INTRODUCTION
In veterinary medicine, the primary treatment for toxicant exposure should be decontamination and detoxification of the 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. 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. Decontamination categories may include ocular, dermal, inhalation, gastrointestinal (GI), forced diuresis, and surgical removal to prevent absorption or enhance elimination of the toxicant. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

CARDIAC MEDICATIONS
Certain cardiac medications include broad categories such as calcium channel blockers, beta-blockers, 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. Calcium channel blocker and beta-blocker 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 ARF can potentially develop.\(^1\)\(^-\)\(^3\) 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).\(^3\) Treatment for any cardiac medication includes decontamination (e.g., emesis induction, gastric lavage, activated charcoal 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.\(^1\)

SELECTIVE SEROTONIN RE-UPTAKE INHIBITORS (SSRIS)
Selective serotonin re-uptake inhibitors (SSRIs) are a class of medications that are commonly used in human medicine for depression. Common examples include drugs like fluoxetine (Prozac\textsuperscript{®} in human beings; Reconcile\textsuperscript{™} in veterinary medicine), citalopram (Celexa\textsuperscript{®}), escitalopram (Lexapro\textsuperscript{®}), paroxetine (Paxil\textsuperscript{®}), and sertraline (Zoloft\textsuperscript{®}). Other similar drugs include selective norepinephrine re-uptake inhibitors (SNRIs), which include common drugs like duloxetine (Cymbalta\textsuperscript{®}), nefazodone (Serzone\textsuperscript{®}), and venlafaxine (Effexor\textsuperscript{®}). 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 sedation or central nervous system (CNS) stimulation, anorexia, and lethargy, even at therapeutic doses. Increases in levels of serotonin, even in small doses, may lead to serotonin syndrome. Clinical signs of serotonin syndrome include: CNS stimulation, vomiting, tremoring, seizures, hyperthermia (secondary to tremoring and seizing), diarrhea, abdominal pain, and mydriasis. Treatment includes decontamination (ideally done at a veterinarian, due to the rapid onset of clinical signs), activated charcoal, hospitalization for sedation (e.g., with acepromazine or chlorpromazine), thermoregulation, intravenous (IV) fluid therapy, blood pressure and electrocardiogram (ECG) monitoring, muscle relaxants (for tremors; methocarbamol 22-55 mg/kg, IV), anticonvulsants (e.g., phenobarbital 4-16 mg/kg, IV), serotonin antagonists [e.g., cyproheptadine (1.1 mg/kg for dogs or 2-4 mg total per cat) PO or rectally q. TID-QID], and supportive and symptomatic care.

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 include dextroamphetamine and amphetamine (Adderall®), D-amphetamine (Dexedrine®), methamphetamine (Desoxyn®), and 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: CNS (e.g., agitation, mydriasis, tremors, seizures), cardiovascular (e.g., tachycardia, hypertension), GI (e.g., vomiting, diarrhea, hypersalivating), 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.

SLEEP AIDS
Sleep aids are often benzodiazepines or non-benzodiazepine hypnotics, and include drugs such as zolpidem (Ambien®) and eszopiclone (Lunesta®). These drugs work similarly to benzodiazepines (e.g., diazepam) as they potentiate GABA transmission, increasing frequency of chloride channel opening and resulting in inhibition of neuronal excitation. While these drugs result in sedation in humans, up to 40-50% of dogs ingesting toxic doses of sleep aids develop paradoxical CNS stimulation rather than expected depression. Clinical signs include CNS depression (e.g., depression, ataxia, weakness, paresis), CNS stimulation (e.g., hyperactivity, anxiety, agitation, panting, tremors), or other signs like nausea, vomiting, diarrhea, and hyperthermia. Treatment includes decontamination, activated charcoal, and for those patients demonstrating signs of CNS stimulation, the use of sedatives or anxiolytics. In patients exhibiting CNS stimulation, benzodiazepines (e.g., diazepam IV) should not be used, as they may worsen the symptoms. Rather, the use of phenothiazines (e.g., acepromazine, chlorpromazine) or barbiturates (e.g., phenobarbital IV) should be used instead. In severe cases of respiratory or cardiac depression, the use of flumazenil, the reversal agent for benzodiazepines, can be considered.

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 gluronidation 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 pyrethin/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 macrocylic lactone derivative in the same family as ivermectin). Certain
breeds with the MDR-1 gene mutation (now known as the ABCB1-1 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.

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 ARF 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 ARF.

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. Renal compromise may be seen at doses of 100-250 mg/kg (resulting in potential ARF), and fatalities have been reported at doses > 300 mg/kg. This differs tremendously from naproxen sodium (dogs), where severe clinical signs can be seen at doses as low as 5 mg/kg. With naproxen, experimental canine doses of 22 mg/kg orally once a day for 3 days have resulted in perforation of the GI tract with secondary septic peritonitis occurring.

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.

LILIES
It is important to keep in mind that not all species of "lilies" are poisonous – the Calla lily (e.g., insoluble oxalate), peace lily (e.g., insoluble oxalate), and lily-of-the-valley (e.g., cardiac glycoside) all have different mechanisms of toxicosis; these particular type of lilies do not result in ARF when ingested by cats. The more dangerous lilies (from the Lilium spp. and Hemerocallis spp.) are 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. All parts of the plant, including the pollen, are toxic to cats, and result in severe ARF. As little as 1-2 leaves or petals, even the pollen, can result in ARF, and clinical symptoms are typically seen within hours. Even the water contained with the vase is considered poisonous, as the toxic is water-soluble. Clinical signs of lily poisoning include early onset vomiting, depression, and anorexia, which progresses to anuric ARF in 1-3 days. Clinicalopathologic testing reveals severe azotemia, epithelial casts (12-18 hrs post-ingestion) on urinalysis, proteinuria, and glucosuria. Treatment includes aggressive decontamination and IV fluid therapy for approximately 48-72 hours (or until resolution of azotemia). The use of SQ fluid therapy is 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 ARF 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.
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.

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.

BATTERIES
Battery ingestions occur quite frequently by dogs. This is often witnessed by the owner, or a chewed battery may be discovered by the owner. Often times, the pet owner may notice that the remote control is chewed on and the batteries are missing. When the casing for a battery is punctured, there is risk for alkaline or acidic material to leak out, resulting in severe ulceration to exposed tissues. The most common battery ingestion is of an alkaline dry cell battery (e.g., 9-volt, D, C, AA, AAA) or button/disc batteries. Alkaline dry cells (the majority of household batteries) contain potassium hydroxide or sodium hydroxide. When the compounds come in contact with tissue, liquefaction necrosis occurs, causing deeply penetrating ulcers. In addition, newer types of “disc shaped” batteries can allow an electric current to pass to the tissues of the GI tract as the battery is passed. This can result in a current-induced necrosis, resulting in tissue damage or even perforation of the oropharynx, esophagus, stomach or small intestine. Lithium button type batteries are the most dangerous, as one 3 volt battery can result in severe necrosis to the GI tract or esophagus within 15-30 minutes of contact. Finally, certain batteries contain heavy metals (e.g., mercury, zinc, cobalt, lead, nickel or cadmium). Heavy metal toxicity can occur, albeit rare, if the battery remains in the GI tract for more than 2-3 days.

With any type of battery ingestion, the pet owner should seek veterinary attention immediately. A thorough oral exam and physical exam should be performed. Oral ulcerations may not be present on physical examination for several hours, and the absence of oral ulcerations does not rule out severe underlying corrosive injury lower in the GI tract. The presence of black powdered material may be seen in the mouth, and occurs when dry cell batteries are punctured. The mouth should be thoroughly flushed and lavaged for 15-20 minutes with tepid tap water. A lateral abdominal radiograph (including the caudal esophagus in the chest) should be performed to evaluate the presence of the battery in the abdomen. Ideally, prompt removal should occur to prevent further corrosive injury. The use of endoscopy or surgery may be necessary. Emesis induction is not typically recommended, as corrosive injury may occur to the esophagus and oropharynx. Treatment includes removal of the battery, anti-ulcer medication (including H₂ blockers and sucralfate) for 5-7 days, a bland or high-fiber diet, and analgesic therapy if necessary.

SILICA GEL 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.

FOOD OXIDIZER PACKS
Some types of “silica gel packs” are actually oxygen absorbers. These are commonly found in beef jerky or rawhide bags and may contain iron. When ingested in large amounts, these packs can potentially
result in iron toxicosis. The powder within these oxygen absorbers is often black in color and magnetic. Treatment for iron toxicosis includes antacid therapy (e.g., milk of magnesia), symptomatic supportive care, monitoring blood iron levels, and potential chelation (in severe cases). The use of activated charcoal is not warranted with iron toxicosis, as it does not reliably bind to heavy metals.

**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. Sugarless products, particularly those with xylitol listed within the first five active ingredients, 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 mg/piece 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. 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, etc. When hepatotoxic doses are ingested, clinical signs and clinicopathologic findings may include melena, icterus, increased liver enzymes, diastasis, 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.

**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), the presence of mycotoxins or pesticide residues on the fruit, 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. 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 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. As there is no current veterinary peer-reviewed, scientific published toxic dose of grapes and raisins, 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. Within the next 12-24 hours, clinical signs of lethargy, dehydration, vomiting, diarrhea, anorexia, abdominal pain, uremic breath, and diarrhea may be seen. Azotemia may develop within 24 hours, with hypercalcemia and hyperphosphatemia occurring first. Oliguria and anuria may develop 48-72 hours post-ingestion, at which point the prognosis is poorer. Treatment includes decontamination, aggressive IV fluid therapy, anti-emetics, blood pressure and urine output monitoring, and serial blood work monitoring (q. 12-24 hours). 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.  

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.).

FERTILIZERS
Fertilizers generally have a wide margin of safety, and result in mild GI signs when ingested directly. Ingestion of grass that had a fertilizer applied to it previously rarely results in serious toxicosis; more serious clinical signs can be seen when the product is directly ingested (e.g., directly out of the bag). When appropriately applied or diluted, these chemicals typically wash into the soil after rainfall, resulting in low-risk to patients. What is key is to make sure that the compound was not mixed or does not contain more dangerous insecticides such as carbamates or organophosphates.

BONE OR BLOOD MEAL
Bone meal and blood meal are by-products from the meatpacking industry that are widely utilized as soil amendment products, fertilizer components, or as deer, rabbit and wildlife repellants. Bone or blood meal are “organic” compounds, and with the increased use of organic products in lawn and gardening, have resulted in increased exposure opportunities for animals. These are often considered low-level toxicities, but can result in FBO, severe pancreatitis, or GI tract irritation with ingestion. A thorough history must be obtained from the pet owner, as these products are often mixed with more toxic agents (such as organophosphates [OPs] found in rose fertilizers) which result in severe toxicity. Bone meal and blood meal are highly palatable to dogs and can result in unintentional, large ingestions. Tulip, daffodil and hyacinth bulbs are often “dusted” in bone meal when planted to fertilize and aid in repelling squirrels. The scent of bone meal may entice dogs to dig up newly planted bulbs and subsequently ingest both the potentially toxic bulb and bone meal. Large ingestions of bone meal can congeal into a solid ball or bezoar in the stomach, resulting in a FBO. Bone meal and blood meal are highly palatable to dogs and can result in unintentional, large ingestions. Tulip, daffodil and hyacinth bulbs are often “dusted” in bone meal when planted to fertilize and aid in repelling squirrels. The scent of bone meal may entice dogs to dig up newly planted bulbs and subsequently ingest both the potentially toxic bulb and bone meal. Large ingestions of bone meal can congeal into a solid ball or bezoar in the stomach, resulting in a FBO. Decontamination is recommended with recent large ingestions or with dogs with a prior history of pancreatitis. Radiographs should be performed to determine if the material has passed out of the stomach prior to emesis induction, and to evaluate for the presence of gastric contents or FBO. With massive ingestions demonstrating evidence of FBO, surgical intervention may be necessary. In general, decontamination and symptomatic and supportive care are indicated.

CONCLUSION
Pet owners should be appropriately educated on how to pet-proof the house, and be trained on what common household products and kitchen items are poisonous. Pet owners should also be appropriately educated on crate training to help minimize toxin exposure. Once a pet is exposed to a toxicant, it is imperative to determine if emesis is appropriate, and to understand when it may be contraindicated (e.g., symptomatic patient, delayed time since exposure, hydrocarbons, etc.). Knowledge of the underlying mechanism of action, the pharmacokinetics (including absorption, distribution, metabolism, and excretion), and the toxic dose of the toxicant are imperative in determining appropriate decontamination and therapy for the patient.

REFERENCES


