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Toxicology: Managing Common Household, Yard, and Garden Hazards

Lead

Lead is a heavy metal that is used in a large variety of products including lead weights, lead-based paints, lead solders, wire shielding, auto batteries, caulks, lead-containing toys and lead shot. Although household paints manufactured after 1977 can only contain 0.06% lead, it may still be present in large amounts in agricultural or industrial paints. Lead absorption is high in the acidic environment of the stomach and lower in the intestine, therefore the biggest risk of intoxication is when lead is in the stomach.

Lead poisoning can be either acute or chronic. Acute signs in dogs and cats include anorexia, agitation, behavior changes, ataxia, muscle tremor and intermittent seizures. Chronic effects include vomiting, diarrhea, abdominal discomfort, anorexia, personality changes, lethargy, ataxia, intermittent seizures, weight loss, anemia and megaesophagus which occurs primarily in cats. Lead can also cause a variety of hematological abnormalities including anemia, basophilic stippling, nucleated red blood cells and elevated liver and kidney values.

Lead levels is measured in whole blood (heparinized or EDTA), not serum, since >90% of circulating lead is bound to RBCs. Normal background levels in animals are usually less than 0.1 ppm (10 µg/dl). Levels exceeding 0.35 ppm (35 µg/dl) are suggestive of lead toxicity and are usually associated with clinical signs. In the presence of appropriate clinical signs, levels greater than or equal to 0.35 ppm are diagnostic of lead toxicity. Levels between 0.1 and 0.35 ppm suggest significant lead exposure, and, in the presence of consistent clinical signs, are suggestive of lead toxicity. In chronic lead toxicity, blood lead levels may not be appreciably high, due to distribution of lead from blood to tissues. Blood lead levels are not reflective of total body burden nor do they necessarily correlate to severity of signs.

Management of lead intoxication consists of first stabilizing the patient and addressing any severe clinical signs such as controlling seizures. Lead should then be removed from the GI tract (by bulking the diet or surgery). Once the lead is removed from the GI tract, chelation can be carried out. General symptomatic and supportive care should also be carried out as needed.

Batteries

Flashlights, remote controls, battery-operated toys, watches, calculators, hearing aids, etc. all provide the opportunity for pets, especially dogs, to be exposed to alkaline or disc batteries. Most alkaline dry cell batteries use potassium hydroxide or sodium hydroxide to generate current, and disc, nickel-cadmium, and silver batteries are generally of the alkaline type. The alkaline gel within a battery can cause liquefactive necrosis of tissue, resulting in burns that can penetrate deeply. Lithium disc batteries may lodge in the esophagus, increasing the risk of esophageal ulceration. In addition, batteries casings may result in respiratory or gastrointestinal obstruction if inhaled or swallowed.

When batteries are chewed and the contents released, alkaline burns result. Signs of foreign body obstruction (vomiting, anorexia, tenesmus, etc.) may occur when casings are swallowed; disc batteries may be inhaled, resulting in acute dyspnea and cyanosis.

Treatment of battery exposures is as for exposure to any alkaline product. Complete evaluation of the oral cavity and pharynx for ulceration or irritation should be performed upon presentation of the animal to the veterinarian, although with very recent exposures the oral cavity may appear normal. Evidence of oral discomfort and inflammation generally develop within 2 to 4 hours, although the full extent of injury may not be evident until 12 hours post exposure.

It is important to remember that the absence of oral burns does not preclude the development of esophageal burns. Endoscopy may be elected for cases in which esophageal damage is a concern, although delaying endoscopy for 12 hours will allow the full extent of the burns to develop. If mucosal burns develop, treatment should include antibiotics, pain medication as needed, gastrointestinal protectants (e.g. sucralfate slurries) and general supportive care. Corticosteroid use is controversial. In cases with severe oral burns or esophageal burns, placement of a gastrostomy tube will facilitate nutritional support while allowing for mucosal healing. Esophageal lesions may take weeks to heal and there may be risk of stricture formation, leading to impairment of esophageal function.

In the case of lithium batteries, administration of tap water in 20 ml boluses every 15 minutes has been shown to decrease the severity and delay the development of current-induced tissue injury in dogs. Radiography to determine the location of the battery casing should be performed in cases where the casing is missing.

The decision to remove a battery present in the stomach depends on the size of the animal, battery size, and evidence of battery puncture. Batteries that are small relative to the size of the animal will often pass uneventfully through the GI tract and into the stools. Bulky diets may assist in the passage of the battery. If the battery is not seen in the stools within 3 days of ingestion, radiography is recommended to verify the location of the battery. Batteries that have not passed through the pylorus within 48 hours are unlikely to do so and may require endoscopic or surgical removal, although endoscopic removal is not recommended in cases where there is suspicion that the battery has been punctured.

Mothballs

Mothballs may be composed of either 100% naphthalene or 99% paradichlorobenzene. Naphthalene-based mothballs are approximately twice as toxic as paradichlorobenzene, and cats are especially sensitive to naphthalene. Naphthalene causes Heinz bodies, hemolysis, and, occasionally, methemoglobinemia in dogs at doses > 400 mg/kg. Mothballs generally weigh ~ 5 g. One 5 g mothball contains 5000 mg of naphthalene. Therefore in a 20-lb dog, 1 naphthalene moth ball is enough to cause toxicosis. Paradichlorobenzene primarily affects the liver and CNS, although methemoglobinemia and hemolysis have been reported in humans.

Signs of ingestion of naphthalene mothballs may include vomiting, weakness, icterus, lethargy, icterus, brown-colored mucous membranes, and collapse. Rarely, hepatitis has been reported 3-5 days post-ingestion. Paradichlorobenzene mothballs may cause GI upset, ataxia, disorientation, and depression. Elevations in liver serum biochemical values may occur within 72 hours of ingestion.

Treatment of mothball ingestion includes early emesis, activated charcoal, and cathartic. Treatment for hemolysis or methemoglobinemia (blood replacement therapy, N-acetylcysteine, etc.) may be necessary. Intravenous fluid diuresis should be maintained in cases with hemolysis in order to minimize

the risk of hemoglobin-induced renal nephrosis. Evidence of hepatic damage, based on biochemical values, would indicate that symptomatic therapy for general liver failure (SAMe, dietary management, etc.) should be instituted.

Paintballs

Paintballs are used in recreation "war" games. They contain different types and colors of paint inside a gelatin capsule. Ingestion of large numbers of paintballs has been associated with acid/base imbalances, electrolyte disorders (most frequently hypernatremia), neurologic signs (ataxia, seizures), and occasional death in dogs. The mechanism of action is thought to be an alteration of the body's water balance. Ingredients found in paintballs, such as polyethylene glycol, glycerol, and sorbitol, are osmotically active agents capable of causing fluid shifts from the vasculature into the bowel lumen with a resultant increase in plasma osmolality and hypernatremia.

Management should include emesis, in asymptomatic patients, if large numbers of paintballs are ingested. Activated charcoal is contraindicated as it will pull more fluids into the GI tract. Monitor electrolytes carefully and correct any imbalances. Warm water enemas may help stimulate movement of paintballs through the GI tract and will help correct hypernatremia. In symptomatic animals, monitor electrolytes and acid-base values every 2-4 hours until clinical signs resolve and values normalize. In hypernatremic patients, administer low sodium IV fluids (D5W, 2.5% dextrose + 0.45% NaCl) and repeat enemas until serum sodium levels return to normal. Fluid rates may need to be quite high due to compensate for loss of fluids into the GI tract.

Ice Melters

Ice melters can contain multiple different ingredients. Many contain one or more of the flowing salts: sodium chloride, potassium chloride, magnesium chloride or calcium chloride. Some products can contain calcium carbonate, calcium magnesium acetate or urea. The type and severity of the clinical signs that can develop will depend on the ingredients and the amount ingested.

In most cases, ingestion of small amounts of most ice melts (e.g. licking paws after walking through product) will cause mild gastrointestinal upset that is usually self-limiting. Management of low-level ice melt exposure involves dilution in asymptomatic animals and management of vomiting (NPO, antiemetics) in symptomatic animals.

Sodium chloride-based ice ingested in large amounts may cause hypernatremia which can lead to vomiting, polyuria/polydipsia, fasciculations, tremors, seizures, tachycardia, and acidosis. In acute exposures to sodium chloride, hypernatremia develops quickly so it is safe to decrease the serum sodium rapidly. The fluid of choice is 5% dextrose in water. Anticonvulsants can be used to help control CNS signs. In cases of acute hypernatremia of short duration (less than 12 hours), warm water enemas may aid in correction of the electrolyte abnormality.

Ingestion of large amounts of potassium chloride-based ice melts may cause severe GI irritation, potentially resulting in GI hemorrhage. Hyperkalemia may also occur, primarily in patients with decreased renal efficiency. Signs associated with hyperkalemia are vomiting/diarrhea, weakness, hypotension, and abnormal cardiac conduction. Fluids (LRS or saline) and furosemide or hydrochlorothiazide are used to treat the hyperkalemia. Other recommended treatments include electrolyte, glucose and renal function monitoring.

Ingestion of ice melts containing magnesium chloride may cause hypermagnesemia in patients with compromised renal function, in addition to gastrointestinal irritation. More severe signs would be weakness, respiratory depression, hypotension, cardiac abnormalities and impaired neuromuscular transmission. Treatment is mainly symptomatic and supportive.

Ingestion of calcium-based ice melts may cause transient elevated serum calcium levels, but they usually do not cause serious hypercalcemia following acute ingestion. Calcium carbonate and calcium-magnesium acetate ice melts are irritants that can cause mild gastritis while calcium chloride ice melts are capable of causing severe irritation, including oral/esophageal ulceration and gastrointestinal hemorrhage. Because of the potential for oral mucosal injury from calcium chloride ice melts, induction of emesis is contraindicated following ingestion. Treatment is symptomatic and supportive including treatment for severe mucosal irritation with exposure to the calcium chloride form including GI protectants such as sucralfate slurries and an H2 blocker or proton-pump inhibitor. The patient should also be started on broad-spectrum antibiotics and pain control provided.

Ingestion of large amounts of urea-based ice melters can cause GI upset but life-threatening signs are not expected. Management of the GI signs is important to prevent dehydration.

Hydrocarbons

Hydrocarbons which are found in gasoline, kerosene and a variety of other products have the potential to affect 5 body systems: CNS, GI, ocular, respiratory and skin. Ingestion and dermal exposure can cause GI and skin irritation. CNS depression is also possible. Aspiration of hydrocarbons causes the largest concern. The more volatile a hydrocarbon is, the greater potential for adverse clinical effects. If an animal is exposed and remains asymptomatic for 6 - 12 hours, they are unlikely to develop problems. Treatment is symptomatic and supportive in animals that are showing clinical signs.

Bleach

Bleaches are irritants at low concentrations. Exposure usually involves dermal, ocular, oral, respiratory, or gastrointestinal irritation. Bleaches with alkalinity property (pH>11) could potentially be caustic. The severity of injury depends on the concentration of the product and the duration of contact. When bleach is combined with an acid or ammonia, chloramine gas can be produced, which if inhaled, can cause mucous membrane and respiratory irritation.

Fabric Softener

Fabric Softeners contain cationic detergents. Cationic detergents are more toxic than non-ionic/anionic detergents and can cause extensive systemic and local effects at levels as low as 2% or less. Cats are much more sensitive to the effects of cationic detergents than dogs. Local tissue injury caused by cationic detergents resembles that seen with exposure to alkaline products. In addition, cationic detergents can cause systemic toxicity including CNS depression, coma, seizures, hypotension, muscular weakness and fasciculations, collapse, pulmonary edema, and metabolic acidosis. The mechanism of these signs is not known.

Dryer sheets contain cationic detergents so ingestion can cause localized corrosive injury and potentially foreign body obstruction. Dryer sheets that have already been used contain less of the detergents and

pose less danger. Treatment would include monitoring for signs of corrosive injury in the mouth and GI tract, bulking the diet to help pass the sheet, and managing and CNS effects, hypotension and pulmonary edema. In general the remainder of the treatment would be as discussed for batteries above.

Tremorgenic Mycotoxins

Tremorgenic mycotoxins produced by molds on foods are a relatively common, and possibly under-diagnosed, cause of tremors and seizures in pet animals. Because of their relatively indiscriminate appetites, dogs tend to be most commonly exposed to tremorgens. These toxins are produced from a variety of fungi, however tremorgens produced by *Penicillium* spp. are the most commonly encountered. These molds grow on practically any food, including dairy products, grains, nuts, and legumes; compost piles may also provide a source of tremorgens. Tremorgens have a several different mechanisms of actions: some alter nerve action potentials, some alter neurotransmitter action, and while others alter neurotransmitter levels. The overall affect is the development of muscle tremors and seizures.

Clinical signs include fine muscle tremors that may rapidly progress to more severe tremors and seizures. Death generally occurs in the first 2 to 4 hours and is usually secondary to respiratory compromise, metabolic acidosis or hyperthermia. Other signs that may be seen include vomiting (common), hyperactivity, depression, coma, behavior alterations, tachycardia, and pulmonary edema.

Asymptomatic animals exposed to moldy foods should be decontaminated via emesis or lavage followed by activated charcoal and cathartic. In symptomatic animals, control of severe tremors or seizures has priority over decontamination. Seizures may respond to diazepam, however others have had better success with methocarbamol (Robaxin®; 55-220 mg/kg IV to effect), especially in seizuring animals. Barbiturates may be used in animals that are unresponsive to other anticonvulsants. Supportive care should include intravenous fluids, thermoregulation, and correction of electrolyte and acid-base abnormalities. In severe cases, signs may persist for several days, and residual fine muscle tremors may take a week or more to fully resolve. Testing of stomach content, suspect foods, or vomitus for tremorgens is available through many diagnostic labs.

Ant and Roach Traps

Ant and Roach Traps contain multiple active ingredients including chlorpyrifos, sulfluramid, fipronil, propoxur, boric acid, and hydramethylnon at very low concentrations. These are not likely to cause any significant clinical effects. Bait containing avermectins and arsenic are potentially more serious (avermectins in Collie breeds or dogs with microfilaria and arsenic in cats). Baits also contain inert ingredients such as peanut butter, breadcrumbs, sugar and vegetable or animal fats to attract the insects. These may cause GI upset and the plastic or metal may pose a foreign body hazard.

Pennies

Ingestion of coins by pets, especially dogs, is not uncommon. Of the existing US coins currently in circulation, only pennies pose a significant toxicity hazard. Pennies minted starting in 1982 contain 97.5% zinc and 2.5% copper, making ingested pennies a rich source of zinc. Other potential sources of zinc include hardware such as screws, bolts, nuts, etc., all of which may contain varying amounts of zinc.

In the stomach, gastric acids leach the zinc from its source, and the ionized zinc is readily absorbed into the circulation, where it causes intravascular hemolysis.

The most common clinical signs of penny ingestion are vomiting, depression, anorexia, hemoglobinuria, diarrhea, weakness, collapse and icterus. Secondarily, acute renal failure may develop. Clinical laboratory abnormalities will be suggestive of hemolysis (elevated bilirubin, hemoglobinemia, hemoglobinuria, regenerative anemia) and may also indicate the development of kidney failure. Serum zinc levels may be obtained—blood should be collected in all plastic syringes (no rubber grommets) and shipped in Royal blue top vaccutainers to minimize contamination with exogenous zinc. Radiography of the abdomen may reveal the presence of coins or other "hardware" within the stomach. In some cases, bismuth subsalicylate tablets (such as Pepto Bismol®) can be mistaken for coins on radiographs since they are radio-opaque.

Treatment for recently ingested pennies may include induction of vomiting. Activated charcoal is not indicated, as it is of little benefit in binding metals. Removal of zinc-containing foreign bodies via endoscopy or gastrotomy/enterotomy may be required. The patient should be radiographed following removal of the coins to ensure that coins hidden in gastric ruggae were not missed. Treatment for symptomatic animals should include blood replacement therapy as needed, intravenous fluids, and other supportive care. The use of chelators is generally not required as zinc blood levels drop rapidly as soon as the source is removed.

Grapes and Raisins

The ingestion of raisins or grapes by dogs may result in acute renal failure. APCC also has a few reported cases in cats and one ferret. All cases of grape and raisin exposure should be considered as potentially serious since the minimum toxic dose in dogs for grapes and raisins isn't known. There is a case in the APCC database of 2 Maltese dogs (10 and 15 pounds) that were each fed 3 grapes. 48 hours post exposure, the dogs were asymptomatic but referred for lab work. Mild azotemia was documented but urine specific gravity was not obtained. 48 hours of IV fluids corrected the azotemia in both dogs and no other signs developed. To date the toxic principle is unknown. Analysis of grapes or raisins involved in some of these cases has been negative for heavy metals, pesticides, and known mycotoxins.

In most cases, dogs have shown vomiting, usually within 6 hours of ingestion. Grapes/raisins are often seen in the vomitus. Other signs reported in the first 24-36 hours were diarrhea (+/- blood), anorexia, lethargy, and abdominal pain. Most dogs have elevated serum creatinine and BUN upon presentation to the veterinarian. Some dogs also have elevations in serum calcium, phosphorus, glucose, liver enzymes, amylase or lipase. Many of the dogs will develop anuric or oliguric renal failure within 36-72 hours of ingestion of grapes or raisins. In one study, 47% of symptomatic dogs either died or were euthanized due to poor response to treatment for renal failure. One dog with anuric renal failure recovered following peritoneal dialysis.

Dogs, cats, or ferrets who have ingested grapes or raisins, especially in large quantities, should be managed aggressively. Early decontamination via emesis or lavage followed by activated charcoal is recommended. Fluid diuresis (two times maintenance) for 48 hours should be instituted, and serial serum chemistries should monitored for at least 72 hours post ingestion. Use of diuretics to maintain adequate urine flow is essential in cases of oliguria or anuria. If available, peritoneal dialysis or hemodialysis may be considered in cases of refractory anuria/oliguria. Symptomatic care for vomiting,

diarrhea, or other signs may be required. Animals who develop severe oliguria or anuria have a poor prognosis.

Xylitol

Xylitol is a sugar alcohol which is approximately as sweet as sucrose. It is obtained by acid hydrolysis of birch bark and occurs naturally in low levels in fruits. Xylitol is used in low-carbohydrate products, in gums and other candies, in toothpaste, mouthwashes, and some sun screens. It was used regularly in Scandinavian countries and Japan and over the past few years has become very popular in the United States.

The popularity of xylitol is due to the following characteristics: it is a naturally occurring substance so consumers may believe it is safe, it has a low glycemic index so it is a good sugar substitute for diabetics, it has fewer calories than sugar and it is anti-bacterial so it prevents tooth decay. In humans, xylitol does not significantly raise glucose in blood and it does not significantly stimulate insulin release.

In dogs, xylitol can cause significant hypoglycemia and liver failure. The most common signs reported in dogs following ingestion include vomiting, depression, lethargy, weakness, ataxia and seizures. Hypoglycemia and hypokalemia can develop. Signs can be seen within 15-30 minutes. The APCC uses 50 mg/kg for as a trigger dose for hypoglycemia.

Treatment may include inducing emesis if the patient is asymptomatic. Activated charcoal is not expected to bind to xylitol since it is a small alcohol. In asymptomatic patients, monitor blood glucose and provide frequent small meals. Symptomatic dogs should be treated with first a bolus of dextrose then a CRI. Small frequent meals should be provided. Potassium supplementation should be instituted if serum potassium falls below 2.5 mEq/L. Monitor for liver effects. Increases in ALT, AST, and ALKP usually occur within 24 hours. Most dogs appear to recover but aggressive liver support is appropriate (Fluids, SAMe, Marin, Milk Thistle, n-acetlycysteine).

Macadamia Nuts

Macadamia nuts are cultivated from *Macadamia integrifolia* trees commonly found in Hawaii and Australia. Macadamias are a popular nut for snacking, and are used in baking. Clinical signs are reported at ingestions as low as 2.4 g/kg body weight in dogs. No deaths have been reported at this time. Doses of 1 g/kg, or higher, require decontamination. Clinical signs include weakness, depression, vomiting, ataxia, tremors, transient paresis, and hyperthermia. Mild elevations in serum triglycerides, lipase and alkaline phosphatase may be seen, and should return to normal in 48 hours. Treatment of clinical signs includes fluids and thermoregulation. A cold water enema can speed recovery. Prognosis of macadamia nut ingestion is good. Only dogs appear to be at risk.

Bread Dough

Bread dough ingestion can cause the following problems: foreign body obstruction, gastric dilation and/or volvulus, ethanol toxicosis and metabolic acidosis. When rising bread dough is ingested, the internal body temperature causes the dough to rise rapidly and the yeast to produce ethanol which is rapidly absorbed from the stomach. Clinical signs may include vomiting, retching, abdominal distension, behavioral changes, vocalization, ataxia, and CNS depression.

Treatment is symptomatic and supportive. If the ingestion was recent and no clinical signs have developed, inducing emesis may be effective. If the patient has developed clinical signs, gastric lavage can be performed to release the gas and remove a portion of the dough. The use of cool water for the lavage may slow the fermentation process by the yeast. In extreme cases, surgical removal of the dough may be necessary. Acidosis should be managed and IV fluids started. The prognosis is good if signs are managed.

Alcoholic Beverages

Ethanol is found in these beverages. All alcohols are rapidly absorbed orally; dermal absorption can also occur. Inhalation, particularly of concentrated fumes in a confined area, can also cause systemic signs. Signs develop rapidly, often within 30-60 minutes, and include vomiting, ataxia, tremors, hypothermia, hypoglycemia, acidosis, aspiration pneumonia, respiratory depression, and coma. Due to rapid onset of signs, decontamination should be performed only within the first 30 minutes following ingestion. Other treatment is symptomatic and supportive and includes fluid diuresis, thermoregulation, and correction of acidosis and hypoglycemia.

Chocolate

Methylxanthines are alkaloids that occur naturally in plants and are found in tea, coffee beans, cola beans, and cocoa beans. The methylxanthines in chocolate include caffeine and theobromine. Some methylxanthines are used therapeutically as bronchodilators including theophylline and aminophylline. Chocolate toxicity is a very common, year round problem with peak times or "Chocolate Season" beginning with Halloween then Christmas through Valentine's Day and ending with Easter.

Methylxanthines are rapidly absorbed by mouth and metabolized in the liver. Elimination is via the bile and/or urine and varies with the age and species of the patient. Methylxanthines undergo enterohepatic recirculation and can be reabsorbed via the bladder wall. These characteristics will guide decontamination recommendations (see below).

The elimination half-life of methylxanthines varies widely by species. For theophylline, the elimination half-life is 5.7 h in dogs, 7.8 h in cats and 20-36 h in humans. For theobromine, the elimination half-life is 17.5 h in dogs and 6-10 h in humans. For caffeine, the elimination half-life is 4.5 h in dogs, 3-6 h in humans (longer in infants and for overdoses in adults) and 1.5 - 2 h in mice and rats. The toxic doses for dogs are theobromine LD_{50} 250-500mg/kg, caffeine MLD 140-150 mg/kg and theophylline LD_{50} 250 mg/kg. For cats, theobromine LD_{50} 200 mg/kg, caffeine MLD 100-150 mg/kg and theophylline LD_{50} 800 mg/kg.

Methylxanthines work by blocking adenosine receptors (adenosine is a bronchoconstrictor, anticonvulsant, and regulates heart rhythm), by increased calcium movement into cells and by increasing cAMP via inhibition of phosphodiesterase. These actions show clinically as stimulation of the CNS and cardiovascular systems.

In dogs, the listed chocolate dosages are expected to show the following clinical effects. 20 mg/kg: mild signs (vomiting, hyperactivity, PD). 40-50 mg/kg: moderate to severe signs (cardiotoxicity). 60 mg/kg: seizures possible. And 100 mg/kg is the minimum lethal dosage.

Clinical signs usually develop within 6-12 hours of ingestion and can include polydipsia, vomiting, diarrhea, bloating, restlessness, hyperactivity (progressing to ataxia, tremors and seizures), cardiac effects (PVCs, tachycardia), tachypnea, hypertension, hyperthermia and coma. Pancreatitis is also possible since chocolates have high fat and sugar content and may contain nuts.

Calculating the dosage of methylxanthines exposure is an important step in managing these cases. It is often difficult to identify the amounts and types of chocolate used with products from a bakery or a prepackaged mix and the same problem can arise with assorted filled candies and chocolate-covered nuts. It is therefore best to assume the worst case scenario and calculate the dosage as if the product was solid chocolate. First determine the amount of methylxanthines in the type of chocolate using the table below, then calculate the dosage of total methylxanthines per kg of patient body weight.

Type of Chocolate	Theobromine Content per oz	Caffeine Content per oz	Total Methylxanthines per oz
White	0.25	0.85	1.1
Milk	58	6	64
Dark	130	20	150
Semi-Sweet	138	22	160
Unsweetened Baking	393	47	440
Cocoa Powder	737	70	807

An example dosage calculation follows: A 9 # dog eats 5 oz dark chocolate. Dark chocolate contains approximately 130 mg theobromine and 20 mg caffeine per ounce. To calculate dose based on total methylxanthines: 5 oz X 150 mg/oz = 750 mg and 750 mg divided by 9 kg equals 83.3 mg/kg as the total methylxanthine exposure in the dog.

Calculating the dosage for the newer gourmet chocolate bars on the market is a little different. To calculate the dosage in those cases follow this example. The % cocoa actually refers to the % of chocolate liquor in the bar. Chocolate liquor is cooled into blocks and used as unsweetened baking chocolate. So the % cocoa listed on the label should be multiplied by the value for unsweetened baking chocolate (~400 mg/oz). A 70% bar would have (0.7 X 400) = 280 mg/oz and a 9 kg dog would only have to eat 2 oz = 60 mg/kg.

Management of chocolate exposures includes stabilizing the patient then deciding on decontamination. Emesis may be effective even hours after ingestion since chocolate can form a bezoar in the stomach. Lavage can be considered in cases where emesis is contraindicated, not possible, or has been unsuccessful. For example, lavage is an option if the patient is agitated, seizing or recumbent, or has other health concerns. Lavage is not likely to be as effective as emesis and is associated with potential risks so should not be decided on haphazardly. Activated charcoal is also used frequently in the management of chocolate exposures. Repeated doses may be used due to the extensive enterohepatic recirculation (q 4-6 hr X up to 3 treatments). It is important to monitor serum sodium levels when using activated charcoal due to the risk of hypernatremia developing.

The patient should be monitored including heart rate and rhythm, blood gases and baseline pancreatic enzymes and repeat as needed. Supportive care may include thermoregulation, diazepam, barbiturate,

methocarbamol for tremors/seizures and oxygen as needed for cardiopulmonary compromise. Treat arrhythmias as needed (propranolol). IV fluids are used to enhance excretion and provide CV support. A urinary catheter can be placed to decrease reabsorption across bladder wall or the patient should be walked frequently.

Onions, Garlic, Leeks, Chives (*Allium* Species)

Onions and garlic contains a variety of sulfur-containing compounds especially alk(en)cystein sulfoxides. Ingestion of these compounds can cause oxidative injury to hemoglobin resulting in the precipitation of hemoglobin and the formation of Heinz bodies in the RBC and/or methemoglobinemia. Heinz-body containing RBC's are subject to lysis or removal by the spleen. The result is an anemia that may develop a few days after ingestion of the agent.

Cats are more sensitive to the effects of *Allium* toxicity than dogs. In dogs, the Japanese breeds (such as Shiba Inus and Akitas) may be at higher risk due to an inherited RBC defect. Consumption of as little as 5 g/kg of onions in dogs may result in anemia. Effects may be seen with chronic ingestion of the agents.

Treatment is supportive and symptomatic. Following a large ingestion of the agent, emesis and activated charcoal may be useful. If anemia develops, RBC transfusions should be performed particularly if the animal is showing signs of hypoxia due to anemia.

Avocados

Species sensitivity varies. In dogs, avocados are likely of low toxicity leading to GI upset or obstruction if the pit was ingested. At least one report of cardiac failure in dogs exists in the literature although the reliability of the report cannot be guaranteed. In rabbits, inflammation of the mammary glands may be seen. In some bird species, avocado ingestion can lead to myocardial necrosis and failure with respiratory distress, pericardial effusion, and death. Treatment is symptomatic but the prognosis is poor.

Teflon Pans

Polytetrafluoroethylene (PTFE) is a synthetic polymer used to make Teflon® and Silverstone® non-stick cookware. Over-heating (above 280° C or 530°F) PTFE cookware releases toxic pyrolysis products. While these fumes are toxic to all species, but birds are the most susceptible. In birds, the condition is generally a rapidly fatal one. Signs include acute pulmonary distress with noisy respiration and dyspnea. These signs are shortly followed by "rocking movements," eyelid blinking sometimes described as somnolence, and agonal convulsions (in some cases), followed by death. On necropsy, extensive pulmonary hemorrhage and congestion are found and particles may be seen in some lung sections. Unfortunately in most cases the birds are found dead and there is no time for intervention. If a bird is still alive, it should be removed from the area to prevent further exposure. Symptomatic care including steroids, a warm humid environment, fluids and antibiotics is indicated.

Non-Ionic and Anionic Detergents

Non-ionic and anionic detergents are found in a wide variety of household products, including body and hand soaps, shampoos, dishwashing detergents, various household cleaners, etc. These products are GI and ocular irritants with few to no systemic effects.

Clinical signs consist of hypersalivation, vomiting, and diarrhea, and are generally mild and self-limiting, although ingestion of large quantities may result in more severe vomiting (+/- blood) requiring veterinary intervention. Protracted vomiting may also cause dehydration and electrolyte abnormalities necessitating parenteral fluid therapy. Aspiration of liquid detergent can cause dyspnea and a chemical pneumonitis.

Management includes symptomatic treatment for gastric upset and parenteral fluid therapy, if indicated. Treat ocular exposures by flushing eyes with room temperature water or sterile saline solution for 5 minutes. While corneal injury is unlikely, if persistent photophobia, blepharospasm, or lacrimation should occur, fluorescein stain of the eye should be performed to rule out corneal erosions or ulcers. For respiratory signs, oxygen and other supportive care may be needed.

Ethylene Glycol

Ethylene glycol is most commonly thought of as an automotive radiator antifreeze, but it is also present in high concentrations in many brake fluids and aircraft deicers. In addition, ethylene glycol is often used in condensers, heat exchangers, home solar units and portable basketball goal post bases. Ethylene glycol may also be used to winterize toilets in recreational vehicles and summer homes in colder climates. Ethylene glycol is commonly present as a component in household paints, but it is rarely present in concentrations above 10% and significant ethylene glycol exposure is unlikely unless very large quantities of paint are ingested. Inks, ink pads, polishes, finger moistening compounds (e.g. Tacky Finger®), and other stationery supplies may contain ethylene glycol. Some ink pens contain relatively high levels of ethylene glycol, but the total volume of ink is only a few milliliters, so ink pens would only pose an ethylene glycol risk to very small animals such as birds, pocket pets, or dogs/cats less than 2-3 pounds.

Unfortunately, reliable toxic doses of ethylene glycol have not been established for most animals, including dogs and cats. Much of the acute toxicity data available is based on doses that cause early deaths from acidosis and inebriation and do not take into account the fact that many animals may survive the initial stages of toxicosis only to succumb to kidney failure days later. Because of this, any suspected oral exposure of an animal to radiator antifreeze should be considered a potential toxicosis, and steps should be taken, through historical and diagnostic information, to attempt to determine the extent of the exposure. When doubt still exists, the only prudent course is to treat as if the ingestion was potentially toxic.

It is important to remember that ethylene glycol is a very potent alcohol; for that reason, many of the signs of ethylene glycol toxicosis will relate to severe alcohol intoxication. In addition, ethylene glycol is broken down to metabolites (e.g. oxalic acid) that cause damage to the kidney tubules, resulting in renal failure. Because of the different mechanisms involved in ethylene glycol toxicosis, clinical signs frequently change throughout the course of the toxicosis. The clinical signs can be broken down into 3 different stages, although considerable overlap between these stages may be seen and some animals will not experience each stage. Death can occur at any stage. The stages are 1) neurologic, the initial inebriation due to the effects of alcohol on the CNS, 2) cardiopulmonary, due to severe acidosis and electrolyte disturbances, and 3) renal, due to renal tubular injury from calcium oxalate crystals.

Stage 1: Neurologic. Generally begins within 30 minutes of exposure and lasts up to 12 hours. In mild to moderate cases, this stage may pass quickly and may not be noted by the pet owner or veterinary staff. Animals are initially ataxic, disoriented, “drunk,” stuporous, hypothermic (especially cats), polyuric, and

polydipsic (PD/PU more pronounced in dogs). Coma and death may occur during this stage, or the animal may appear to partially or fully recover over 3-6 hours. By 6-12 hours, the neurologic status of the animal may again deteriorate due to development of severe metabolic acidosis from ethylene glycol metabolites. You may see marked CNS depression, stupor or coma and seizures are possible.

Stage 2: Cardiopulmonary. This stage generally occurs 12 to 24 hours following exposure. Signs may be more recognizable in dogs than cats. Tachypnea, tachycardia, depression, +/- seizures, and pulmonary edema may occur. At this time, a high anion gap and severe metabolic acidosis are generally present.

Stage 3: Oliguric renal failure. This stage can be seen as early as 12 hours, especially in cats, but generally within 24-72 hours following exposure. Clinical signs may include azotemia, depression, anorexia, vomiting, abdominal pain, oral ulcers, and oliguria progressing to anuria. Laboratory findings may include low urine specific gravity, glucosuria and calcium oxalate crystals may be visualized in the urine (please note, absence of crystalluria does NOT rule out the possibility of EG toxicosis). Seizures are possible.

Clinical pathologic abnormalities include increased osmolal gap and anion gap, hyperglycemia, hyperkalemia, decreased blood pH, and hypocalcemia. BUN and creatinine become elevated but usually not before 12 hours post exposure; therefore BUN and creatinine are of minimal benefit in diagnosing early exposures.

Diagnosis is based on history, clinical signs, and confirmatory laboratory testing. There are two in-house ethylene glycol tests presently on the market. Both tests have benefits and disadvantages which will be discussed below. The PRN ethylene glycol and REACT tests are no longer sold. There is a newer test on the market called VetSpec™ which is made by Catachem, Inc. It is very similar to the old PRN ethylene glycol test with the additional benefits of having both a feline and canine control. Cats are much more sensitive to ethylene glycol toxicosis than dogs. The level requiring treatment in cats is > 20 mg/dL while the level for treatment in dogs is > 50 mg/dL. This test will give a false positive with propylene glycol but not methanol, ethanol or isopropyl alcohol and it can be used to determine the length of treatment. Links to information about this test: http://catacheminc.com/tools/data.php?id=134 http://www.catacheminc.com/brochures/Ethylene-Glycol-Flyer.pdf

Another ethylene glycol test, produced by Kacey, Inc. uses a test strip onto which a drop of plasma is deposited and a color change indicates whether the sample contains ethylene glycol. Advantages to this test include ease of use, short time to finish (8 minutes), and separate indicator pads for cats (measuring > 20 mg/dL) and dogs (measuring >50 mg/dL). Disadvantages of the test as it currently exists include it gives a false positive with any alcohol (glycerol, sorbitol, mannitol, formaldehyde, methanol, ethanol, etc) and since the color change involves green dyes, people with red-green color blindness might not be able to distinguish color changes.

Other means of diagnosing ethylene glycol exposure in pets include having levels run at a human hospital on a STAT basis. Many human hospitals are willing to do this, although sometimes it takes talking to the laboratory technician rather than a receptionist. Levels of 50 mg/dl (or 5 µg/ml, be sure to check the units reported) or greater in dogs would be considered significant. In cats, any level above zero should be considered significant. Measuring anion gap (>25 mEq/L) or serum osmolality (> 20 mOsm/kg) may assist in diagnosing ethylene glycol toxicosis. Observation, via Wood's lamp, of fluorescence in urine, stomach contents or on paws/muzzle may suggest exposure (fluorescein dye is added to automotive antifreeze to help in detecting radiator leaks).

Treatment of ethylene glycol toxicosis must be timely and aggressive. Failure to institute appropriate therapy within the first several hours may result in irreversible renal damage or death of the animal. For recent (within 45 minutes) exposures and asymptomatic animals, induce vomiting or perform gastric lavage; because food in the stomach may slow absorption, emesis or lavage may be of benefit up to 1 hour in animals that have recently eaten. The use of activated charcoal is not likely to be useful since aliphatic alcohols are not well adsorbed by charcoal. Additionally, activated charcoal products can interfere with in-house tests and the animals may have significant CNS depression and nausea putting them at risk for aspiration. Based on exposure history and/or diagnostic test results, the use of either fomepizole or ethanol infusion (see below) may be indicated.

Symptomatic animals should be stabilized as needed. Seizures can be controlled with diazepam or barbiturates, but care must be taken to minimize any further CNS depression. Intravenous fluids are the cornerstone of treatment, especially in symptomatic animals. High infusion rates of crystalloids are necessary to correct dehydration and hypoperfusion; fluid ins and outs should be monitored to avoid fluid overload and possibly pulmonary edema. Treatment of acidosis and renal failure may be required. Oliguric or anuric animals may require peritoneal dialysis.

Intravenous ethanol and, more recently fomepizole (4-MP, 4-methylpyrazole, Antizol-Vet™), have been used successfully in the management of ethylene glycol toxicosis in animals and man. The primary goal of using these compounds is to delay the breakdown of ethylene glycol to its more toxic metabolites, allowing the parent compound to be excreted in the urine unchanged. Best results with either of these treatments require initiation of treatment as soon as possible following ingestion, preferably within the first 6-8 hours for dogs and first 3 hours for cats.

Ethanol has the advantages of being inexpensive and readily available, but it has some serious drawbacks, including worsening of metabolic acidosis and CNS depression, making evaluation of the degree of ethylene glycol toxicosis difficult. Additionally, ethanol treatments are time-intensive and require constant patient monitoring because of the severe side effects. Ethanol can be used in both cats and dogs. The preferred treatment regime is to administer 8.6 ml/kg (600 mg/kg) of a 7% (70 mg/ml) ethanol solution and then maintain at 1.43 ml/kg/hr (100 mg/kg/hour), up to 200 mg/kg/hr as a constant rate infusion. The animal must be constantly monitored and the dosage adjusted to prevent severe respiratory depression and acidosis. The other method of ethylene glycol treatment would be to make a 20% ethanol solution. Dogs are given 5.5 ml/kg every 4 hours for 5 treatments then every 6 hours for 4 treatments. Cats are given 5.0 ml/kg every 6 hours for 5 treatments, then every 8 hours for 4 treatments.

Fomepizole will not cause hyperosmolality or metabolic acidosis. In contrast to ethanol, which is administered every 4 hours or as a constant-rate infusion, fomepizole is administered every 12 hours for 36 hours. The initial dosage in dogs is 20 mg/kg (slow IV over 15-30 minutes), then 15mg/kg (slow IV) at 12 and 24 hours, and then 5mg/kg is given at 36 hours. Fomepizole is not expected to cause sedation in dogs. The main drawbacks with fomepizole are the cost of the medication and the fact that in 2015 the FDA withdrew approval for Antizol-Vet™ so now it must be compounded. In addition, fomepizole is only useful in cats if given within 3 hours of exposure. In cats, the dosage is 125 mg/kg initially, followed by 31.25 mg/kg at 12, 24 and 36 hours. Sedation is expected with this protocol in cats but it is expected to produce better results than ethanol treatment in cats. In one study, fomepizole used within first 3 hrs of administration of a lethal ethylene glycol dose resulted in a 75% recovery rate (versus a 25% recovery

rate with ethanol). At 4 hrs post ethylene glycol dosing, there was 100% mortality with fomepizole and ethanol.

Treatment should be continued until the patient is clinically normal and has had at least 24 hours with normal renal function and acid base parameters. The VetSpec™ test can be used to determine when all of the parent ethylene glycol compound has been excreted and fomepizole or ethanol can be discontinued. The prognosis for recovery depends on degree of exposure, length of time between exposure and treatment, and aggressiveness of treatment. Surviving animals may fully recover or may have residual renal insufficiency requiring lifetime maintenance. The presence of oliguria/anuria indicates a grave prognosis.

Grill Cleaners/Pool Hazards

Many grill cleaners contain alkaline corrosive ingredients including potassium hydroxide and sodium hydroxide. Pool products can also contain alkaline ingredients but more commonly contain acid corrosive ingredients. Acids produce pain on contact with sensitive tissues because of coagulative necrosis. This pain usually causes the animal to stop chewing on an acid. Alkaline corrosives result in liquefaction necrosis and don't cause immediate pain so the exposure may be more serious. In both cases, monitor for 12 – 24 hours for oral and GI ulceration. Attempts to chemically neutralize an acid or alkali agent with a weak alkali or acid, respectively, are contraindicated, as this may stimulate an exothermic reaction that will exacerbate tissue injury. Treatment of oral exposure includes immediate dilution with water or milk. Induction of emesis is contraindicated due to the risk of increasing corrosive injury. Activated charcoal is ineffective for caustic agents and should not be used. Feeding soft food for a few days post exposure may prevent worsening of the irritation. Treatment of oral lesions is symptomatic, and should include antibiotics to prevent infection; pain management (opioids), sucralfate slurries to treat oral, esophageal or gastric ulcers; intravenous fluids to maintain hydration; and provision for nutritional support (e.g. gastrostomy tube). The use of corticosteroids to decrease inflammation and esophageal stricture formation is controversial, as steroids will delay wound healing and may increase susceptibility to infection.

Fertilizers and Herbicides

Fertilizers contain salts of nitrogen, phosphorus and potassium (N-P-K). In most exposures, they are GI irritants. Be sure to check for added iron, insecticides or pesticides which could increase the toxic potential. If the iron level >5%, more significant effects may occur. "Organic" Fertilizer/Bone Meal/Blood Meal are very attractive to dogs and cats. If the product is moldy or rancid, bacterial gastroenteritis or tremorgenic mycotoxins are a concern. There is also a risk of possible impaction if a large amount is ingested. Herbicides, in general, do not cause severe systemic signs when the cat or dog has access to an appropriately treated yard. You may note mild GI upset which is less likely after the product has dried.

Fireflies

Fireflies contain cardiac glycosides which are structurally similar to the toxins in certain plants (Foxglove, Oleander, squill) and in Bufo toads. Lizards fed fireflies have developed head shaking, oral gaping, dyspnea, color changes and sudden death. No reported patient has survived long enough to reach a veterinarian for treatment. Reptile owners should be informed of the dangers of feeding fireflies.

Anticholinesterase Insecticides

Carbamates and Organophosphates (OP) are both anticholinesterase insecticides. They cause their effects by binding to the enzyme acetylcholinesterase and competitively inhibiting it. The neurotransmitter acetylcholine is normally catabolized by acetylcholinesterase. When the enzyme is inhibited, acetylcholine accumulates and overstimulates muscarinic and nicotinic receptors in the nervous system. Organophosphosphates and carbamates differ in their affinity for acetylcholinesterase. OPs bind to acetylcholinesterase irreversibly in a process called aging whereas carbamates bind reversibly. There are many different insecticides in this group. Some common OPs are chlorpyrifos, diazinon, and disulfoton. Some common carbamates are methomyl, propoxur and carbaryl.

Clinical signs of intoxication are grouped by which receptors are involved. Overstimulation of muscarinic receptors leads to the SLUDDE signs: salivation, lacrimation, urination, defecation, dyspnea and emesis. Miosis and bradycardia are also common. The dyspnea is due to increased respiratory secretions and bronchoconstriction. Overstimulation of nicotinic receptors leads to tremors, paresis, seizures, coma and death in some cases.

The toxicity of these agents varies widely with some causing death in seconds while others rarely cause problems. In cases of suspected anticholinesterase poisoning, an atropine test dose is used to confirm the diagnosis. A preanesthetic dose (0.02 mg/kg IV) of atropine is given. If the muscarinic signs improve then the patient was **not** poisoned by an OP or carbamate since it takes a very high dose of atropine (about 10X) to compete with the excess acetylcholine at the receptors.

Consider emesis (only if asymptomatic) and activated charcoal in patients with a large oral exposure. Bathe with liquid dishwashing detergent if the exposure was topical. If significant muscarinic signs are present, including bradycardia or copious respiratory secretions, give atropine (0.1 - 0.2 mg/kg for dogs and cats). If the patient is only drooling, do not give atropine. Valium or barbiturates can be given for seizures and methocarbamol for tremors. Provide ventilatory support and oxygen when needed. Pralidoxime (2-PAM) is antidotal since it frees the OP from the enzyme acetylcholinesterase. 2-PAM should be used with OPs only since carbamate binding is reversible.

Mulches

The biggest concern with ingestion of most mulch is mechanical GI irritation and possible obstruction. Mulch that is moldy can contain tremorgenic mycotoxins. Cocoa mulch can cause more serious effects due to the methylxanthines caffeine and theobromine in the mulch. The total methylxanthine content in cocoa mulch is estimated to be 0.25% - 3.87%. Methylxanthines are alkaloids that occur naturally in plants and are also found in tea, coffee beans and cola beans.

Methylxanthines are rapidly absorbed by mouth and metabolized in the liver. Elimination is via the bile and/or urine and varies with the age and species of the patient. Methylxanthines undergo enterohepatic recirculation and can be reabsorbed via the bladder wall. These characteristics will guide decontamination recommendations (see below).

Methylxanthines work by blocking adenosine receptors (adenosine is a bronchoconstrictor, anticonvulsant, and regulates heart rhythm), by increased calcium movement into cells and by

increasing cAMP via inhibition of phosphodiesterase. These actions show clinically as stimulation of the CNS and cardiovascular systems.

In dogs, the following methylxanthine dosages are expected to show the following clinical effects. **20 mg/kg**: mild signs (vomiting, hyperactivity, PD). **40-50 mg/kg**: moderate to severe signs (cardiotoxicity). **60 mg/kg**: seizures possible. And **100 mg/kg** is the minimum lethal dosage.

Clinical signs usually develop within 6-12 hours of ingestion and can include polydipsia, vomiting, diarrhea, bloating, restlessness, hyperactivity (progressing to ataxia, tremors and seizures), cardiac effects (PVCs, tachycardia), tachypnea, hypertension, hyperthermia and coma.

Management of cocoa mulch exposures includes stabilizing the patient then deciding on decontamination. Lavage can be considered in cases where emesis is contraindicated, not possible or has been unsuccessful. For example, lavage is an option if the patient is agitated, seizing or recumbent or has other health concerns. Lavage is not likely to be as effective as emesis and is associated with potential risks so should not be decided on haphazardly. Activated charcoal is also used frequently in the management of cocoa mulch exposures. Repeated doses may be used due to the extensive enterohepatic recirculation (q 4-6 hr X up to 3 treatments). It is important to monitor serum sodium levels when using activated charcoal due to the risk of hypernatremia developing.

The patient should be monitored including heart rate and rhythm, blood gases and baseline pancreatic enzymes and repeat as needed. Supportive care may include thermoregulation, diazepam, barbiturate, methocarbamol for tremors/seizures and oxygen as needed for cardiopulmonary compromise. Treat arrhythmias as needed (propranolol). IV fluids are used to enhance excretion and provide CV support. A urinary catheter can be placed to decrease reabsorption of the methylxanthines across bladder wall or the patient should be walked frequently.

Metaldehyde

Many snail and slug baits contain metaldehyde. These products also contain ingredients such as molasses or bran to attract snails and slugs. Dogs are attracted to these ingredients and readily ingest the bait. Metaldehyde is highly toxic: a 10-pound dog only needs to ingest 1 teaspoon of 2% metaldehyde bait to be at risk for significant toxicosis.

Metaldehyde's mechanism of action is not fully understood, but may involve depletion of neurotransmitters such as GABA and serotonin in the CNS. Signs can occur within several minutes to a few hours following ingestion. In dogs, clinical signs can include anxiety, agitation, drooling, ataxia, stiffness and rigidity, tremors, seizures, hyperthermia and potentially death. Liver failure may develop within 2-3 days of exposure.

Treatment of metaldehyde exposures includes decontamination in asymptomatic animals, and symptomatic management of clinical signs. Methocarbamol is preferred for managing tremors. Electrolyte and acid/base should be monitored and abnormalities corrected as needed. Hyperthermia frequently resolves once tremors and seizures are under control, but external cooling measures may be required. Liver and renal values should be monitored for up to 72 hours following exposure.

Glow Jewelry

Glow-in-the-dark items are popular novelties that are sold at fairs, carnivals, novelty stores and skating arenas; they are most popular around the 4th of July and Halloween holidays. These items include glow-sticks and glow-in-the-dark jewelry (necklaces, bracelets, etc). The primary luminescent agent in these types of products is dibutyl phthalate (n-butyl phthalate), an oily liquid that is also used as a plasticizer and insect repellent. Dibutyl phthalate is of relatively low toxicity (LD_{50} >8000 mg/kg in rats). Pet exposures to glow-in-the-dark items are unlikely to cause serious problems due to the low toxicity, the extremely unpleasant taste and the small amounts of dibutyl phthalate in these types of items.

Exposures generally occur when cats bite into glow-sticks or jewelry. The extremely unpleasant taste of dibutyl phthalate is thought to be responsible for the clinical signs seen and to limit exposure to these items. Signs generally occur within seconds of the cat biting into the item, and cats will often have a much exaggerated reaction to the taste of dibutyl phthalate. They may display profuse salivation and foaming, with occasional retching and/or vomiting. More dramatic are the behavioral effects in cats from exposure to glow items, with neurological signs such as hyperactivity, aggression, head shaking, hiding, and agitation being reported. Rarely, transient panting, dyspnea, tremors and urinary incontinence have been reported in cats.

In spite of their initial intensity, signs from these items are generally self-limiting and should resolve once the cat gets the taste of the product out of its mouth. The goal of managing an exposure to glow items is to dilute the taste using milk or highly palatable food (e.g. canned tuna). Any chemical that has gotten on skin or fur should be bathed off to prevent re-exposure should the cat groom itself; taking the pet into a darkened room will aid in identifying the luminescent chemical on the skin or coat. For ocular exposure, copious flushing of the eyes is recommended. In most cases, once the disagreeable taste is dealt with, cats will return to normal with no further treatment needed.

Plants

In general, even plants considered “non-toxic” may cause mild GI upset if ingested therefore the owner should be instructed to watch for GI effects. When faced with an exposure to a plant, it is important to obtain the genus and species names whenever possible since many plants share similar sounding common names but the toxic potential may be quite different. Animals ingesting water in the reservoir of a houseplant may also ingest the toxic principle of plant (e.g. lilies or cardiac glycosides).

Lily

Easter lilies (*Lilium longiflorum*), tiger lilies (*Lilium tigrinum*), rubrum or Japanese showy lilies (*Lilium speciosum* and *Lilium lancifolium*), and various day lilies (*Hemerocallis* species) have been incriminated in causing acute renal failure and death in cats. The toxic principle is unknown. Even minor exposures (a few bites on a leaf, ingestion of pollen, etc.) may result in toxicosis, so all feline exposures to lilies should be considered potentially life-threatening and merit aggressive clinical intervention. It should be noted that not all plants with “lily” in the name are members of Liliaceae, e.g. calla lily (*Zantedeschia* spp. which are oxalate-containing plants) or lily of the valley (*Convallaria* spp., which contain cardiac glycosides).

Affected cats often vomit within a few hours of exposure to lilies, but the vomiting usually subsides after a few hours, during which time the cats may appear normal or may be mildly depressed and anorexic. Within 24 to 72 hours of ingestion, oliguric to anuric renal failure develops, accompanied by vomiting,

depression, anorexia, dehydration, and hypothermia; additionally, disorientation, ataxia, facial and paw edema, dyspnea, and seizures have been less commonly reported.

Elevations in blood urea nitrogen (BUN), creatinine, phosphorus and potassium are detectable as early as 12 hours post ingestion. Creatinine elevations may be especially striking, with levels as high as 44 mg/dl reported. In some cases, hypoglycemia and mild liver enzyme elevations may occur. Pancreatitis may also develop. Abundant casts, proteinuria, glucosuria, and isosthenuria are usually detectable on urinalysis within 24 hours of ingestion, reflecting lily-induced necrosis of promixal renal tubular epithelial cells. In severe cases, death or euthanasia due to acute renal failure generally occurs within 3 to 6 days of ingestion.

When initiated within 18 hours of ingestion, decontamination (emesis, oral activated charcoal, and cathartic) and fluid diuresis at twice maintenance infusion rate for 48 hours have been effective in preventing lily-induced acute renal failure. Conversely, delaying treatment beyond 18 hours frequently results in death or euthanasia due to severe renal failure. Baseline renal values should be obtained upon presentation and then repeated at 12, 24, and potentially 48 hours.

Because the tubular injury from lily ingestion spares the renal tubular basement membrane in many cases, regeneration of damaged tubules may be possible. In severe cases, peritoneal dialysis may aid in managing renal failure until tubular regeneration occurs (10-14 days or longer).

Cycads

Cycads are commonly known as Sago palms, Coontie palms and Cardboard palms (*Zamia floridana, Cycas revolute, Macrozamia* spp.). These large, attractive evergreen plants are commonly found in yards in warmer climates like in Florida, North Carolina and Georgia. Recently a smaller (bonsai-like) version of the plant is being sold as a houseplant all over the country. All parts of the plant are toxic but the seeds (nuts) contain the highest concentration of the toxic principle, cycasin. GI, hepatic and CNS effects are possible. All species are at risk.

Since intoxication is often life-threatening, all exposures to cycads should be considered serious and merit aggressive clinical intervention. Gastrointestinal upset usually develops within 24 hours whereas laboratory abnormalities may not develop for 24 – 48 hours. Vomiting, depression, diarrhea and anorexia are the most common signs in dogs following ingestion of a cycad. Elevations in bilirubin, serum alanine aminotransferase and alkaline phosphatase are also common due to liver necrosis. Hematemesis, seizures and coagulopathies are possible. Signs usually continue for 24 – 48 hours but can be prolonged for longer than one week.

Induction of emesis is appropriate for asymptomatic patients that have recently ingested the cycad. Once the vomiting is controlled, give one dosage of activated charcoal. Repeated doses of activated charcoal may be beneficial in large ingestions. A baseline chemistry panel and CBC should be done and liver values monitored daily for 48 hours. In patients that have GI effects, control vomiting with emetics and begin GI protective medications such as a proton-pump inhibitor and sucralfate. Fluid therapy with 5% dextrose in water, since this may be liver-protective, should be started. Begin liver-protective medications such as SAMe, Marin or Denamarin and manage any seizures that develop with diazepam. Treat any coagulopathies as needed.

The prognosis is good if treatment is instituted soon after ingestion before clinical signs develop. Symptomatic patients have a guarded prognosis and the reported mortality rate in dogs with clinical signs is greater than 30%.

Grayanotoxin-Containing Plants

Azalea, rhododendron (*Rhododendron* spp.), Laurel (*Kamia* spp.), Japanese pieris (*Pieris japonica*) and Labrador tea (*Ledum glandulosum*) contain grayanotoxins which are found in all parts of the plants. These plants have a wide distribution throughout the United States and most species are susceptible to poisoning by these plants.

Grayanotoxins cause GI, neurologic and cardiovascular dysfunction and act by modulating the gating kinetics of sodium channels in cell membranes, resulting in prolonged depolarization of nervous tissue. The most common sign seen in pets is GI upset including vomiting, diarrhea, drooling, abdominal pain and anorexia. More serious signs including cardiac arrhythmias or arrest, hypotension, weakness, pulmonary edema, dyspnea, CNS depression, limb paralysis, seizures and coma can occur but are not typical. Clinical signs can develop very soon after ingestion up to 12 hrs post ingestion and may persist 1 to 2 days.

For asymptomatic patients, consider emesis. Very large ingestions may necessitate gastric lavage or a gastrotomy. Activated charcoal can be used in addition to or instead of emesis or lavage. The patient should be monitored for neurologic and cardiovascular effects. GI upset should be managed with standard anti-emetics. IV fluids should be given as needed and cardiac arrhythmias should be managed as appropriate. Diazepam can be used for seizures.

Cardiac Glycoside Plants

Plants containing cardiac glycosides are widely distributed throughout the United States and include Foxglove (*Digitalis purpurea*), Oleander (*Nerium oleander*), Lily-of-the-valley (*Convallaria majalis*), Milkweeds (*Aesclepias* spp), Kalanchoe (*Kalanchoe* spp.), and Squill (*Urginea maritima*). Cardiac glycosides interfere with the sodium/potassium ATPase enzymes throughout the body resulting in an elevation of the intracellular sodium. The elevated sodium is exchanged for calcium, elevating the intracellular free calcium. This leads to increased calcium-dependent contraction of the cardiac muscle cells. Cardiac glycosides also decrease the intracellular potassium concentrations which affects the resting membrane potential.

All parts of plant (green or dry) are toxic and most species are susceptible to poisoning by these plants. Signs develop within 6-12 hours of ingestion and commonly include vomiting, diarrhea and abdominal pain. Weakness, cardiac arrhythmias, hypotension, dyspnea, tremors, ataxia, coma and death are possible. Serum potassium may increase or decrease.

Treatment should include decontamination by emesis or lavage and/or activated charcoal. Monitor for GI, CV and CNS effects and begin IV fluid therapy. Manage GI signs and arrhythmias if they develop. Bradyarrhythmias can be treated with atropine and tachyarrhythmias with lidocaine or phenytoin. Treatment should continue until all signs resolve and the prognosis depends on the severity of the signs that develop. Digibind® can be used as an antidote to bind to and remove cardiac glycosides from the systemic circulation but it can be expensive and difficult to find.

Ornamental Bulbs

Most ornamental bulbs are in the family Amaryllidaceae and include daffodils, jonquils, Clivia lilys, Snowdrop, Barbadoes lily and amaryllis. Iris and tulips are also ornamental bulbs and cause similar clinical signs. These plants contain a number of alkaloids like lycorine and galanthamine. Lycorine is the most important toxic alkaloid. In most cases, bulbs cause GI upset, lethargy and anorexia. In some cases, hypotension with resultant weakness and ataxia may develop. Large quantities of foliage or bulb ingestion can cause muscle tremors and seizures. Signs usually develop within a few hours and can last 2-3 days. If a bulb is swallowed whole, an obstruction may occur. If the bulbs are moldy, there is the potential that tremorgenic mycotoxins are present and may cause clinical effects.

Autumn crocus (*Colchicum autumnale*) is another ornamental bulb that is much more dangerous. It contains colchicine which is used as an anti-metabolite and can cause severe clinical signs including shock, seizures and death.

Castor Bean

The Castor Bean plant (*Ricinus communis*) grows in the temperate areas of the United States. It is an attractive plant and is used for landscaping. Castor oil is obtained from the plant and doesn't contain the toxic principle which is found in the seeds. Castor oil is used in the manufacture of soaps, lubricants, brake fluids, paints and dyes. The seeds, which are also attractive and are used in folk jewelry, contain one of most deadly toxins known – a lectin called ricin. Ricin was used in the 1978 KGB assassination of Bulgarian defector and BBC World Service commentator, Georgi Markov. In a fashion befitting a Hollywood spy movie, a poison pellet containing ricin was stabbed into his thigh with the tip of an umbrella. Three days later he died of cardiac failure, having been afflicted with a host of other ailments.

All species are susceptible to ricin poisoning. Ricin degrades rRNA, preventing protein synthesis leading to cell death. Clinical signs include vomiting and diarrhea (+/- blood), depression, abdominal pain, anorexia and liver +/- renal injury. Treatment should include decontamination by emesis or lavage and/or activated charcoal. Begin GI protectant medications and pull a baseline CBC and serum biochemistry. Begin fluid therapy. Seizures can be controlled with diazepam. Laboratory alterations may take 12-24 hrs to develop. Monitor the patient until levels return to normal and clinical signs resolve. Manage hepatic injury or renal failure by standard means. The prognosis good with prompt and aggressive care: the mortality rate is approximately 9%. These patients may require short- to long-term management of hepatic and/or renal compromise.

Precatory bean (*Abrus precatorius*) also contains lectins. These grow in the warm regions of the Americas. The toxic principle is abrin. Abrin is thought to be even more toxic than ricin.

Yew

Japanese yew (*Taxus cuspidate*), English yew (*Taxus baccata*) and Chinese yew (*Taxus chinensis*) are some of the most toxic plants in the United States and are commonly found in household landscapes. Pacific or western yew (*Taxus brevifolia*) is less toxic then the others. All parts of the plant, except the aril (the fleshy red seed covering) contain the toxic taxine alkaloids. Dried plant material is also very toxic. A dog playing with a stick could get exposed to enough of the toxic principle to lead to death.

The taxine alkaloids are cardiotoxic and act by blocking myocyte calcium channels. GI upset may also occur from the volatile irritants in the plant and CNS excitation has also been reported. The mechanism of the CNS effects is unknown.

Sudden death is often the only clinical sign associated with yew ingestion since the plant is highly toxic and signs can occur rapidly. Trembling, tremors, dyspnea, nausea, hypotension, vomiting and diarrhea may also occur. ECG findings in intoxicated patients may include bradycardia, absent p waves and an increase in QRS duration.

Treatment should include decontamination by emesis or lavage and/or activated charcoal and monitoring for GI, CV and CNS effects. If signs develop, treatment is symptomatic and supportive and may include atropine for bradycardia, fluid therapy to manage hypotension, GI protectants and antiemetics and diazepam to control tremors. Prevention and education of owners is critical since yew poisoning has a very guarded prognosis. Yew branches should never be used as play sticks for dogs and owners should dispose of yew trimmings in an area where their pets do not have access to them.

Insoluble Calcium Oxalate-Containing Plants

This group of plants is large and most are in the Araceae family. They are common household plants. Some of the common members are Elephant's Ear (*Colocasia* spp.), Flamingo Plant (*Anthurium* spp.), Jack-in-the-pulpit (*Arisaema triphyllum)*, Cuckoo-pint (*Arum* spp.), *Caladium* spp., Dumb cane (*Dieffenbachia* spp.), pothos (*Epipremnum* spp.) and *Philodendron* spp.

These plants contain sharp, needle-shaped calcium oxalate crystals called raphides which reside in idoblast cells in the leaves. When the leaves are chewed, bruised or otherwise damaged, the idoblasts swell then break open and shoot the raphides out. The raphides lodge into the oral mucosa causing oral irritation, drooling, gagging and vomiting. These signs are usually mild and self-limiting but in significant ingestions can cause stomatitis, pharyngeal edema, gastritis and enteritis.

Milk or yogurt may aid in easing discomfort since the calcium in those products will bind to the crystals and help pull them from the mucosal tissues. GI protectants and antiemetics may also be needed.

Saponin-Containing Plants

Dracaena spp*.,* including corn plant, dragon tree, money tree and lucky bamboo, contain steroidal saponins and glycosides which are soapy and bitter-tasting and irritating to the GI tract. All parts of these plants are toxic. The most common clinical signs following ingestion are vomiting (occasionally with blood), hypersalivation, anorexia, depression and ataxia. Cats may also develop mydriasis, dyspnea and tachycardia. Most animals recover without treatment.

Sansevieria spp. (common names: mother-in-law's tongue and snake plant) also contain saponins and can cause similar signs. *Aloe* spp. (aloe vera) contains saponins in their white sap so can lead to GI upset. The gel from this plant is non-toxic and is used medicinally for burns. English ivy (*Hedera helix*) is used as a ground cover or house plant. It also contains a saponin called hederagenin which can lead to GI upset. All parts of the plant are toxic.

Pokeweed (*Phytolacca americana*) also contains saponins. Ingestion can cause severe gastroenteritis. The roots are the most toxic portion. Young shoots and leaves can be eaten after 2-3 changes of boiled

water (poke salad). The ripe berries (with the seeds removed) can be used to make jams and jellies. The berries contain a red dye that was used by Native Americans to decorate their horses and the United States Constitution is written in pokeberry ink.

Hops

Hops (*Humulus lupulus)* cause a Malignant Hyperthermia-like syndrome in dogs. Plant material, and hops used for brewing, either unused or spent, can cause clinical signs. The signs develop within 3 hours and include tachypnea, severe hyperthermia (108°F +) and death within 6 hours.

The toxic principle is unknown. Decontamination by emesis is indicated within 1 hour of exposure if patient is asymptomatic. Activated charcoal can be useful. Dantrolene (direct-acting skeletal muscle relaxant) can be used to control the hyperthermia. Cyproheptadine may also be helpful. Treatment is otherwise symptomatic and supportive including IV fluids and diazepam for tremors. The prognosis is guarded to poor.

Soluble Oxalate-Containing Plants

The shamrock plant (*Oxalis* spp.), a common household plant sold around St. Patrick's Day and rhubarb (*Rheum rhaponticum*) contain soluble oxalates. The entire shamrock plant is toxic whereas only the leaves and roots of rhubarb are toxic whereas the stems are edible.

The soluble oxalates are absorbed into the systemic circulation from the GI tract and combine with the calcium in blood leading to severe hypocalcemia. The calcium oxalate crystals that are formed then concentrate in the renal tubules leading to renal failure.

Emesis is indicated for recent exposures. Administration of yogurt or milk immediately after the ingestion of soluble oxalate containing plants has shown to help decrease the amount of oxalates that are absorbed by changing them into insoluble calcium oxalate crystals. Monitor for hypocalcemia and correct with IV calcium gluconate as needed. IV fluid diuresis is indicated to prevent acute renal failure. Hypocalcemia is expected within 2 – 12 hours and renal effects within 24 – 48 hours.

***Brunfelsia* spp.**

This group includes *Brunfelsia americana* (Lady of the Night), *Brunfelsia australis* (Paraguay jasmine, Morning-Noon-and-Night plant, Yesterday-Today-and-Tomorrow Plant), *Brunfelsia grandiflora*, *Brunfelsia lantifolia* (Kiss-Me-Quick), *Brunfelsia pauciflora* (Yesterday-Today-and-Tomorrow Plant, Morning-Noon-and-Night, Yesterday-and-Today). These are outdoor evergreen shrubs found in temperate areas including California, coastal Texas, and Florida. These can also be grown in planters in more northerly areas.

All parts of the plant are toxic but the fruit is the most toxic part. The plants contain multiple toxins including brunfelsamidine, hopeanine which cause tremors and seizures and scopoletin, a smooth muscle relaxant. Clinical signs include seizures, extensor rigidity similar to strychnine, tremors, diarrhea, ataxia, and vomiting. The onset of action is minutes to hours and the prognosis is guarded once neurologic signs appear. The tremors may last for weeks after exposure.

Emesis is not recommended following ingestion due to rapid onset of clinical signs. Activated charcoal can be used in an attempt to prevent absorption of the toxins. Control tremors with methocarbamol and seizures with valium or propofol. Minimizing external stimuli (sound and light) may reduce the incidence and severity of seizures. IV fluids to maintain urine output and prevent damage to renal tubules from myoglobinuria or acidosis. Thermoregulation is important due to the risk of hyperthermia from tremors and seizures.