

COMMON HOUSEHOLD HAZARDS

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“The Good”

Desiccants

Desiccant packs are included as moisture absorbents. They are found in shoeboxes, electronics, medications and food. Silica gel, one of the most common desiccants, is a white powder or a lustrous granule. Silica gel comes in paper packets or plastic cylinders. Packages of silica gel are attractive to pets because of the rustling noise, and the packages are easy to bat around. Most ingestions will not cause clinical signs, although a mild gastrointestinal upset may occur. If a large amount is ingested, there is potential for osmotic diarrhea occurring. Ingestion of the intact packet may cause a gastrointestinal obstruction.

Food products often contain desiccants composed of iron. Deli meats, pepperoni, etc are likely to have this type of desiccant. The iron content can range from 30-60%. Once the iron has oxidized, the resulting compound (iron oxide) is inert and non-toxic. Vomiting is very common.

Ant and Roach Baits

Ant and roach baits are common objects found in households. They are also referred to as hotels, traps, or stations. The insecticides used most commonly in these baits are sulfluramid (n-ethylperfluorooctanesulfonamide), fipronil, avermectin, boric acid, and hydramethylnon, all of which are of low mammalian toxicity and present in very low concentrations within the baits. The baits also contain inert ingredients such as peanut butter, breadcrumbs, fats and sugar to attract the insects; these agents are also sometimes attractive to pets. Exposures of pets to these types of ant baits usually do not require decontamination or treatment. Most often, if signs are seen at all, they are mild in nature and self-limiting and consist of vomiting attributed to the inert ingredients rather than the active ingredient. Ingestion of multiple avermectin-based ant baits in a small dog, or ingestion of single ant baits containing arsenic trioxide (rarely available) would be cause for decontamination and monitoring.

Birth Control Pills

Birth control pills generally come in 28 tablet packs with 21 hormone tablets (estrogen +/- progesterone) and 7 placebo tablets. Most hormone pills contain 0.035 mg of estrogen or less. In general, estrogen doses of less than 1 mg/kg are not of concern. At higher doses, bone marrow suppression may be seen. However, due to the low

estrogen content of the pills, estrogen exposure is generally not sufficient to require treatment. Some placebos may contain an iron supplement; elemental iron doses of >20 mg/kg may require decontamination and other treatments.

Non-ionic and Anionic Detergents

Non-ionic and anionic detergents are found in a wide variety of household products, including body soaps, shampoos, dishwashing detergents, various household cleaners, etc. These products are gastrointestinal 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.

Toilet Water (Tank Drop-Ins)

Tank "drop in" products typically contain anionic/nonionic detergents, cationic detergents, bleach, and/or acids. However, when a tank "drop in" cleaning product is used in a toilet, the actual concentration of the cleaner is very low in the bowl. With dilution by the bowl water, the cleaning agent is just a gastric irritant. Common signs seen with ingestion include mild vomiting and nausea

Glow-in-the-dark Sticks and Jewelry

Glow-in-the-dark items (glo-sticks, necklaces) are popular novelty items that are sold at fairs, carnivals, novelty stores and skating arenas. 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 low toxicity (LD₅₀ >8000 mg/kg in rats) so serious problems are unlikely. Even though the extremely unpleasant taste of dibutyl phthalate may limit exposure, some very dramatic signs may be seen. Signs generally occur within seconds of the pet biting into the item. Compared to dogs, cats tend to have a much more exaggerated reaction to the taste of dibutyl phthalate. Cats may display profuse salivation and foaming, with occasional retching and/or vomiting. In all cases, signs are generally self-limiting and should resolve once the pet gets the taste of the product out of their mouth. The exposure is managed by diluting the taste of the dibutyl phthalate using milk or highly palatable food (e.g. canned tuna). Any chemical that has gotten on skin or fur should be bathed or wiped off to prevent re-exposure when the animal grooms themselves; taking the pet into a darkened room will aid in identifying the luminescent chemical on the skin or coat.

“The Bad”

Acids

Products containing acids include cleaning agents (e.g. toilet bowl cleaners), anti-rust compounds, etching compounds, automotive batteries, and pool sanitizers. The relative toxicity of an acid is related to its concentration and decreases with dilution. Acids produce localized coagulative necrosis of tissue and generally produce immediate pain upon exposure, which helps to limit ingestion. In most cases, clinical signs occur almost immediately upon exposure. Oral exposure results in oral pain, vocalization, dysphagia, vomiting (+/- blood), abdominal pain, and irritation or ulceration of oral and/or esophageal mucosa. Lesions often appear milky white to gray initially, then gradually turn black. Esophageal lesions are less common than with alkaline products. With high levels of exposure, gastric ulceration is also possible. Dermal exposure results in dermal irritation or ulceration, accompanied by intense local pain. Inhalation of acid fumes may result in dyspnea, pulmonary edema, tracheobronchitis or pneumonitis. Ocular exposure may result in corneal erosion or ulceration.

Attempts to chemically neutralize with a weak alkali 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. Gastric lavage and induction of emesis are contraindicated due to the risk of increasing corrosive injury. Activated charcoal is ineffective for caustic agents and should not be used. Treatment of oral lesions is symptomatic, and should include: antibiotics to prevent infection; pain management (butorphanol); 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. Dermal exposures should be treated with copious flushing with clear water for 15 minutes. For ocular exposures, eyes should be flushed with room temperature water or sterile saline solution for 15 minutes. Fluorescein staining of the eyes should be performed, and corneal erosion or ulceration should be treated as needed. Animals with significant respiratory signs (coughing, dyspnea, etc.) should be monitored for a minimum of 24 hours for the development of pulmonary edema. Supplemental oxygen or other respiratory supportive care should be used as needed.

Alkalis

Alkaline products include sodium or potassium hydroxide, ammonium hydroxide, and potassium permanganate. Common sources of alkaline products include drain openers, automatic dishwasher detergents, alkaline batteries, toilet bowl cleaners, swimming pool products and radiator cleaning agents. Agents with pH greater than 11

should be considered to be capable of causing significant corrosive injury. Alkaline agents penetrate local tissue rapidly and deeply, causing liquefactive necrosis. Unlike acidic products, very little pain may be evident upon initial contact with an alkaline product, which may encourage further contact and ultimately result in more extensive exposures.

Clinical signs may not develop immediately, and it may require up to 12 hours for the full extent of tissue damage to become apparent. Acute signs include depression, hypersalivation, anorexia, oral inflammation or ulceration, smacking of lips, tongue flicking, dysphagia, vomiting (+/- blood), abdominal pain, and melena. Significant hyperthermia (>104° F) may accompany oral inflammation. Esophageal and/or pharyngeal ulceration may occur. Inhalation of corrosive material may result in coughing, dyspnea, and moist lung sounds. Sequelae can include esophageal perforations or strictures and pleuritis or peritonitis from leakage of ingesta through perforated mucosa. As with oral acid exposures, emesis should NOT be induced and activated charcoal should not be given. 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. Should mucosal burns develop, treatment should include antibiotics, pain medication as needed, gastrointestinal protectants (e.g. sucralfate), anti-inflammatories (corticosteroid use is controversial) and general supportive care. 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 is risk of stricture formation, leading to impairment of esophageal function.

Cationic Detergents

Cationic detergents are contained in fabric softeners, some potpourri oils, hair mousse, algaecides, germicides and sanitizers. 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. Local tissue injury caused by cationic detergents resembles that seen with exposure to alkaline products (see Alkali section). 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. Treatment of local exposure is similar to that for alkaline products (see Alkali section). Systemic

signs should be treated symptomatically (i.e. fluids for hypotension, diazepam for seizures, etc.).

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 since 1983 contain 99.2% zinc and 0.8% 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. Secondly, 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 vacutainers to minimize contamination with exogenous zinc. Radiography of the abdomen may reveal the presence of coins or other “hardware” within the stomach.

Treatment for recently ingested pennies would 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. Treatment for symptomatic animals should include blood replacement therapy as needed, intravenous fluids, and other supportive care. The use of chelators may not be necessary in cases where prompt removal of the zinc source is accomplished. If chelation therapy is instituted, careful monitoring of renal parameters is important for the duration of therapy.

Polyurethane adhesives

Isocyanate glues (Gorilla Glue®, Elmer's ProBond Polyurethane Adhesive®) are expanding wood glues that have been associated with gastric foreign bodies (FB) in dogs. These products contain isocyanates. When ingested (chewing a 2 oz bottle of adhesive has been sufficient) the adhesive polymerizes into a large, friable FB that can form a cast of the gastric lumen. The adhesive is hygroscopic, absorbing water from the stomach as it expands and the warm body temperature may also play a role in expansion. Dogs licking small amounts off of the floor or ingesting paper towels soaked with the product generally had mild, transient GI signs but no FB. Attempts to dilute recently ingested glues with food or liquids have not prevented FB development. Do not induce emesis due to risk of expanding FB in esophagus. Radiographs can be

performed to determine the presence of a FB in the stomach (looks like kibble). Sometimes the FB is large enough to palpate. Evidence of a foreign body has been seen as early as 4 hours post-ingestion, but radiographs at 24 hours post-ingestion are likely to be more reliable. If present, the FB will require surgical removal.

“The Tasty”

Chocolate

There are a wide variety of chocolate and cocoa products to which pets may be exposed, including candies, cakes, cookies, brownies, and cocoa bean mulches. Not surprisingly, the incidence of accidental chocolate exposures in pets occurs around holidays, especially Easter, Halloween and Christmas. The active (toxic) agents in chocolate are methylxanthines, specifically theobromine and caffeine. Methylxanthines stimulate the CNS, act on the kidney to stimulate diuresis, and increase the contractility of cardiac and skeletal muscle. The relative amounts of theobromine and caffeine will vary with the form of the chocolate (see table).

Cocoa beans may contain up to 255 mg theobromine per ounce of beans, although the exact amount will vary due to natural variation of the cocoa beans. The LD₅₀'s of theobromine and caffeine are 100-300 mg/kg, but severe and life threatening clinical signs may be seen at levels far below these doses. Mild signs have been seen with theobromine levels of 20 mg/kg, moderate signs have been seen at 40-50 mg/kg, and seizures have occurred at 60 mg/kg. Clinical signs occur within 6-12 hours of ingestion. Initial signs include polydipsia, bloating, vomiting, diarrhea, and restlessness. Signs progress to hyperactivity, polyuria, ataxia, tremors, seizures, tachycardia, PVC's, tachypnea, cyanosis, hypertension, hyperthermia, and coma. Death is generally due to cardiac arrhythmias or respiratory failure. Hypokalemia may occur later in the course of the toxicosis. Because of the high fat content of many chocolate products, pancreatitis is a potential sequela.

Management of chocolate ingestion includes decontamination via emesis followed by gastric lavage. Activated charcoal may be given in some instances. Intravenous fluids at twice maintenance levels will help maintain diuresis and enhance urinary excretion. Because caffeine can be reabsorbed from the bladder, placement of a urinary catheter is recommended. Cardiac status should be monitored via EKG and arrhythmias treated as needed; propranolol reportedly delays renal excretion of methylxanthines, so metoprolol is the beta-blocker of choice. Seizures may be controlled with diazepam or a barbiturate. In severe cases, clinical signs may persist up to 72 hours.

Compound	Milligrams per ounce	
	Theobromine	Caffeine
White Chocolate	0.25	0.85

Milk Chocolate	58	6
Semi-sweet Chocolate chips	138	22
Baker's Chocolate (unsweetened)	393	47
Dry cocoa powder	737	70

Bread Dough

Raw bread dough made with yeast poses mechanical and biochemical threats to animals ingesting it. The warm, moist gastric environment stimulates yeast growth, resulting in expansion of the dough mass, resulting in gastric distention, which if severe, can result in respiratory and vascular compromise. Perhaps more significant is the release of alcohol from yeast fermentation, resulting in profound metabolic acidosis, CNS depression and death. Early clinical signs may include unproductive attempts at emesis, abdominal distention, and depression. As alcohol intoxication develops, the animal becomes ataxic and disoriented. Eventually, profound CNS depression, weakness, recumbency, coma, hypothermia may occur. Management of exposure includes decontamination and treatment for alcohol toxicosis. Because emesis is often unsuccessful, gastric lavage with ice water is recommended.

Moldy Food (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, while others alter neurotransmitter levels. The overall effect 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 seizing 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 the Animal Health Diagnostic Laboratory, Michigan State University (517-355-0281).

Macadamia Nuts

Macadamia nuts are cultivated from *Macadamia integrifolia* trees commonly found in Hawaii and Australia. After ingesting macadamia nuts, dogs develop weakness, depression, vomiting, ataxia, tremors, transient paresis, and hyperthermia. The mechanism of macadamia nut toxicosis in dogs is not known. Signs develop within 12 hours and most dogs return to normal with minimal care within 48 hours. Clinical signs are reported at ingestions as low as 2.4 g/kg body weight. Treatment of clinical signs includes fluids and thermoregulation. Prognosis of macadamia nut intoxication is good.

Xylitol

Xylitol is a sugar alcohol. It is used in sugar-free products such as gums and candies as well as for baking. It doesn't cause significant increases in blood glucose or insulin in humans. However, in dogs, xylitol causes a rapid, dose-dependent insulin release followed by potentially significant hypoglycemia. Signs can include vomiting, weakness, ataxia, depression, hypokalemia, seizures, and coma. Some dogs have developed liver dysfunction or failure following ingestion of xylitol although the mechanism of action is unknown.

Treatment of xylitol ingestion by dogs should include emesis, if asymptomatic. A dog can show signs of hypoglycemia in as few as 30 minutes. Activated charcoal does not bind xylitol. Frequent small meals or oral sugar supplementation may be used to manage dogs not showing signs. If clinical signs of hypoglycemia develop, a bolus of IV dextrose followed by a dextrose CRI should be used to control moderate to severe hypoglycemia. Hypokalemia, likely secondary to insulin-induced movement of potassium into cells, should be treated if significant. Treatment should continue until blood glucose normalizes. Liver enzymes should be monitored for 24 hours.