring.

Therapies other than steroids to consider in head trauma patients include:

- Aggressive fluid resuscitation to help normalize or maintain blood pressure and maximize perfusion
- Oxygen therapy
- 15- to 30-degree head elevation (to lower ICP)

- Minimal jugular restraint or pressure (to prevent increased ICP)
- Tight glycemic control

NOT STABILIZING A PATIENT MORE AGGRESSIVELY BEFORE SURGERY

Rivers and colleagues (5) found that early goal-directed therapy was imperative in the initial treatment and stabilization of severe sepsis and septic shock. With aggressive IV fluid therapy, blood transfusion, vasopressors, and normalization of perfusion parameters (e.g., UOP, base excess, lactate, central venous oxygen saturation, central venous pressure), mortality was reduced from 46% to 30% (5). Likewise, when we are presented with a patient in shock (e.g., hemoabdomen, septic peritonitis, uroabdomen), it is imperative to aggressively stabilize the patient before anesthesia and surgery. By adequately perfusing the patient before anesthesia (e.g., with IV crystalloids, colloids, blood products), we are able to maximize cardiac output, improve heart rate, and increase overall mean arterial pressure and, indirectly, cellular perfusion. Inhalant agents, propofol, preanesthetic medications, and analgesics can result in bradycardia and hypotension; these agents also blunt the body's response to poor cardiac output and mask the underlying "shocky" state of the patient.

HAVING A NEGATIVE LAPAROTOMY

Nobody likes a negative laparotomy—not the veterinarian, the patient, or the pet owner. Aggressive diagnostics should be done to evaluate the necessity of surgery. The use of serial radiographs; advanced diagnostics (such as barium series, fluoroscopy, and abdominal ultrasonography); repeat physical examination; and venous blood gas evaluation are all necessary to prevent a negative exploratory procedure. Serial radiographs (e.g., 8-12 hours after initial presentation) are often beneficial after aggressive IV fluid therapy has been initiated to evaluate changes in gas patterns. When performing a barium series, keep in mind that a common mistake is not using enough barium, resulting in poor imaging and interpretation. Delivery of barium through a stomach tube is an effective method to ensure adequate delivery of volume (barium dose: 3–6 ml/lb for a cat or dog).

Venous Blood Gas Analysis

Venous blood gas measurement is a helpful way to rule out a pyloric outflow obstruction. In veterinary medicine, the presence of a metabolic alkalosis is typically only due to two primary differential diagnoses: pyloric outflow obstruction and loop diuretic administration. This is due to underlying hypochloremia, either from gastric acid vomiting or chloride loss through the kidney (due to the effects of using a Na-K-2Cl loop diuretic). Because chloride is an anion, it must be paired with a cation (Na^+) to maintain electroneutrality. When the body is depleted of chloride, sodium absorbs bicarbonate (HCO_3^-) instead to maintain electroneutrality, resulting in metabolic alkalosis. The absence of metabolic alkalosis does not exclude a foreign body obstruction further distal to the pylorus, but the presence of metabolic alkalosis should make the clinician highly suspicious of an upper GI outflow obstruction.

If in the event of a negative exploratory, it is imperative to obtain appropriate biopsies from relevant or diseased organs (e.g., liver, stomach, small intestine, pancreas) as deemed by medical necessity. In cases of acute pancreatitis, aggressive lavage of the abdominal cavity is beneficial to dilute activated pancreatic zymogens and to reduce the severity of inflammation. Lastly, feeding-tube placement (e.g., duodenal) may be beneficial at the time of surgery (particularly for pancreatitis cases), bypassing the stomach and allowing for enteral support.

NOT ASSESSING YOUR PATIENT MORE FREQUENTLY WITH SIMPLE TESTS

In veterinary medicine, the temperature, pulse rate, respiratory rate, and weight are typically evaluated during the initial presentation. These simple, inexpensive physical examination parameters are an

important part of serial assessment and often provide clues on hydration status, disease process, and response to treatment.

Temperature

When examining a hyper- or hypothermic patient, differentiate between exogenous and endogenous sources. Hyperthermia is typically caused by an exogenous heat source (e.g., sun exposure, humidity, locked inside a car, upper airway obstruction resulting in lack of ability to thermoregulate). This is semantically different from fever, which is caused by an endogenous heat source (e.g., neoplasia, inflammatory cytokines). With hyperthermia, patients should be cooled by using cold water baths, cold IV fluids, fans, and relieving the upper airway obstruction. Patients should only be cooled to 103.5°F (39.7°C) to prevent severe rebound hypothermia. The use of “fever-breaking” medications (e.g., dipyrone) is not indicated, as resetting of the hypothalamus may have already occurred.

Patients with fever should not undergo cooling methods, as the fever is a physiologic response to an underlying pathology (e.g., viruses, bacteria). Three key differential diagnostics should be considered with fever:

- Infection
- Inflammation
- Neoplasia

For hypothermia, it is important to determine whether it is due to an exogenous source (e.g., living in a cold environment with inadequate shelter, hair coat, or underlying hypothyroidism) or an endogenous one. Hypothermic patients should be warmed passively (e.g., blankets, concurrent warm IV fluids) and *slowly*. With hypothermic patients, it is important not to rapidly warm patients via surface warming alone (e.g., BAIR hugger), particularly if they are hypotensive, as rewarming can result in peripheral vasodilation. During states of poor perfusion or hypotension, patients should physiologically vasoconstrict peripheral blood flow to direct blood to more important organs—the heart and lungs. Rapid surface rewarming of hypothermic patients without adequate IV fluid replacement can result in inappropriate shunting of blood.

Weight

Weight is often underutilized as a means of assessing hydration. Because we can calculate dehydration (kg weight X % dehydration), we can also estimate appropriate weight gain, as 1 liter = 1 kg. Patients should be weighed daily while hospitalized, ideally on the same scale. This is important because it is an easy way to evaluate hydration and appropriate (or inappropriate) weight gain. For patients in which volume–fluid balance is tenuous (e.g., acute renal failure with anuria, congestive heart failure, volume overload), weight should be evaluated every 6 to 8 hours.

For example, if you determine that a 30 kg dog is 10% dehydrated, the amount of fluid required to hydrate him is:

Calculated dehydration: $30 \text{ kg} \times 0.1 \text{ (percent dehydration)} \times 1000 \text{ mls} = 3000 \text{ ml} = 3 \text{ L}$

In other words, a 30 kg dog needs 3L of IV crystalloids for rehydration alone and thus should weigh 33 kg after hydration (in 8–12 hours, depending on patient stability). If that same dog weighs 32 kg by the next day, he may still be inadequately hydrated. Likewise, if the patient weighs 34.8 kg the next day, he may be overhydrated, volume overloaded, and retaining water inappropriately (e.g., acute renal failure).

Pulse Quality

Assessing pulse quality frequently is imperative in unstable, shocky emergency patients. Palpating the femoral pulse enables assessment of pulse quality, which is the difference between the systolic and diastolic pressures. Pulse palpation, quality, and duration are a gross estimate of blood pressure and, indirectly, stroke volume. In a normal healthy animal, the pulses should be strong and synchronous, with a palpable pulse for each heart beat (therefore, make sure that you are simultaneously auscultating your patient and palpating for femoral pulses). A palpable femoral pulse is consistent with systolic blood pressure of at least 60 mm Hg. Poor femoral pulses typically indicate profound hypotension and should be treated aggressively and appropriately. A palpable dorsal metatarsal pulse is consistent with a systolic blood pressure of at least 90 mm Hg, and can be used as a basic “poor man’s Dinamap,” particularly during volume resuscitation.

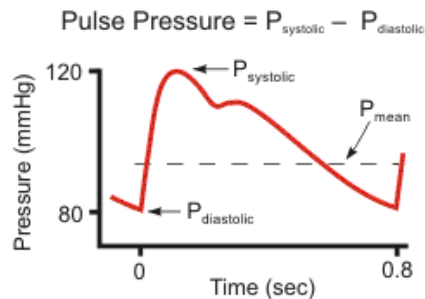


Figure 1. Pulse pressure. Image courtesy of Klabunde RE, www.cvphysiology.com, 2010.

When palpating the femoral pulse of a patient, one can determine pulse quality based on the duration, width, and strength. A patient’s pulse is normal if it has a normal waveform duration (Figure 2). A thready pulse indicates a narrow waveform, whereas a weak pulse refers to a small-amplitude pulse-pressure difference (Figure 3). Either of these may be indicative of decreased stroke volume. Thready pulses are often associated with peripheral vasoconstriction and may indicate low diastolic pressure. Thready pulses are consistent with volume depletion. Bounding pulses have a large pulse-pressure difference and a wide waveform, usually associated with increased cardiac output and vasodilatation (Figure 4). However, bounding pulses may also result from a larger difference between systolic and diastolic blood pressures. This is classically seen in cases of chronic anemia (e.g., immune-mediated hemolytic anemia). These patients adapt to the anemia and thus are able to maintain normal systolic function, but their blood vessels may be “empty”; hence, diastolic pressure is often low. This large net difference usually results in bounding pulses.

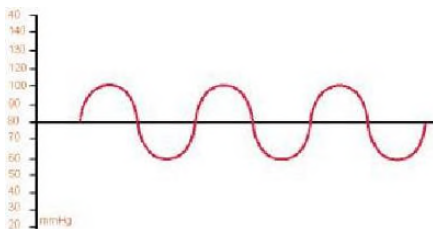


Figure 2. Normal pulse. Image courtesy of Klabunde RE, www.cvphysiology.com, 2010.

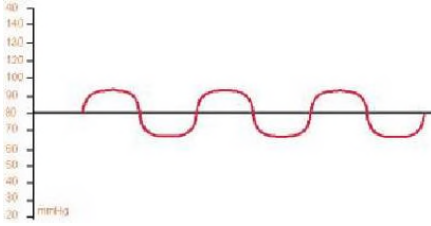


Figure 3. Weak pulse. Image courtesy of Klabunde RE, www.cvphysiology.com, 2010.

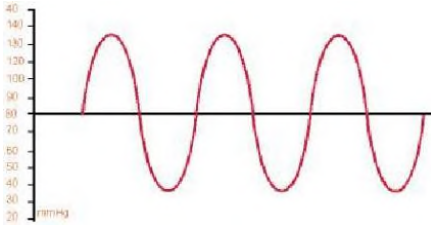


Figure 4. Bounding pulse. Image courtesy of Klabunde RE, www.cvphysiology.com, 2010.

Patients with systolic blood pressure < 90 mm Hg should be treated with IV fluids (if hypovolemic) and vasopressors (once adequately volume resuscitated) if evidence of shock (e.g., hypovolemic, septic, hyperdynamic, hemorrhagic) is present (provided cardiovascular shock has been ruled out as a differential). Patients with systolic blood pressure > 180 mm Hg (normal, 120 mm Hg) should be treated with antihypertensives, such as hydralazine, nitroprusside, amlodipine or enalapril, to minimize secondary complications from hypertension, such as detached retinas, cardiovascular and renal effects, and ischemic events. Frequent monitoring of blood pressure is imperative to ensure adequate care.

Serial physical examination is imperative to adequately evaluate a patient's hydration status—checking for return of skin turgor, appropriate weight gain, and moisture of mucous membranes. However, physical examination findings are subjective, and <5% dehydration is subjective and difficult to assess on physical examination. The concurrent use of evaluation of PCV/TS, blood glucose, blood urea nitrogen (BUN or AZO) weight, UOP, and urine specific gravity (USG), and thirst can be used in conjunction with physical examination findings to better assess hydration status.

Packed Cell Volume/Total Solids, Blood Glucose, and Blood, Urea, Nitrogen (BUN/AZO) (“The Big 4”)

Patients on IV fluids should have a minimum database (including PCV/TS and blood glucose) measured daily, along with basic electrolytes to make sure Na^+ and K^+ are normal. Because patients often experience hemoconcentration when they are dehydrated (e.g., PCV/TS 55%/7.8 g/dl [78 g/L]), the goal of fluid therapy is to ensure that these numbers improve with appropriate therapy (consistent with hemodilution). Ideally, the PCV/TS in a normal, systemically healthy patient on IV fluids at sea level should be 35%/5.0 g/dl (50 g/L). In fact, oxygen delivery is maximal at such a “hemodilute” PCV/TS, as there is less viscosity of red blood cells and “sludginess.” We can still evaluate the PCV/TS in abnormal, metabolically inappropriate patients. Classically, a 10% to 12% dehydrated, cachectic, geriatric cat with chronic renal failure may present to you with a PCV/TS of 28%/11 g/dl (110 g/L). Once that patient is adequately hydrated, the PCV/TS may decrease to 20%/7 g/dl (70 g/L), unmasking the anemia from lack of erythropoietin.

Urine Specific Gravity (USG)

USG can be evaluated in patients on IV fluids to help assess hydration status. Ideally, USG should be measured before fluid administration to allow for evaluation of renal function. Dehydrated patients with concentrated urine demonstrate adequate renal function (cat > 1.040, dog > 1.025)—in other words, the kidneys are working and trying to absorb as much water from the urine as possible. Once started on IV fluids, normal, systemically healthy patients should have isosthenuric urine. Patients on IV fluids for > 6 to 12 hours should have adequate dilution of USG, and the ultimate goal of fluid therapy and adequate hydration should be USG of 1.015 to 1.018 on IV fluids. Patients on IV fluids with USG > 1.020 are still likely dehydrated and should be treated more aggressively with IV fluids if other parameters of dehydration persist (e.g., hemoconcentration). Hydration can be determined by assessing the color, volume, and USG of urine. A patient that is still dehydrated while hospitalized on IV fluids may have decreased UOP and dark-yellow urine (provided, for example, that no pigmentation, myoglobinuria, or bilirubinuria are present). This is a result of antidiuretic hormone release and renin-angiotensin stimulation, resulting in maximum absorption of free water and sodium.

Urine Output (UOP)

UOP should be monitored carefully, particularly in azotemic patients. Fluid therapy should be directed toward achieving normal UOP (i.e., 1–2 ml/kg/hour). Again, one can assess the hydration status of the patient by evaluating the volume and USG of urine. Excessive urination with dilute, clear urine may indicate copious or excessive IV fluid therapy, whereas hypersenturia may suggest ongoing dehydration, and aggressive fluid resuscitation may be further warranted. If UOP is decreased (particularly in azotemic patients), fluid therapy and vasopressor support (to increase renal blood flow) should be initiated to prevent anuria (< 0.5 ml/kg/hour) or oliguria (< 1 ml/kg/hour). If UOP is decreasing and renal function is normal (based on creatinine, BUN, and pre-fluid therapy USG), the patient should be reassessed for hydration status, and fluid therapy adjusted as indicated. Classically, a cat with urethral obstruction may have a profound postobstructive diuresis. A sudden decrease in UOP should elicit assessment for reobstruction. If no obstruction is found, USG should be remeasured. If hypersenturia is found (>1.025 on IV fluids), decreased UOP is likely due to continued dehydration from a postobstructive diuresis—the patient is attempting to absorb as much free water as possible from the kidneys, resulting in decreased UOP. In this example, the IV fluid rate should be increased.

- Normal UOP: 1–2 ml/kg/hour
- Oliguria: 0.5–1 ml/kg/hour
- Anuria: < 0.5 ml/kg/hour

Note that underlying diseases such as postobstructive diuresis (posturethral obstruction); diabetes mellitus (with secondary osmotic diuresis due to glucosuria); hyperthyroidism (increased glomerular filtration rate due to increased metabolic rate); and chronic renal failure (inability to adequately concentrate and absorb water) may result in dramatic water losses through the kidneys, and these patients may need a higher rate of fluids to compensate for ongoing losses. Likewise, these disease processes prevent us from differentiating renal versus prerenal disease on the basis of USG alone, as these patients have isosthenuria due to metabolic disease. Regardless, appropriate fluid therapy and urine monitoring (e.g., “measuring ins and outs”) may be necessary, particularly in azotemic, oliguric renal failure.

A Water Bowl

Any hospitalized animal should always have access to fresh, clean water unless it is contraindicated due to vomiting, pancreatitis, fasting for anesthesia or sedation, or to maximize mannitol or furosemide

effects (fasted for 20 minutes only). If a hospitalized patient on IV fluids continues to drink water in front of you, you should be concerned that the patient is still dehydrated. Due to the timidity of cats, they often will not drink water when stressed and hospitalized. If a dog or cat drinks in your presence, that patient is probably still dehydrated, and their thirst mechanism continues to be stimulated in an attempt to hydrate. Take that as a hint that your patient is trying to tell you to increase the fluid rate! Rare situations when hydration status cannot be based on the thirst mechanism include diabetes insipidus and psychogenic polydipsia.

NOT USING ENOUGH SQ FLUIDS

We often use SQ fluids in veterinary outpatient medicine to help hydrate a patient. Because fluids are so slowly absorbed when given in this manner, SQ administration is not appropriate for hypovolemic or severely dehydrated patients. SQ fluids are ideally utilized for outpatient medicine (e.g., the vomiting patient that needs to be fasted overnight but still needs to maintain hydration). But just how much fluid can you give SQ? The calculation for how many ml/kg to give SQ is typically *maintenance fluids*. We do not adjust for dehydration or ongoing losses with SQ fluids.

Example:

5-kg, male castrated cat presents for 4 episodes of vomiting

Physical examination: no string on oral examination, nonpainful abdomen

Amount of SQ fluids to potentially give: $5 \text{ kg} \times 60 \text{ ml/kg/day} = 300 \text{ ml SQ}$

40 kg, female spayed Labrador presents for 3 vomiting episodes in 12 hours after ingesting garbage

Physical examination: nonpainful abdomen; abdominal radiographs: no significant findings, no obstruction, but some fluid-filled loops of intestine

Amount to give: $40 \text{ kg} \times 50 \text{ ml/kg/day} = 2000 \text{ ml SQ}$

Giving too small of an amount of SQ fluids often does not benefit the patient. Having owners give < 50 ml/adult cat for SQ fluids is often not aggressive enough (not worth the needle poke!). That said, if a patient has a heart murmur (particularly in cats), this maintenance amount should be *reduced* to prevent volume overload.

Clinical application

- If you're giving SQ fluids, take the time to calculate maintenance, so you know the exact amount to give.
- The most common error with SQ fluid administration in dogs is giving too little.
- Caution must be used in cats—even SQ fluid administration can result in volume overload.

NOT DOING ENOUGH FAST (FOCUSED ASSESSMENT WITH SONOGRAPHY FOR TRAUMA) ULTRASOUNDS

The focused assessment with sonography for trauma (FAST) ultrasound is a 2-minute procedure that detects the presence of fluid in the abdominal cavity to allow for rapid therapeutic intervention (e.g., fluid resuscitation, abdominocentesis, cytology, clinicopathologic testing) (6). This has also been modified for the pleural (T-FAST) and pericardial space. This rapid method of ultrasound is designed to be used by health care professionals with limited ultrasonographic training and is not designed for extensive examination of the abdomen. The added benefit of the FAST examination is the ability to detect very small amounts of fluid. Typically, 5 to 25 ml/kg of fluid needs to be present to be removed by blind abdominocentesis; > 10 to 20 ml/kg of fluid has to be present before it can be detected by fluid-wave assessment on physical examination; and approximately 8.8 ml/kg of fluid needs to be present

before it can be detected radiographically. On the contrary, as little as 2 ml/kg of fluid can be detected on a FAST examination, allowing for rapid diagnosis and identification of underlying pathology.

The FAST examination typically involves assessment of 4 sites of the abdomen: caudal to the xiphoid, cranial to the bladder, and the right and left dependent flank (6). The presence of fluid at any of the sites is considered positive. Evaluation of the xiphoid region allows you to check for fluid between the liver and diaphragm and the liver lobes, as well as for pericardial or pleural effusion (6). Evaluation of the bladder view evaluates for fluid cranial to the bladder and for the presence of a bladder (6). The right dependent flank allows for fluid detection between the intestines and the body wall, whereas the left dependent flank view allows for identification of the spleen, abdominal effusion near the spleen and body wall, the kidney and spleen, and the liver and spleen (6).

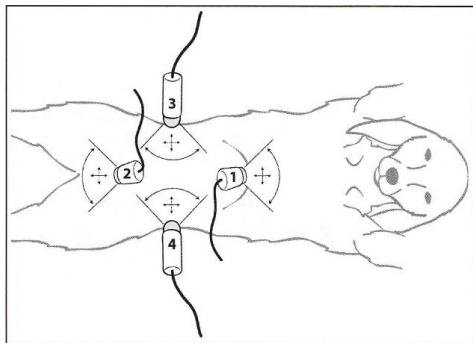


Figure 5. Illustration of the probe placements and movements used to obtain ultrasonographic views of the abdomen via FAST in a dog. Figure courtesy of Boysen SR from IVECCS proceedings 2006.

RELUCTANCE TO PENETRATE BODY CAVITIES

The use of abdominocentesis or thoracocentesis is a benign procedure that is both diagnostic and therapeutic. Referring a stressed, hypoxic, frantic, dyspneic cat with 300 ml of pleural effusion for a 1-hour car ride to a specialist can easily result in the cat's demise. Shaving and surgically preparing a wide area near the umbilicus (abdominocentesis) or thorax (thoracocentesis) should be done quickly but aseptically. For the thorax, thoracocentesis should be performed either dorsally (for air) or ventrally (for effusion) at the 7th to 9th intercostal space (ICS). Likewise, an imaginary line can be drawn from the end of the xiphoid to the lateral body wall, which is approximately the 8th ICS. This will allow for rapid identification of where to perform an emergency thoracocentesis. Pericardiocentesis should be performed on the right side at the region of the 3rd to 5th ICS at the point of the flexed elbow. Abominocentesis should be aseptically performed via a four-quadrant tap in the periumbilical region. The use of a 3-way stopcock, 20- to 60-ml syringe, extension tubing, and appropriately sized needles dependent on patient size and volume of effusate (usually 20–22 gauge for cats and 16–22 gauge for dogs) is indicated.

CONCLUSION

Veterinarians should avoid these key, common mistakes in emergency medicine. By avoiding these errors, the overall quality of care and survival of the emergency patient may improve. Simple, easy monitoring tools (e.g., Big 4, pulse quality, weight) can be used to more carefully monitor our critically ill patients in a cost-effective, simple, repeatable manner.

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NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb's Veterinary Drug Handbook*.

TOP 20 POISONS AFFECTING DOGS & CATS

Justine A. Lee, DVM, DACVECC, DABT

CEO, VETgirl

justine@vetgirlontherun.com

<http://www.vetgirlontherun.com>

Saint Paul, MN

INTRODUCTION

Each year, the [ASPCA Animal Poison Control Center](#) (APCC) manages hundreds of thousands of poisoning calls. At the ASPCA APCC, an estimated 50% of pet poisonings comprise human over-the-counter (OTC) and prescription medications. In this lecture, we will review the mechanism of toxicosis, clinical signs, and overall treatment of the top 10 most common poisons affecting dogs and cats.

In the veterinary poisoned patient, the goal of decontamination is to “inhibit or minimize further toxicant absorption and to promote excretion or elimination of the toxicant from the body.”^{1,2} When treating the poisoned patient, the clinician should have an understanding of the toxic dose (if available), the pharmacokinetics (including absorption, distribution, metabolism, and excretion), the underlying mechanism of action, and the potential clinical signs that can be observed with the toxicant.² This will help determine appropriate decontamination and therapy for the patient. If this information is not readily available, the reader is advised to contact the [ASPCA Animal Poison Control Center](#) (888-426-4435) for life saving, 24/7 advice as needed. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

CALCIUM CHANNEL BLOCKERS, BETA-BLOCKERS, ACE-INHIBITORS, STATINS AND DIURETICS

Certain cardiac medications include broad categories such as calcium channel blockers (CCB), beta-blockers (BB), and angiotensin-converting enzyme (or “ACE”) inhibitors. These medications are commonly used in both human and veterinary medicine to treat underlying cardiac disease or hypertension. Each category of cardiac medication has different margins of safety. CCB and BB toxicosis should be treated aggressively, as these two categories of medications have a narrow margin of safety. Toxicosis of these agents can result in myocardial failure, severe bradycardia, and hypotension; untreated, cardiac output becomes reduced, and secondary severe hypoperfusion and acute kidney injury (AKI) can potentially develop.³⁻⁵ With ACE-inhibitors, severe overdoses can cause hypotension, dizziness, weakness, and hypotension. In general, there is a wider margin of safety with ACE-inhibitors, which are typically considered much safer. Pets ingesting small amounts of ACE-inhibitors can potentially be monitored at home, unless they have underlying disease (e.g., kidney failure, cardiac disease, etc.). With ACE-inhibitors, ingestions > 10-20X a therapeutic dose are generally considered toxic, and can result in severe clinical symptoms (e.g., hypotension).⁵ Treatment for any cardiac medication includes decontamination (e.g., emesis induction, gastric lavage, activated charcoal (AC) administration), blood pressure monitoring, aggressive IV fluid therapy if hypotension is detected, and blood work monitoring. With severe toxicosis, the use of high-dose insulin therapy or intravenous lipid emulsion may be warranted as a potential antidote for calcium channel blocker toxicosis.³

SELECTIVE SEROTONIN RE-UPTAKE INHIBITORS (SSRI)

Selective serotonin re-uptake inhibitors (SSRIs) are a class of medications that are commonly used in human medicine for depression. Common examples include the following drugs:

- Fluoxetine (Prozac® in human beings; Reconcile™ in veterinary medicine)

- Citalopram (Celexa[®])
- Paroxetine (Paxil[®])
- Sertraline (Zoloft[®])

Other similar drugs include selective norepinephrine re-uptake inhibitors (SNRIs), which include common drugs like duloxetine (Cymbalta[®]), nefazodone (Serzone[®]), and venlafaxine (Effexor[®]). SNRI and SSRI drugs result in similar clinical signs of toxicosis, and therefore are treated the same. In veterinary medicine, SSRIs are used for a wide array of behavioral problems, including feline urine spraying, canine separation anxiety, lick granulomas, etc. These SSRI drugs work by blocking the reuptake of serotonin in the pre-synapse, thereby increasing the levels of serotonin in the pre-synaptic membrane. In small animal patients, common clinical signs from SSRIs include the following:

- Sedation or central nervous system (CNS) stimulation
- Anorexia
- Lethargy
- Serotonin syndrome

Clinical signs of serotonin syndrome include: gastrointestinal (GI) signs (e.g., hypersalivation, vomiting, diarrhea, abdominal pain) and CNS signs (e.g., stimulation, mydriasis, tremors, seizures, hyperthermia secondary to tremoring and seizing). Treatment for antidepressants includes decontamination (ideally done at a veterinarian, due to the rapid onset of clinical signs), sedation (e.g., with acepromazine or chlorpromazine), intravenous (IV) fluid therapy, blood pressure and electrocardiogram (ECG) monitoring, thermoregulation, muscle relaxants (for tremors; methocarbamol 22-55 mg/kg, IV, PRN), anticonvulsants (e.g., phenobarbital 4-16 mg/kg, IV, PRN; diazepam 0.25-0.5 mg/kg, IV, PRN), serotonin antagonists [e.g., cyproheptadine (1.1 mg/kg for dogs or 2-4 mg *total* per cat) PO or rectally q. 6-8], and supportive and symptomatic care. In general, the prognosis for antidepressant toxicosis is excellent.

AMPHETAMINES

Amphetamines are used for a variety of medical and illicit reasons. Legal forms include prescription medications for attention-deficit disorder/attention deficit-hyperactivity disorder (ADD/ADHD), weight loss, and narcolepsy. Examples of amphetamines include:

- Dextroamphetamine
- Amphetamine (Adderall[®])
- D-amphetamine (Dexedrine[®])
- Methamphetamine (Desoxyn[®])
- Lisdexamfetamine (Vyvanse[®])

Illegal forms of amphetamines include street drugs like methamphetamine, crystal meth, and ecstasy. This class of drugs acts as sympathomimetic agents, meaning they stimulate the sympathetic system. Amphetamines also cause stimulation of α and β -adrenergic receptors, and stimulate release of serotonin and norepinephrine; this results in increased catecholamine stimulation in the synapse. Amphetamines also increase release of serotonin from the presynaptic membrane, resulting in serotonin syndrome. With amphetamine toxicosis, secondary stimulation of certain body systems can result in significant clinical signs: GI (e.g., vomiting, diarrhea, hypersalivating), CNS (e.g., agitation, mydriasis, tremors, seizures), cardiovascular (e.g., tachycardia, hypertension), and respiratory (e.g., panting). Both clinical signs and treatment for amphetamine toxicosis are similar to SSRI toxicosis, and

include IV fluids, cooling measures, sedation (e.g., with acepromazine or chlorpromazine), muscle relaxants, anticonvulsants, thermoregulation, blood pressure monitoring, and symptomatic/supportive care.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

NSAIDs are competitive inhibitors of prostaglandin synthesis (cyclooxygenase or “COX” inhibitors) and result in decreased prostaglandin, which is important for normal homeostatic function (including maintaining renal blood flow, maintaining mucous production in the stomach, etc.). Common OTC human NSAIDs include active ingredients such as ibuprofen and naproxen sodium. Examples of human NSAIDs include Advil[®], Aleve[®], certain types of Motrin[®], etc. Common prescription veterinary NSAIDs can also result in toxicosis, particularly when available in the chewable, palatable formulation. Examples of veterinary NSAIDs include carprofen, deracoxib, etogesic, previcoxib, etc. With NSAID toxicosis, the GI tract, kidneys, CNS, and platelets can be affected. Cats and certain breeds of dogs (e.g., German shepherds) seem to be more sensitive to NSAIDs, and should be treated aggressively. With cats, severe acute kidney injury (AKI) is often more clinically seen with NSAID toxicosis at lower doses (as compared to dogs). With dogs, signs secondary to GI ulceration (e.g., vomiting, diarrhea, melena, hematemesis, etc.) are more commonly seen initially, followed by secondary AKI.

With NSAID toxicosis, it is important to keep in mind that each NSAID has a different toxic dose, margin of safety, half-life, and route of excretion, and an animal poison control should be contacted to identify what specific NSAID and toxic dose was ingested. For example, in dogs, ibuprofen results in GI signs at doses as low as 16-50 mg/kg, while severe GI signs may be seen at 50-100 mg/kg.⁶ Renal compromise may be seen at doses of 100-250 mg/kg (resulting in potential AKI), and fatalities have been reported at doses > 300 mg/kg.⁶ This differs tremendously from naproxen sodium (dogs), where severe clinical signs can be seen at doses as low as 5 mg/kg.⁶

Clinical signs of NSAID toxicosis include anorexia, vomiting, hematemesis, diarrhea, melena, abdominal pain, lethargy, malaise, uremic halitosis, dehydration, etc. Treatment includes decontamination, the use of activated charcoal (often multiple doses due to enterohepatic recirculation, if appropriate), GI protectants (e.g., H₂ blockers, sucralfate), aggressive IV fluid therapy (to help maintain renal blood flow), anti-emetic therapy, and symptomatic and supportive care. With high doses, anti-convulsants may also be necessary if CNS signs develop.

ACETAMINOPHEN

Acetaminophen (N-acetyl-p-aminophenol), a cyclooxygenase (COX)-3 inhibitor, is a popular OTC analgesic and antipyretic medication used frequently in humans. It is not considered a true NSAID as it lacks anti-inflammatory properties. Normally, part of this drug is metabolized into non-toxic conjugates via the metabolic pathways (glucuronidation and sulfation);⁷ some is metabolized into the toxic metabolite, N-acetyl-para-benzoquinoneimine [NAPQI] via the cytochrome P-450 enzyme pathway.⁷ Typically, NAPQI is detoxified by conjugation with glutathione in the liver.⁷ Toxicosis occurs when glucuronidation and sulfation pathways are depleted; this results in toxic metabolites building up and secondary oxidative injury occurring.⁷

While this drug is very safe for human use, it has a narrow margin of safety in dogs and cats; the severity of toxicosis and development of clinical signs is species-dependent. Cats have an altered glucuronidation pathway and a decreased ability to metabolize acetaminophen, making them much more susceptible to toxicosis. In cats, red blood cell (RBC) injury is more likely to occur in the form of methemoglobinemia (methHb), and toxicity can develop at doses as low as 10 mg/kg.⁷ In cats, lethargy, swelling of the face or

paws, respiratory distress, brown mucous membranes, cyanosis, vomiting, and anorexia may be seen secondary to metHb. In dogs, hepatic injury is more likely to occur; acetaminophen toxicosis can occur at doses > 100 mg/kg, while metHb can develop at doses of > 200 mg/kg.⁷ Dogs may develop clinical signs of keratoconjunctivitis sicca (dry eye), malaise, anorexia, hepatic encephalopathy, vomiting, melena, and icterus secondary to hepatotoxicity.

Treatment includes decontamination, administration of activated charcoal (AC), anti-emetic therapy, IV fluid therapy, treatment for hypoxemia (e.g., oxygen, blood transfusion, etc.), antioxidant therapy (e.g., Vitamin C), provision of a glutathione source (S-adenosyl-methionine or SAME), and the antidote n-acetylcysteine (NAC, ideally IV) to limit formation of the toxic metabolite NAPQI by providing additional glutathione substrate.⁷ Baseline blood work and follow-up biochemical panels should be performed to monitor for the presence of metHb, Heinz body anemia, or evidence of hepatotoxicity. Generally, prognosis is fair to excellent with therapy. If clinical signs resolve and liver enzymes are within normal limits after 48 hours of NAC therapy, patients can be discharged with SAME (for 30 days). Those with severe hepatic failure have a poorer prognosis.

PYRETHRINS AND PYRETHROIDS

Pyrethrins and their synthetic derivative, pyrethroids, are commonly found in household insect sprays and insecticides (e.g., permethrin, cypermethrin, cyphenothrin, etc.). Due to a cat's altered liver glucuronidation metabolism, cats are significantly more sensitive to pyrethrins than dogs. While a precise toxic dose for cats is not well established, products containing greater than a 5-10% concentration of pyrethrins may lead to systemic toxicosis. The diluted amount found in household insect sprays and topical flea sprays and shampoos is typically < 1%. Toxicosis from exposure to these products is highly unlikely. The application of canine spot-on pyrethrin/pyrethroid based insecticides (typically ~40-50% concentration) to cats is the primary cause of feline pyrethrin toxicosis. Cats that groom dogs following recent spot-on applications are also at high risk for toxicosis; ideally, pets should be separated until the spot-on product has completely dried on the dog to prevent cat exposure. Signs of systemic toxicosis in cats include GI signs (e.g., hypersalivation, vomiting, nausea), neurologic signs (e.g., disorientation, weakness, hyperexcitability, tremors, seizures) and respiratory signs (e.g., tachypnea, dyspnea). Tremors are extremely responsive to methocarbamol (22-220 mg/kg, IV PRN to effect), a centrally acting muscle relaxant, although oral absorption of methocarbamol is often slower in onset of action. In general, tremors are less responsive to benzodiazepines (e.g., diazepam). Seizures may be controlled with Phenobarbital (e.g., 4-16 mg/kg, IV PRN to effect) or general gas anesthesia. Dermal decontamination is crucial but should be performed after stabilization. This should be performed with a liquid dish detergent (e.g., Dawn, Palmolive). Supportive care including the monitoring and maintenance of hydration, body temperature and blood glucose levels are necessary. Signs may persist for 1-4 days, depending on the animal. The prognosis is excellent with aggressive dermal decontamination and treatment.

INSECT BAIT STATIONS

Household ant and roach bait stations are rarely toxic, as the active ingredient is often a low-concentration of abamectin (a macrocyclic lactone derivative in the same family as ivermectin). Certain breeds with the MDR-1 gene mutation (now known as the ABCB1-1 Δ polymorphism), including collies, Border collies, old English sheepdogs, and collie-mixed breed dogs, may be more at risk when large amounts of bait stations are ingested. Typically, the plastic on the bait station is more of a problem, as it can result in GI signs or potentially foreign body obstruction (FBO), when ingested in large amounts.

FIRE STARTER LOGS

Fire starter logs typically do not pose a “toxicosis” risk, but rather a FBO risk. Most types (e.g., Duraflame®) are made of compressed sawdust and wax, and do not break down readily in the stomach, resulting in a FBO. Rarer types of fire starter logs may contain heavy metals to provide a “color sparkle” to the fireplace. With recent ingestion, emesis induction should be performed to prevent FBO. If unknown ingestion or prolonged ingestion has occurred, abdominal radiographs should be performed to evaluate for the presence of gastric contents or FBO. If the material has passed out of the stomach, the use of a high-fiber diet, anti-emetic therapy, and careful monitoring (based on clinical signs, radiographic evidence of obstruction, etc.) should be performed. With massive ingestions demonstrating evidence of FBO, surgical intervention may be necessary, albeit rare.

SILICA GEL PACKS & FOOD OXIDIZER PACKS

Silica gel packs, while commonly ingested by pets, rarely result in toxicosis as they have a wide margin of safety (despite their labeling of “Do not eat”). When ingested in large amounts, they can potentially result in FBO; however, this is generally rare. These need to be differentiated from food oxidizer packs which contain small amounts of iron; food oxidizer packs are commonly found in human and pet food products (e.g., box of cookies, cereal, pasta containers, jerky treats, rawhide packs, etc.). The contents of a food oxidizer pack are typically brown or black in color (e.g., pellets, powder) and stick to a magnet; this is consistent with iron. As iron does not bind well to activated charcoal (AC), the use of AC is not indicated. Rather, with iron toxicosis, the use of antacids like milk of magnesia should be considered, which readily bind to heavy metals. In general, treatment is typically not necessary as these packs are very small; however, in a very small patient, outpatient treatment may be warranted as needed.

HYDROCARBONS

Hydrocarbons consist of chemicals containing a hydrogen and carbon group as their main constituents. Examples include liquid fuels such as kerosene, engine oil, tiki-torch fuels, gasoline, diesel fuels, paint solvents, wood stains, wood strippers, liquid lighter fluids, asphalt/roofing tar, etc. These are often referred to as “petroleum distillates” based on their viscosity, carbon chain length, and lipid solubility. It is *contraindicated* to induce emesis with hydrocarbon toxicosis due to the risks of aspiration pneumonia; due to the low viscosity of hydrocarbons, these compounds are more easily aspirated, resulting in respiratory injury and secondary infection. In general, hydrocarbons are GI tract irritants, but can also be irritants to the respiratory system (if inhaled), eyes, and skin also. Clinical signs include vomiting, nausea, tachypnea, and dermal or ophthalmic irritation. Typically, GI tract irritation is self-limiting. Patients should be treated with anti-emetic therapy, possible SQ fluid therapy (to assist in hydration), fasting (no food per os), and initiation onto a bland diet. Patients demonstrating any coughing, retching, or tachypnea post-ingestion should have chest radiographs performed to rule out aspiration pneumonia, of which treatment is supportive (e.g., oxygen therapy, IV fluids, antibiotic therapy, nebulization and coupage, etc.).

ASTHMA INHALERS (e.g., ALBUTEROL)

Asthma inhalers are often used in both human and veterinary medicine. Various types of medications may be used, including steroids (e.g., fluticasone) or beta agonists (e.g., albuterol, salbutamol, etc.). When beta-agonist inhalers are accidentally chewed and punctured by dogs, they can result in a severe, life-threatening, acute toxicosis. (Inhaled steroids are not a large toxicity issue). Because inhalers often contain approximately 200 metered, concentrated doses, a massive amount of beta-agonist is released with just one puncture. Clinical signs include cardiac (e.g., tachycardiac, a “racing heart rate” per the owner, injected gums, hypotension, hypertension, severe arrhythmias), electrolyte changes (e.g., severe hypokalemia, hyperglycemia), GI (e.g., vomiting), and CNS (e.g., mydriasis, agitation, weakness, collapse,

death). Treatment includes stat electrolyte monitoring, IV fluids, potassium supplementation, blood pressure and ECG monitoring, sedation/anxiolytics (if the patient is agitated, hypertensive, and tachycardiac), anti-arrhythmics such as beta-blockers (e.g., propranolol, esmolol, etc.), and symptomatic supportive care. Treatment for 24-36 hours is typically necessary, until clinical signs resolve.

DECONGESTANTS

Cold and flu medications (e.g., “Claritin-D”) often carry decongestants such as pseudoephedrine (PSE) and phenylephrine (PE). The exact mechanism of how these drugs work is unknown but thought to stimulate alpha and beta-adrenergic receptors by releasing norepinephrine. Phenylephrine is typically considered to be less toxic than PSE as it is less bioavailable with oral ingestion. Clinical signs seen with decongestant ingestion include cardiac (e.g., tachycardia, hypertension, reflex bradycardia), CNS (e.g., mydriasis, agitation, trembling, seizures), and various miscellaneous signs (e.g., hyperthermia). With PSE, moderate to severe clinical signs can be seen at 5-6 mg/kg, while death has been reported at 10-12 mg/kg. With phenylephrine, similar clinical signs can be seen, although GI signs such as vomiting are the most common sign observed. Treatment includes decontamination (if appropriate), administration of one dose of charcoal with a cathartic, IV fluid therapy (to enhance urinary elimination), blood pressure monitoring, anti-emetics, sedatives/anxiolytics (e.g., acepromazine), muscle relaxants for tremoring (e.g., methocarbamol 22-100 mg/kg, IV PRN), anticonvulsants (e.g., phenobarbital 4-6 mg/kg, IV, PRN), and rarely, anti-hypertensives (e.g., hydralazine).

HOUSEHOLD CLEANERS

Most surface cleaners are generally benign, and when ingested directly from the bottle, can result in minor GI signs. However, certain concentrated cleaners can be highly toxic or corrosive. Household bleach is a GI irritant, but “ultra” bleach can be corrosive, resulting in severe esophageal or upper GI damage. Concentrated lye products, toilet bowl cleaners, and oven cleaners are also corrosive, and immediate flushing out the mouth for 10-15 minutes should be performed prior to veterinary visit to minimize tissue injury. Appropriate pet-proofing (such as keeping toilet seats down or securing cleaners in a locked or elevated bathroom cabinet) are the easiest way to prevent this specific toxicosis.

GRAPES, RAISINS, AND CURRANTS

Grapes and raisins (*Vitis* spp) have been recently associated with development of acute renal failure (ARF) with ingestion. All types have been implemented with toxicosis, including organic grapes, commercial grapes, homegrown grapes, and seedless or seeded grapes. While the mechanism of toxicosis is unknown, there are several suspected hypotheses, including individual inability to metabolize certain components of the fruit (e.g., tannins, high monosaccharide content),⁸ the presence of mycotoxins or pesticide residues on the fruit,⁸ or salicylate-like chemicals within the grape or raisin. Common kitchen items also contain grapes, raisins, or currants in their active ingredient, including raisin bread, trail mix, chocolate-covered raisins, cereal with raisins, etc. Currently, grapeseed extract has not been associated with nephrotoxicity.⁸ Treatment for grape and raisin ingestion includes aggressive decontamination as the first-line of therapy. Grapes and raisins seem to stay in the stomach for a prolonged period of time, and are not rapidly broken down or absorbed from the gastrointestinal (GI) tract; hence, delayed emesis induction even several hours post-ingestion can still be initiated to maximize decontamination methods. One dose of activated charcoal can also be administered to prevent absorption of the unknown nephrotoxin. In general, all ingestions should be treated as potentially idiosyncratic and be appropriately decontaminated and treated. Initially, vomiting may be observed within the first 24 hours of ingestion.⁸ Within the next 12-24 hours, clinical signs of lethargy, dehydration, vomiting, diarrhea, anorexia, abdominal pain, uremic breath, and diarrhea may be seen.⁸ Azotemia may develop within 24 hours, with hypercalcemia and hyperphosphatemia occurring first.⁸

Oliguria and anuria may develop 48-72 hours post-ingestion,⁸ at which point the prognosis is poorer. Treatment includes decontamination, aggressive intravenous (IV) fluid therapy, anti-emetics, blood pressure and urine output monitoring, and serial blood work monitoring (q. 12-24 hours for several days). In severe cases, hemodialysis or peritoneal dialysis may be necessary. Asymptomatic patients that have been adequately decontaminated and survive to discharge should have a renal panel and electrolytes monitored 48-72 hours post-ingestion. Overall, the prognosis varies from good to poor, depending on time to decontamination, response to therapy, and prevalence of oliguria or anuria. While 50% of dogs that ingest grapes and raisins never develop clinical signs or azotemia, aggressive treatment is still warranted.⁸

XYLITOL

Xylitol is a natural sweetener found in small quantities in certain fruit. Xylitol has gained recent popularity because it is sugar-free, and is often found in diabetic snacks, foods, baked foods, mouthwashes, toothpastes, chewing gum, mints, candies, and chewable multivitamins.⁹ Sugarless products, particularly those with xylitol listed within the first 3 to 5 active ingredients (AI), can result in severe toxicosis within 15-30 minutes of ingestion. Ingestion of xylitol results in an insulin spike in non-primate species, resulting in severe hypoglycemia. Many pieces of candy and gum (e.g., Orbit™, Trident™, Ice Breakers™) contain various amounts of xylitol ranging, on average, from 2 mgs to 1.0 grams/piece. Unfortunately, not all sources are disclosed by the company (e.g., how many grams of xylitol may be in each piece of gum) due to a proprietary nature. With xylitol toxicosis, it is imperative to calculate whether a toxic dose has been ingested. Doses > 0.1 g/kg are considered toxic and result in profound, sudden hypoglycemia from insulin stimulation.⁹ Higher doses (> 0.5 g/kg) of xylitol have been associated with acute hepatic necrosis. Clinical signs of xylitol toxicosis include lethargy, weakness, vomiting, collapse, anorexia, generalized malaise, tremors, and seizures (from hypoglycemia). When hepatotoxic doses are ingested, clinical signs and clinicopathologic findings may include melena, icterus, increased liver enzymes, diarrhea, hypoglycemia, hypocholesterolemia, decreased BUN, hypoalbuminemia, etc.

When presented a patient that has ingested a toxic amount of xylitol, a blood glucose should be checked immediately upon presentation; if hypoglycemic, a bolus of 1 ml/kg of 50% dextrose, diluted with an additional amount of 0.9% NaCl (in a 1:3 ratio) should be given IV over 1-2 minutes. Emesis induction should not be performed until the patient is euglycemic. Keep in mind that activated charcoal does *not* reliably bind to xylitol, and is not routinely recommended for xylitol toxicosis. Hypoglycemic patients should be hospitalized for IV fluid therapy [supplemented with dextrose (2.5 to 5% dextrose, CRI, IV)] for approximately 24 hours, and frequent blood glucose check should be performed every 1-4 hours. For patients ingesting a hepatotoxic amount of xylitol, the use of hepatoprotectants (e.g., SAMe), anti-emetics, and supportive care (including frequent liver enzyme monitoring) are warranted.

CHOCOLATE (THEOBROMINE/CAFFINE)

Chocolate is one of the most well-known toxic foods that pet owners are aware of. Chocolate contains methylxanthines such as theobromine and caffeine. Methylxanthines antagonize adenosine receptors and inhibits cellular phosphodiesterases, causing an increase in cAMP. Methylxanthines also stimulate release of catecholamines (e.g., norepinephrine) and cause an increase of calcium entry into cardiac and skeletal muscle, resulting in central nervous system (CNS) stimulation, diuresis, and myocardial contraction. When ingested in toxic doses, clinical signs may include agitation, vomiting, diarrhea, panting, tachycardia, polyuria, hyperthermia, muscle tremors, and seizures. Clinical signs of theobromine toxicosis can be seen at within a few hours, up to 10-12 hours out (as the absorption time

is slow). As theobromine has a very long half-life (e.g., 17 hours), treatment may be necessary for 72-96 hours.¹⁰ Toxic doses of theobromine can be seen at:

- > 20 mg/kg: mild signs of agitation and gastrointestinal distress (e.g., vomiting, diarrhea, abdominal pain)
- > 40 mg/kg: moderate signs of cardiotoxicosis can be seen in addition to aforementioned signs (e.g., tachycardia, hypertension)
- > 60 mg/kg: severe signs of neurotoxicosis can be seen in addition to aforementioned signs (e.g., tremors, seizures)
- 250-500 mg/kg: LD₅₀ (for dogs)¹⁰
- 200 mg/kg: LD₅₀ (for cats)¹⁰

Rarer secondary complications may also be seen from chocolate toxicosis, including pancreatitis and secondary aspiration pneumonia. In general, the darker and more bitter the chocolate, the higher the concentration of methylxanthines in the product. For example, a 20 kg dog would need to ingest approximately 14 oz. of milk chocolate, or 4.5 oz. of semi-sweet, or 2 oz. of unsweetened chocolate to cause moderate signs of toxicity (e.g., agitation, tachycardia). As chocolate tends to stay in the stomach for a prolonged period of time, delayed emesis induction (e.g., even several hours after ingestion) - provided the patient is asymptomatic - may help decontaminate the patient, as chocolate tends to remain in the stomach for quite some time. Further decontamination includes the administration of multiple doses of activated charcoal (1-2 g/kg every 4-6 hours X 4 doses), as methylxanthines undergo enterohepatic recirculation. Treatment includes gastrointestinal support (e.g., anti-emetics), supportive care, IV fluid therapy, frequent walks (to prevent reabsorption of methylxanthines from the urine across the bladder wall), sedatives for agitation (e.g., acepromazine, butorphanol), beta-blockers for persistent tachycardia or hypertension (e.g., propranolol), methocarbamol for tremors, and anticonvulsants for seizures, as needed.

BROMETHALIN

Bromethalin, a neurotoxic rodenticide, is marketed under several common brand names of Assault[®], Tomcat Mole Killer[®], Talpid[®], Real Kill[®], Clout[®], Fastrac[®], Vengeance[®], etc. Bromethalin is *not* an anticoagulant rodenticide and should *not* be treated with Vitamin K₁ as an antidote. Bromethalin works by uncoupling oxidative phosphorylation in the brain and liver mitochondria.¹¹ This results in decreased ATP production, which affects sodium and potassium pumps; as a result, lipid peroxidation occurs, resulting in sodium accumulation within the cell.¹¹ Edema of the central nervous system (CNS) may result.¹¹

In dogs, the LD₅₀ of bromethalin is 2.38-3.65 mg/kg, with a minimum lethal dose being 2.5 mg/kg.¹¹ Cats are more sensitive to the effects of bromethalin, and the LD₅₀ is significantly lower (0.54 mg/kg).¹¹ Clinical signs are dose-dependent, and the onset of clinical signs depends on the amount ingested. Typically, with acute ingestion, signs may be seen within 2-24 hours.¹¹ Clinical signs of CNS stimulation or depression, abnormal behavior, ataxia, hyperesthesia, seizures, and coma may be seen.¹¹ Other common signs include paresis, hind limb paralysis, anisocoria, nystagmus, changes in the pupillary light reflex, and tremors may also be seen. Treatment includes early decontamination, prevention of cerebral edema, and symptomatic supportive care. With recent ingestion in an asymptomatic patient, the use of decontamination (e.g., emesis induction, activated charcoal) is warranted. As bromethalin undergoes enterohepatic recirculation, the use of multiple doses of activated charcoal (without a cathartic) can be administered q 6 hours for 24 hours. Patients should be monitored for signs of neurotoxicity. The use of IV fluid therapy, oxygen support, head elevation, mannitol (to decrease cerebral edema), anticonvulsant

therapy, and thermoregulation is warranted if clinical signs develop. The prognosis varies depending on the amount ingested and the severity of clinical signs; however, prognosis is generally fair to excellent with appropriate decontamination and treatment *prior* to development of clinical signs. If persistent seizures or paralytic syndrome is seen, the prognosis is poorer.

CHOLECALCIFEROL

Cholecalciferol, the chemical name for vitamin D₃, is one of the most deadly– and costly – rodenticides to pets. Ingestion of toxic levels of cholecalciferol can result in severe hypercalcemia and hyperphosphatemia, with secondary acute kidney injury (AKI) developing as a result of dystrophic mineralization to the soft tissue and kidneys. Common sources of Vitamin D₃ include over-the-counter (OTC) or prescription vitamins (typically found in a calcium/Vitamin D₃ combination), psoriasis creams (in the form of calcipotriene), and rodenticides. With cholecalciferol-containing rodenticides, only a tiny amount of rodenticide needs to be ingested before clinical toxicosis occurs due to a very narrow margin of safety within these products. In dogs, cholecalciferol has an LD₅₀ of 85 mg/kg (based on the rodenticide concentration of 0.075%).¹² Doses of Vitamin D₃ > 0.1-0.5 mg/kg can result in clinical signs and hypercalcemia, respectively.¹² Typically, clinical signs often do not develop for 1-3 days until the patient has already developed clinical signs of AKI.¹² That said, renal failure can occur within 12-36 hours following toxic ingestion. Clinical signs and clinicopathologic findings include increased thirst and urination, weakness, lethargy, anorexia, vomiting, generalized malaise, uremic halitosis, dehydration, hypercalcemia, hyperphosphatemia, azotemia, melena, hemorrhagic diarrhea, and death.¹²

Aggressive treatment must be initiated with cholecalciferol toxicosis, due to the narrow margin of safety. Decontamination should include emesis induction, if ingestion was recent and the patient is asymptomatic. As cholecalciferol undergoes enterohepatic recirculation, the administration of multiple doses of activated charcoal (without a cathartic) is warranted q 6 hours X 24 hours. Additional treatment includes the aggressive use of IV fluid therapy to promote calciuresis (e.g., 0.9% NaCl), calcium monitoring, GI support (e.g., anti-emetics, H₂ blockers, sucralfate, phosphate binders, etc.), and the use of medications to increase calciuresis (e.g., prednisone, furosemide) and prevent hypercalcemia (e.g., pamidronate, calcitonin). Treatment is often expensive, and requires hospitalization for an extended period of time. Most patients are continued on oral furosemide and prednisone for weeks, following discharge from the hospital. Frequent monitoring of renal function and electrolytes is imperative. Calcium, phosphorous, BUN, creatinine, and ionized calcium should be evaluated every 12-24 hours while hospitalized, and then every 2-3 days thereafter for the next 2-4 weeks. This will allow one to assess the ability to titrate the prednisone and furosemide therapy, and to ensure that the patient does not develop secondary ARF [or potentially chronic renal failure (CRF)]. Even with aggressive treatment, CRF may be a secondary sequela. The prognosis for this rodenticide is poor due to the risk of CRF once clinical signs and azotemia develop.

INSOLUBLE CALCIUM OXALATES (DIEFFENBACHIA/PHILODENDRON)

According to the [ASPCA Animal Poison Control Center](#), the most common plant exposure is to the *Araceae* plant family.¹³ These plants contain insoluble calcium oxalate crystals, and include the *Dieffenbachia* family of plants. These are common houseplants, as they require little water or light, and can survive in office conditions. Other types of insoluble oxalate containing plants include:¹³

- Arrowhead vine
- Calla lily
- Devil's ivy
- Dumbcane

- Elephant's ear
- Mother-in-law's tongue
- Peace lily
- Philodendron
- Pothos
- Sweetheart vine
- Umbrella plant

The plants contain needle sharp crystals, which are often arranged in bundles called raphides.¹³ When dogs or cats bite or chew into the plant, it releases the crystals, resulting in acute, profuse pain to the oropharynx. Clinical signs of insoluble calcium oxalate plant toxicosis includes: hypersalivation, pawing at the mouth or muzzle, anorexia, vomiting, and edema of the lips, tongue, and oropharynx may be seen.¹³ Very rarely, dyspnea and upper airway swelling can be seen secondary to severe inflammation and swelling of the laryngeal area. If ocular exposure occurs (rare), severe photophobia, pain, and conjunctival swelling can occur. While clinical signs may appear to be dramatic the pet owner, signs are primarily localized to the oropharynx and generally are self-limiting. Treatment can potentially be done at home by the pet owner, and includes removal of the plant, flushing of the mouth (if possible), and offering small amounts of palatable fluid (e.g., canned tuna water, milk, yogurt, chicken broth, etc.) to flush the crystals from the mouth.¹³ For more severe clinical signs that present to the veterinarian, the use of anti-emetics, fluid therapy [e.g., subcutaneous (SQ) or intravenous (IV)], or analgesics may be necessary. Atropine is *not* recommended for the hypersalivation.

SOLUBLE CALCIUM OXALATES

A similarly sounding plant is the soluble oxalate-containing plant. These plants contain oxalic acid and oxalate salts, and must be differentiated from the plant above. Some examples of soluble calcium oxalate-containing plants include: star fruit, common or garden rhubarb, and then shamrock plant.¹⁴ This plant toxicosis is less commonly seen in small animals, and is generally considered more of a concern in large animals (that are chronically grazing on these plants).¹⁴ That said, if this type of plant is ingested in large enough quantities by small animals, it can result in toxicosis.

Soluble calcium oxalates are present in varying degrees in all parts of the plant. For example, rhubarb stems are edible, but the leaves are not.¹⁴ When soluble oxalate salts are absorbed from the gastrointestinal tract (GIT), they bind with systemic calcium, resulting in an acute hypocalcemia.¹⁴ The accumulation of calcium oxalate crystals then potentially can result in nephrosis and acute kidney injury (AKI).¹⁴ While the likelihood of AKI is rare from soluble oxalate-containing plants, there is no known toxic dose reported in small animals. Dehydrated patients or those with underlying renal insufficiency may be more at risk for toxicosis, and should be treated more aggressively.¹⁴ Clinical signs include hypersalivation, anorexia, vomiting, diarrhea, lethargy, weakness, and tetany/tremors (secondary to hypocalcemia).¹⁴ Once AKI has developed, signs of pu/pd, oliguria, oxaluria, hematuria, etc., may be seen 24-36 hours post-ingestion. Treatment for large ingestions includes decontamination (e.g., emesis induction, one dose of activated charcoal), fluid therapy, clinicopathologic monitoring (e.g., for hypocalcemia, oxaluria, azotemia, etc.), anti-emetic therapy, and symptomatic supportive care.

LILIES

The common "true" Lily (from the *Lilium spp.* and *Emerocallis spp.*) is often found in gardens, floral arrangements, or as fresh cuttings. These beautiful, fragrant flowers are known as the common Easter, tiger, Japanese show, stargazer, rubrum, and day lily.¹⁵ All parts of the plant, including the pollen and

water in the vase, are toxic to cats, and result in severe AKI. As little as 2-3 leaves or petals (even the pollen or water from the vase) can result in AKI, and clinical symptoms are typically seen within hours. Clinical signs include early onset vomiting, depression, and anorexia, which progresses to anuric AKI in 1-3 days.¹⁵ Clinicopathologic testing reveals severe azotemia, epithelial casts (12-18 hrs post-ingestion) on urinalysis, proteinuria, and glucosuria.¹⁵ Treatment includes aggressive decontamination (e.g., emesis induction, administration of one dose of activated charcoal), GI support (e.g., anti-emetics, H₂ blockers, etc.), and IV fluid therapy for approximately 48-72 hours (or until resolution of azotemia). The use of SQ fluid therapy is generally not sufficient for the treatment of lily toxicosis. While rarely performed in veterinary medicine, the use of peritoneal or hemodialysis has been successful in anuric AKI cases. With treatment, the prognosis is good if treatment is initiated early and aggressively. Adequate decontamination is of the utmost importance. If aggressive IV fluid therapy is initiated *within* 18 hours, the overall response to therapy is good. However, if treatment is delayed beyond 18-24 hours, or anuria has already developed, the prognosis is grave.¹⁵

CONCLUSION

Pet owners should be appropriately educated on how to pet-proof the house, and be trained on what common human medications can be toxic to pets. Pet owners should also be appropriately educated on crate training to help minimize toxin exposure. When in doubt, the [ASPCA Animal Poison Control Center](#) should be consulted for toxic ingestions that veterinarians are unaware of.



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NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb's Veterinary Drug Handbook*.

COMMON EMERGENCY ROOM PROCEDURES YOU MUST KNOW!

Justine A. Lee, DVM, DACVECC, DABT

CEO, VETgirl, LLC.

Justine@vetgirlontherun.com

www.vetgirlontherun.com

Saint Paul, MN

Performing a thoracocentesis

A thoracocentesis is often life saving, and should be performed immediately in any dyspneic patient that is suspected of having pleural space disease (e.g., pleural effusion, pneumothorax). A thoracocentesis should be performed cranial to the rib, as the blood vessels and nerves lie caudal to the rib (e.g., “hiding” behind the rib). A thoracocentesis should be performed at the 7th to 9th intercostal space (ICS) to avoid the heart (3-5th ICS) or liver (caudal to the 9th ICS). The patient should be shaved, scrubbed, and prepared aseptically. The use of a 3-way stopcock, extension tubing, an appropriately sized needle or catheter, and syringe should be used to collect air or fluid. Appropriate sterile collection tubes should be available for sample collection for fluid analysis, cytology and/or culture purposes. For cats, a 22 gauge, 1-1.5” needle can be used. Depending on the size of dog, an 18 to 22 gauge, 1-3” needle or catheter can be used. NOTE: A short cut technique – rather than counting rib spaces in cases of severe emergency – is to draw an imaginary line caudal to the xiphoid along the lateral body wall. This is approximately the 8th ICS, and thoracocentesis can be performed in this area. If pleural effusion is present, the needle should be directed towards the bottom 1/3 of the chest cavity; if abnormal air is present, the dorsal 1/3 of the chest cavity should be used for thoracocentesis.

Performing an abdominocentesis

An abdominocentesis should be performed when the patient is suspected of having ascites secondary to underlying trauma (e.g., uroabdomen, hemoabdomen, bile peritonitis), metabolic disease (e.g., liver failure, hypoproteinemia), cardiac disease (e.g., right-sided heart failure), neoplasia (e.g., hemangiosarcoma, etc.), etc. An abdominocentesis should be performed using sterile technique. The abdomen should be clipped, shaved and prepared aseptically. A four-quadrant tap around the umbilicus should be performed; note, if you obtain fluid from one location, there is no reason to tap the other remaining 3 regions. Gently, but briskly, insert a sterile 22-ga. needle into these locations. One can use either a closed technique (e.g., needle attached to 3 ml syringe) or open technique (e.g., needle alone). If no fluid is obtained, a “2-tap technique” can be used; a second needle can be inserted several millimeters away from the first sterile needle insertion - this will often allow fluid to gently flow out. Once fluid is seen, gentle suction can be used with an attached 1-, 3- or 6-ml syringe to aspirate the ascites. This fluid should be promptly evaluated for packed cell volume, protein content, cellularity, chemical analyses (e.g., glucose, lactate, creatinine, potassium, or bilirubin if trauma or sepsis is suspected), and presence of intracellular bacterial organisms.

Doing FAST (Focused Assessment with Sonography for Trauma) or TFAST ultrasounds

The focused assessment with sonography for trauma (FAST) ultrasound is a 2-minute procedure that detects the presence of fluid in the abdominal cavity to allow for rapid therapeutic intervention (e.g., fluid resuscitation, abdominocentesis, cytology, clinicopathologic testing). This rapid method of ultrasound is designed to be used by health care professionals with limited ultrasonographic training and is not designed for extensive examination of the abdomen. The added benefit of the FAST examination is the ability to detect very small amounts of fluid. Typically, 5 to 25 ml/kg of fluid needs to be present to be removed by blind abdominocentesis; > 10 to 20 ml/kg of fluid has to be present before it can be detected by fluid-wave assessment on physical examination; and approximately 8.8 ml/kg of

fluid needs to be present before it can be detected radiographically. On the contrary, as little as 2 ml/kg of fluid can be detected on a FAST examination, allowing for rapid diagnosis and identification of underlying pathology.

Ideally, the FAST ultrasound should be performed in the position that the patient is the most comfortable and least stressed (e.g., lateral). The FAST examination typically involves assessment of 4 sites of the abdomen: caudal to the xiphoid, cranial to the bladder, and the right and left dependent flank. The presence of fluid at any of the sites is considered positive. Evaluation of the xiphoid region allows you to check for fluid between the liver and diaphragm and the liver lobes, as well as for pericardial or pleural effusion. Evaluation of the bladder view evaluates for fluid cranial to the bladder and for the presence of a bladder. The right dependent flank allows for fluid detection between the intestines and the body wall, whereas the left dependent flank view allows for identification of the spleen, abdominal effusion near the spleen and body wall, the kidney and spleen, and the liver and spleen.

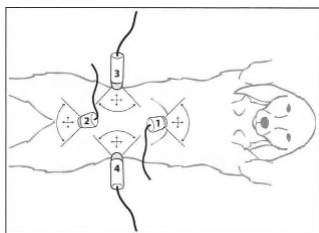


Figure 1. Illustration of the probe placements and movements used to obtain ultrasonographic views of the abdomen via FAST in a dog. Figure courtesy of Boysen SR from IVECCS proceedings 2006.

More recently, the FAST ultrasound has been expanded to include the thorax. The Thoracic Focused Assessment with Sonography for Trauma (TFAST) evaluates for the presence of pleural effusion, pericardial effusion, or even the presence of an occult pneumothorax. In the dyspneic patient, a TFAST can be performed as a rapid diagnostic tool and to assist with ultrasound-guided thoracocentesis (which may be necessary for fluid pockets that may be difficult to obtain blindly). The TFAST can be done quickly and efficiently while the patient is in sternal recumbency. Rapid shaving of the patient (in the least stressful manner possible) allows for a better ultrasonographic image and prepares the patient in the event that a thoracocentesis is necessary. In a dyspneic patient (particularly cats), the TFAST is much less invasive than performing chest radiographs, and is advocated as an easy, repeatable test that can be performed. In a normal patient, the “glide sign” can be seen when imaging the thorax; this is due to the air/tissue interfaces that are created when the parietal pleural slides against the visceral pleura during respiration. Absence of the sliding “slide sign” layers or a “comet-tail artifact” is suggestive of a pneumothorax.

How to perform a gastric lavage

Gastric lavage is a labor-intensive procedure, but is life-saving with certain toxicants. While emesis induction can be safely performed in the majority of poisoned patients (e.g., in asymptomatic patients, with recent ingestion within the past 1-2 hours, etc.), some toxicities warrant the use of gastric lavage. Knowing how to perform a gastric lavage is important, as this procedure needs to be performed in life-threatening situations in the poisoned patient. This should be performed when emesis induction is contraindicated. For example, if the patient is already symptomatic (e.g., too sedate, seizing, trembling, etc.), but the toxicant is still thought to be within the stomach, gastric lavage should be performed. Ideally, this should be performed within 6 hours of ingestion of the toxicant. Rarely,

complications of gastric lavage may occur, and include risks of sedation, secondary aspiration pneumonia (once extubated), mechanical injury (to the mouth, oropharynx, esophagus, stomach), or respiratory effects (e.g., hypoxemia secondary to aspiration, hypercapnea secondary to sedation, etc.). The reader is directed to a toxicology resource for additional information on gastric lavage.

The following materials should be organized prior to the start of the procedure:

- white tape
- mouth gag
- sterile lubricant
- gauze
- orogastric tube
- warm lavage fluid (e.g., tap water) in a bucket
- bilge/drench or stomach pump (or funnel if a bilge is not available)
- sedatives (e.g., pre-drawn and appropriately labeled)
- ETT and anesthesia machine or ambu bag
- empty syringe to inflate the cuff
- material to secure and tie-in the ETT
- IV catheter supplies
- activated charcoal pre-drawn in 60 mls syringes ready for administration (Dose: 1-5 g/kg of charcoal)
- sedation reversal agents if necessary (e.g., naloxone, yohimbine, etc.)
- anti-emetic (e.g., maropitant)

The patient should have IV access established, and then sedated and intubated with an ETT and connected to an ambu bag or anesthesia machine. A potent anti-emetic (e.g., maropitant, ondansetron, etc.) should be given as part of the pre-medication to prevent secondary aspiration. The patient should then be placed in either sternal or right lateral recumbency. An appropriately sized orogastric tube should be pre-measured to the last rib (so you know the maximum distance to insert the tube) and marked with white tape. A mouth gag should be placed in the patient. The tip of the orogastric tube should be lubricated and passed into the stomach using gentle, twisting motions. Appropriate placement of the orogastric tube should be confirmed to ensure that it is not in the airway; once this has been done, warm water should be infused via a bilge/drench pump (or funnel). Copious amounts of lavage fluid can be used for gavage. Attempt to recover the gavage fluid by gravity, emptying it directly into the empty bucket. While gavaging, make sure to frequently palpate the stomach for over-distension. Physical manipulation to massage/agitate the stomach is necessary to help break up stomach contents or bezoars; hopefully, this will allow small material to be removed via the orogastric tube. Perform several lavage cycles (>5-10) to evacuate stomach contents and maximize decontamination. Most of the gavage liquid should be removed prior to activated charcoal administration. Make sure to evaluate the gastric lavage fluid for presence of toxicants (e.g., pills, plant material, etc.). This can potentially be saved for diagnostic evaluation or toxicology testing if malicious or unknown poisoning is suspected. Before removing the orogastric tube, administer activated charcoal (with a cathartic) via the tube, and flush it with additional water (or by blowing forcefully into the tube) to clear it out. Kink the tube (to prevent lavage fluid from being aspirated) prior to immediate removal of the tube. Once kinked, the tube should be removed quickly in one sweeping movement. Extubate the patient only when the gag reflex has returned. Ideally, maintain the patient in sternal (or slightly elevated) recumbency (with the head elevated) to prevent aspiration.

How to trocharize a GDV

Gastric decompression is a necessary part of stabilization of the gastric dilatation-volvulus patient. This can be done by passing an orogastric tube (see “How to perform a gastric lavage”). This author prefers trocharization as compared to orogastric intubation, as it is easy to perform, effective, has minimal side complications, and is less stressful to the patient. The clinician should locate the most tympanic region (estimating where the stomach is) and clip and prep the region. Aseptic technique should be used. A large gauge needle or catheter (e.g., 14 or 16 ga.) should be directed into this area to alleviate gas from the stomach; the sound of hissing gas indicates appropriate placement into the stomach. Rare complications can be seen secondary to trocharization including splenic laceration, gastric perforation, or septic peritonitis. Alternatively, once the patient has been appropriately stabilized, orogastric intubation can be performed to aid in gastric decompression. Goodrich et al evaluated dogs undergoing orogastric intubation versus trocharization in 116 dogs and found that orogastric tube placement was successful in 77% of dogs, with 38% of the dogs requiring sedation.¹ In comparison, trocharization was successful in 86% of the cases, with no need for sedation.¹ (In the author’s opinion, all dogs should be sedated for orogastric intubation unless comatose or obtunded to prevent undue stress and anxiety to the patient.)

How to perform a pericardiocentesis

To perform a pericardiocentesis, light sedation is typically necessary. A cardiovascularly sparing protocol should be used (e.g., butorphanol, fentanyl or hydromorphone and midazolam or diazepam, etc.). Sterile technique and ECG monitoring is imperative.

The appropriate supplies should be prepared in advanced:

- Sterile gloves
- Sterile drape
- Appropriately-sized pericardiocentesis or "centesis" catheter
- Scalpel (to add extra side holes into the centesis catheter, if needed)
- ECG
- Pre-drawn lidocaine (dosed appropriately) in the event of an emergency
- Oxygen supplementation
- A three-way stopcock or one-way valve
- A 12-, 20- or 60- ml syringe (based on patient size)
- An EDTA tube for fluid analysis
- A red top tube for possible culture (rarely done) and to confirm if the sample clots or not (alternatively an ACT tube can be used)

Depending on clinician preference, pericardiocentesis can be performed on the left or right side [The author prefers the right side (e.g., left lateral recumbency) to avoid puncture of the lung (via the cardiac notch)]. Ultrasound-guidance can be used during the procedure, if needed. An over-the-needle catheter system (typically 16 gauge, 1.5 to 4 inches, depending on the size of the patient) can be used, with extra

small side holes smoothly cut into the catheter (via sterile scalpel) to facilitate flow during pericardiocentesis. After the catheter is advanced and the stylet removed, an extension tubing, 3-way stopcock, and syringe should be attached. A small amount should be removed and placed in a red top to evaluate for the presence of a clot. (This is bad. This means you hit the heart. Slowly back out and keep calm). Non-clotting blood is consistent with blood present in the pericardial sac that has already undergone fibrinolysis (This is good. It means you can continue to aspirate during your pericardiocentesis). Once pericardial effusion can no longer be aspirated out gently, ultrasound should be used to confirm improvement or resolution of the pericardial effusion.

How to unblock a feline urethral obstruction

Alleviation of a feline urinary obstruction can be performed using several different techniques or catheter types (e.g., rigid olive tip, Slippery Sam, MILA, etc.), and is dependent on clinician preference. The author prefers using a Tomcat sterile polypropylene catheter initially, followed by a 3.5 to 5 French red rubber catheter. Aseptic technique should be used as much as possible. In order to alleviate the obstruction, the lubricated urinary catheter should be well seated into the tip of the penis, making sure to pull the prepuce caudally to straighten the penile flexure and aid in passing the urinary catheter. A sterile syringe with saline should be used to copiously flush the urethra, with the goal to dislodge and flush the obstructing materials (e.g., crystals, blood clots, cellular debris, calculi, etc.) out of the urethra (either back into the urinary bladder or antegrade out of the tip of the urethra). The author prefers to flush aggressively as the temporary catheter is removed, followed by immediate placement of a longer indwelling urinary catheter (e.g., red rubber, Slippery Sam). The catheter should be sutured in immediately (e.g., Chinese finger trap, etc.) and the bladder copiously flushed. Once the patient is unblocked, a closed collection system should be used to prevent ascending infection.

How to perform a coccygeal block for perineal analgesia

Coccygeal blocks allow us to provide analgesia without affecting motor function. The use of a coccygeal block is beneficial for certain conditions including pelvic fractures, perineal injuries, surgery of the lower urinary tract, feline urethral obstruction, tail pull injuries, and reproductive emergencies (e.g., dystocia). As a coccygeal block will only provide analgesia to the perineal region for approximately 1 hour, additional analgesic therapy (e.g., buprenorphine 11-22 mcg/kg, IV q 6 or long-acting Simbadol at 0.12-0.24 mg/kg, SQ q 24) should be continued. Note that there are some contraindications for performing a coccygeal block including: anatomical abnormalities (e.g., Manx cat, pelvic fractures) that cause loss of appropriate landmarks, skin infections over the insertion site, severe obesity, hypotension, septicemia and coagulopathies. Rare complications can occur with coccygeal blocks, including lidocaine toxicity, infection at the injection site, and inadequate analgesia.

Here, a step-by-step approach on how to perform a coccygeal block:

1. Use a sterile bottle of 2% preservative-free lidocaine at a dose of 0.1-0.2 mL/kg.
2. Sedate the patient. Once sedated, the patient is placed in ventral recumbency and the sacrococcygeal region surgically prepped. The injection site can be found by palpating the space between the sacrum and first coccygeal vertebra, which can be easily palpated when the tail is moved. Draping is not required and may interfere with the procedure, but aseptic technique is imperative.
3. After donning sterile gloves, the injection site is located just cranial to the first coccygeal vertebrae. Alternatively, the first or second coccygeal intervertebral space can be used. To facilitate this step, an assistant can manipulate the tail.
4. A 25 gauge, 1 inch needle is inserted at a 30 to 45° angle at the midline of the sacro-coccygeal space (which is identified with the index finger of the other hand). While

advancing the needle, a characteristic “pop” can be felt. This occurs as the needle penetrates the ligamentum flavum.

5. After entering the epidural space, a syringe is attached and gentle negative pressure applied. If blood or cerebrospinal fluid is obtained, the procedure needs to be started again. If no blood or any other fluid is aspirated, proceed to infuse the calculated volume into the epidural space. No resistance to injection should be noticed. Inappropriate infiltration into the subcutaneous tissue may create resistance. The needle is removed after injection.
6. You can tell if your epidural is working if you notice relaxation of the tail and rectum; also, pinching of the tail should not produce a response. If a pain response is elicited after 5 minutes of the first injection, a second injection can be attempted. Due to the increase risk of complications, no more than 2 attempts are recommended.
7. Urethral de-obstruction can be performed after the lidocaine block has taken effect (which occurs within just minutes).

Conclusion

The veterinary clinician must be comfortable performing certain types of procedures in the emergency room. When in doubt, these procedures should be practiced on a cadaver to ensure appropriate technique and comfort level.

NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb's Veterinary Drug Handbook*.