Poison Control Experts
Consultation with an animal poison control center is recommended when dealing with any toxicity in your clinic or hospital. These centers are open 24/7 and staffed with specialists in Toxicology with an encyclopedic knowledge of toxicants and their treatment in many veterinary species. Consultations with these services are very reasonable, and one fee provides you with unlimited follow-up as needed while treating that patient.

ASPCA Animal Poison Control Center: 1-888-4ANI-HELP (1-888-426-4435)
- www.aspca.org/pet-care/animal-poison-control
- $65 consultation fee

Pet Poison Helpline: 1-855-764-7661
- www.petpoisonhelpline.com
- $59 consultation fee

Many toxic product manufacturers may also cover the cost of consultation with poison control centers, or may have treatment information for exposure to veterinary species. Having the owner call the manufacturer may be helpful in many cases.

Basics of Treating Toxicities
As in any emergency presentation, the first priority must be ensuring that the patient is stable by performing a thorough primary survey, starting with the ABCs: Airway, Breathing, and Circulation. If a patient presents seizing or in shock, these life-threatening conditions must be ameliorated prior to instituting treatment for the specific toxicant. While the patient is being stabilized, a history can be obtained from the owner, including the specifics of the toxicant involved. When gathering this information, it is critical to obtain as much information about the toxicant as possible. Ideally the owner will bring any packaging or bottles along with the pet at presentation. If not, web searches can be helpful for identification of active ingredients and contents of the product if the owner knows the brand name. Other important history information to gather includes the length of time the animal has been exposed to the toxicant (i.e. how long since ingestion or application), the route of exposure (i.e. topical, ingestion, inhalation), and how much of the toxicant the pet was exposed to.

Decontamination
Once the patient is stable, the toxicant has been identified, and the details of the exposure are known, measures should be taken to prevent further absorption of the toxicant. Different decontamination methods are employed depending on the method of exposure and the patient’s status at presentation.

External decontamination: In cases of topical exposure (i.e. alkali cleaning agents, chemicals, pyretherins in cats), thorough bathing in tepid or room temperature water with degreasing dish soap (such as Dawn®) is indicated with copious rinsing to be sure that the agent is completely washed away. Rinsing of the affected area should continue for a minimum of 10 minutes, and longer if signs persist. Extra care must be taken when bathing patients who are recumbent or with reduced responsiveness or reflexes to avoid aspiration while bathing. Ensure that the patient is thoroughly dried after rinsing and check core body temperature frequently to avoid hypothermia, especially in pediatric, geriatric, or debilitated patients. Patients exposed to chemical agents, particularly alkali agents, will likely need extensive wound care in the post-decontamination period. Those patients exposed to alkali agents require longer rinsing periods to help remove and dilute as much of the agent as possible and prevent it from penetrating more deeply in the patient’s tissues.

Internal decontamination: In cases where a toxicant was ingested, there are several methods we can employ to prevent absorption from the GI tract:

Emesis induction: In many cases, inducing emesis is the first step in GI decontamination, which – if successful – will remove between 40 and 80% of stomach contents. If a patient has altered mentation, is recumbent, or lacks a gag reflex, emesis is contraindicated. Emesis is also contraindicated in the ingestion of agents that are corrosive, alkali, acidic, or hydrocarbons. Inducing emesis should ideally be done in the controlled environment of the
animal hospital, under supervision of trained veterinary staff. Emesis induction is far from a benign procedure and can lead to dangerous sequelae, including aspiration pneumonia, which can be a life-threatening complication.

In dogs, the pharmaceutical agent of choice for inducing emesis is apomorphine (aka “apo”). Apo binds to dopamine receptors in the chemoreceptor trigger zone (CRTZ), the area of the brain responsible for inducing nausea. Apo comes in pill form, which can be crushed and placed in the conjunctival sac; vomiting usually begins within five to ten minutes. While relatively effective at inducing emesis, administering apo in this manner can lead to conjunctival irritation, as well as corneal damage. Apo is compounded into an injectable agent by several compounding pharmacies and can be administered IV; vomiting usually begins two to three minutes after administration. Most patients vomit four or five times, but nausea may persist after the stomach is “emptied” so administration of an anti-emetic such as maropitant is indicated, particularly if the clinician wishes to administer agents orally to further decontaminate the gastrointestinal tract (see below). Because apo is a derivative of morphine, it can cause sedation, particularly if multiple doses are administered. Dogs should be monitored closely to ensure they maintain their gag and swallow reflexes. While apo is very effective in dogs it is not an effective emetic agent in cats because cats don’t have as many dopamine receptors. Alpha-2 agonists such as xylazine or dexmedetomidine are better choices, though cats are notoriously difficult patients in which to induce vomiting. Dosing for apo, xylazine, and dexmedetomidine can be found in veterinary formularies.

Hydrogen peroxide at a concentration of 3% is often recommended to induce emesis at home. While I prefer to have patients in the controlled environment of the animal hospital for emesis induction, if clients live far away, or the agent is particularly lethal, reducing absorption as much as possible before presentation to the animal hospital may be indicated. Hydrogen peroxide must be used with caution as its method of inducing vomiting is from direct irritation of the stomach lining. It is a caustic substance, with a pH of around 4 or 5 and aspiration leads to a vicious pneumonia and chemical pneumonitis. A study published in the *Journal of Veterinary Emergency and Critical Care* this year found “[s]ignificant visual and histopathologic gastric lesions” after administration of two doses of 3% hydrogen peroxide. No lesions were found in dogs who were administered apo in the conjunctival sac. It may be prudent for these patients to receive gastric protectants upon presentation to your hospital.

Agents such as salt or mustard powder or other substances are not recommended for emesis induction.

**Dilution:** Induction of emesis is contraindicated in the case of ingestion of caustic or corrosive agents that may cause damage to the physical structure of the GI tract or hydrocarbons that can be quite dangerous if aspirated. In case of digestion of these types of toxicants, dilution with milk or GI-coating agents (e.g. milk of magnesia) is indicated, potentially followed by a cathartic agent. If a lethal amount of toxicant is ingested – or the toxicant is especially dangerous – gastric lavage may be employed to empty the stomach (see below).

**Adsorbent agents:** Activated charcoal can be administered in many toxicity cases to prevent absorption from the GI tract. The charcoal will adsorb certain toxic agents and facilitate excretion with the feces. Often a cathartic agent such as sorbitol is added to the charcoal to speed emptying of the GI tract (see below). Not all toxicants bind to activated charcoal so consultation with a trusted toxicology reference or an animal poison control center is warranted prior to administration.

**Cathartic agents:** Sorbitol, lactulose, magnesium salts, or bulk fiber can be used to speed transit of a toxicant through the intestinal tract. The increased speed decreases chances of absorption from both the large and small intestine. These agents are especially effective when combined with an adsorptive agent, such as activated charcoal.

**Gastric lavage:** Ingestions of hydrocarbons, caustic substances, bulky (e.g. bread dough), or very lethal toxicants may require gastric lavage. Gastric lavage requires general anesthesia and must always proceed with a cuffed endotracheal tube in place to protect the patient’s airway from both lavage fluid and stomach contents. Thorough lavage can achieve 80-90% emptying of the stomach contents.

**Surgical removal:** If the toxicant is solid (i.e. intact batteries, pennies), endoscopic or surgical removal is the most effective method to prevent toxicant absorption.

In cases where absorption has already occurred such as ingestion of xylitol, alcohol, or if presentation to the animal hospital is delayed, it may be possible and necessary to decontaminate the patient’s bloodstream. A
mainstay of treatment is dilution using IV fluids. IV fluid administration will help to dilute any toxicant absorbed, as well as increasing renal blood flow, which may increase excretion of toxin while also providing supportive care for the patient. There are agents available to practitioners to help clear toxins from the blood stream.

Intralipid Emulsion Therapy: Intralipid emulsion (ILE) is the fat portion of partial or total parenteral nutrition solutions. The use of ILE for intoxications began with the study of the treatment of local anesthetic overdoses in the 1990’s. Since then, ILE has been viewed as the “holy grail” of blood stream decontamination in cases of lipophilic toxicants. The more lipophilic the toxin, the more likely it is that ILE could be used as a treatment. Lipophilicity is described using a log P value. If the toxin’s log P value is > 1, it is considered lipophilic. The higher the log P value, the more lipophilic it is. Toxicants with high log P values are listed in Table 1. There are two theories as to how ILE exerts its anti-toxin effects. The first theory posits that providing the body with free fatty acids (FFAs) in the form of ILE floods the mitochondria and blocks the action of many of the toxicants that interfere with proper mitochondrial function and normal use of FFAs. The second theory is called the “lipid sink” theory and posits that providing a large amount of free fat in the bloodstream creates a biochemical compartment that can pull lipophilic substances out of free circulation and into the fat “sink”, inactivating the substance and encouraging its metabolism and excretion along with the ILE.

**Table 1. A short list of substances with high log P values**

<table>
<thead>
<tr>
<th>Amlodipine (hypotensive agent)</th>
<th>Baclofen (muscle relaxant)</th>
<th>Bupivacaine (local anesthetic)</th>
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<tbody>
<tr>
<td>Bupropion (antidepressant; Wellbutrin®)</td>
<td>Carbamazepine (anticonvulsant)</td>
<td>Carprofen (NSAID; Rimadyl®)</td>
</tr>
<tr>
<td>Chlorpheniramine (antihistamine; Chlor-Trimeton®)</td>
<td>Chlorpromazine (antipsychotic)</td>
<td>Clomipramine (TCA; Clomicalm®)</td>
</tr>
<tr>
<td>Cyclosporine (immunosuppressant; Atopica®)</td>
<td>Dexamethasone (glucocorticoid)</td>
<td>Diazepam* (sedative)</td>
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<tr>
<td>Digoxin* (cardiac glycoside)</td>
<td>Diltiazem (Ca+ channel blocker)</td>
<td>Indomethacin (NSAID)</td>
</tr>
<tr>
<td>Itraconazole (antifungal)</td>
<td>Ivermectin (antiparasitic; Heartgard®, Ivomec®)</td>
<td>Ketoprofen (NSAID)</td>
</tr>
<tr>
<td>Lidocaine (local anesthetic, antiarrhythmic)</td>
<td>Loratadine (antihistamine; Claritin®)</td>
<td>Metoprolol (Beta blocker)</td>
</tr>
<tr>
<td>Moxidectin (antiparasitic; Advantage Multi®)</td>
<td>Naproxen (NSAID; Aleve®)</td>
<td>Nicotine (stimulant)</td>
</tr>
<tr>
<td>Nifedipine (antianginal; Procardia®)</td>
<td>Promethazine (antihistamine)</td>
<td>Pyrethrin insecticides</td>
</tr>
<tr>
<td>Trazadone (SSRI)</td>
<td>Verapamil (Ca+ channel blocker)</td>
<td>Vinblastine (chemotherapeutic)</td>
</tr>
</tbody>
</table>

*antidote / reversal agent available

Dosing for ILE can be found via consultation with poison control centers, or in veterinary formularies. When using ILE, it is important to monitor the patient for side effects such as fat emboli, allergic reactions, and Fat Overload Syndrome (FOS). Many of the substances listed in Box 1 may have other, more conventional therapies, antidotes, or reversal agents available. In those cases, ILE should be used only if the patient is not responsive to conventional therapy.

Chelation therapy: In the case of metal ingestions (e.g. lead, iron supplements, zinc), it may be necessary to perform chelation therapy. Chelating agents cause precipitation of metals out of tissues for excretion by the kidneys. This therapy can be nephrotoxic so close monitoring is required.

**Antidotes / Reversal Agents**

In cases where an antidote or reversal agent is available it should be administered as early as possible, while also pursuing stabilization, decontamination, and supportive care efforts.

**Nursing and Supportive Care**

Regardless of the toxicant the patient is exposed to, supportive care is key to good patient outcomes. Most patients will require IV fluid therapy and, in some cases, intensive nursing care and monitoring. Veterinary
technicians who are familiar with toxicology concepts and treatment modalities are central to ensuring that patients recover with minimal long-lasting effects.

*References available on request*