Recognizing & Treating Toxicities

Heidi Houchen, DVM
VCA Northwest Veterinary Specialists
Clackamas, OR

Poisons of the Pacific Northwest

General Overview of the Treatment of Toxicities

Telephone Triage

Often, the first place a veterinary technician interacts with the owner of a suspected poisoned pet is on the telephone. Telephone calls regarding potential poisoning are among the most common calls to a veterinary emergency clinic. Information regarding (a) possible exposure to toxins and (b) the animal's status are the key areas of information to obtain to determine if the patient should be brought to the hospital and what, if any, initial treatment should be initiated at home.

If any question exists, the patient should be brought immediately to the clinic.

During the telephone conversation, it is important to (quickly) ask a standard series of questions so that an accurate assessment of the patient's condition can be made.

1. Signalment: What is the species of the pet and what is its age and weight?
2. Why do you suspect your animal has been poisoned or gotten into a toxic substance?
3. Where does your pet live—outdoors, indoors, both? What does animal have access to in these environments?
4. Do you have any containers of known toxic substances (i.e., antifreeze) to which the animal has access? Are there any containers that appear chewed or are any contents missing? How much of the container's contents is missing?
5. Does the animal have access to any types of bait (slug or rodent)?
6. When did the exposure to the potential toxin occur?
7. Was the poison ingested or topically applied?
8. In what form is the toxin (liquid, capsules, pellets, powder)?
9. Have you given your pet medication or does your pet have access to any human medication?
10. How is the animal acting?

    - Airway and Breathing Questions: How is the animal breathing—rapid or shallow, is it having difficulty breathing?
    - Cardiovascular: What color are the gums? Can you feel a pulse?
    - Neurologic Status: How responsive is the animal—is the animal alert? Aware? Hyperactive? Depressed or semi-conscious? Is the pet acting weak or "drunk"?
    - Gastrointestinal: Vomiting? Diarrhea? What is the color of either material? Is there blood present? Do you notice the toxin in the emesis?

If there is any question regarding the stability of the patient or the toxin ingested, the pet should be brought to the hospital immediately.

In general, if the animal is showing any of the following signs, it should be brought immediately to the clinic:

Weakness	Pale mucous membranes
Difficulty Breathing	Persistent Vomiting
Bleeding	Stupor, coma, seizure
Rapid heart rate	Poor pulses

If the potential toxin is still present in a container, the environment, or in the animal's vomitus, it should be placed in a sealed plastic container and brought to the hospital.

At-Home Treatment

The decision to instruct an owner to begin treatment (induce vomiting) at home is made rarely and on a case-by-case basis. Specific criteria that must be met before a veterinarian *considers* recommending an owner induce vomiting at home include:

- If the toxin has been identified
- If the toxin has been ingested within the preceding 1-2 hours
- If the toxin is not contraindicated for emesis (See Contraindications to Emesis section)
- If the owner is several hours from the nearest veterinary facility
- If the owner is physically and mentally able to monitor the animal during emesis

If the decision is made to instruct an owner to induce vomiting at home, it is essential to make the owner aware that the emetics available at home are not as effective as those used in a veterinary clinic. In addition, many owners find administration of emetics challenging especially if their animal is difficult to medicate under normal circumstances.

Although in the past salt solutions, liquid dishwashing detergent, mustard, and syrup of ipecac were the items available in the home and recommended by veterinarians to induce vomiting in pets, NONE of these items are currently recommended for use as they are unreliable in induction of vomiting and cause SERIOUS side effects.

Hydrogen peroxide 3% administered orally is the only substance recommended for at-home use by veterinary toxicologists. The current consensus on dosing is 0.5 to 1.0 mls/kg (and no greater than a total of 45 mls). It can be repeated in 10 minutes; if no vomiting is induced after two doses, NO FURTHER DOSES are recommended. Administration via an eyedropper or turkey baster can be recommended IF care is taken not to aggressively squirt the material down the animal's throat and cause them to aspirate. If the owner does decide to induce vomiting at home and then bring the animal into the clinic, advise the owner to protect the interior of their car.

Important to remember that if any question exists about whether the animal should be vomited at home, advise the owner to bring the animal to the clinic immediately. The assessment and treatment of the animal in the clinic should not be delayed by the owner's home treatment.

If the pet's eyes or skin have been exposed to a potential toxin, it is often advised that the owner try to decrease further absorption or irritation. Rinsing the eyes with a sterile saline solution (such as saline solution used for contacts) or with water is advisable. If the pet will allow the owner to bathe it, the owner should be advised to use a mild pet shampoo and rinse thoroughly. However, if the pet is acting aggressive, forewarn the owner that they could be injured trying to bathe their pet and it may be better to bring the animal to the clinic to be bathed.

Initial Assessment of Patient

"Treat the patient, not the poison"

Triage

The same basic principles of triage should be used with potentially poisoned animals as with other emergency patients. Follow the "ABCs" (Airway, Breathing, Circulation), evaluate vital signs, and give appropriate emergency care as needed. Animals in respiratory distress need to have their airway evaluated for patency; administer oxygen or mechanical ventilation as needed. Patients in circulatory shock require administration of appropriate IV crystalloids, colloids, or blood products. Tremoring or seizuring patients require immediate catheter placement and administration of seizure control drugs. Hyperthermic animals require cooling through the use of cool baths or cooled intravenous fluids. Once an animal has been determined no longer to be in a life-threatening condition, then the clinician can proceed with obtaining a full physical exam, a full history, appropriate blood samples and baseline labwork, and placing an intravenous catheter.

General Approach to the Toxin Patient

The following principles should be followed for managing the toxin patient:

1. Assess vital signs, treat life-threatening problems, and stabilize
2. Obtain a complete history, including exposure assessment
3. Obtain samples /baseline data
4. Administer appropriate antidote (if one is available)
5. Prevent any continued exposure to, or absorption of, toxicant
6. Promote excretion or elimination of toxic compound
7. Monitor patient and provide continued supportive care

The order that these steps are performed can vary from patient to patient depending on presentation and toxin. For example, administering the antidote 100% oxygen to a known smoke inhalation patient or atropine to an animal in the throes of an OP neuromuscular blockade are part of the triage component of treating a toxin patient. In contrast, removing the zinc penny from an animal's GI tract would come before administration of Calcium Disodium EDTA, often eliminating the need for the chemical chelator altogether.

History

Asking the questions as outlined above in the Telephone Triage section is essential to providing a successful outcome for the poisoned patient. Recommended reading in this area is the short but extremely well written chapter "Taking a Toxicologic History" in Gwaltney-Brant & Poppenga's *Small Animal Toxicology Essentials*.

Exposure Assessment

Once the patient has been stabilized, further assessment for the possible underlying cause should be made. Unless there is a witnessed exposure to a toxin or a specific laboratory analysis has been made, a definitive diagnosis of "poisoning" is often challenging in veterinary medicine. If the toxicant is unknown or if an animal is suspected of ingesting a toxin but is not clinical, it is important to do an "exposure assessment." This is defined as the process of determining whether the amount of toxin to which an animal has been exposed is consistent either with the clinical signs or with the potential to cause clinical signs. What you need to know to make this kind of assessment is the weight of the animal, the estimated amount of toxin to which the animal was exposed, and the toxicity of the chemical.

As Paracelsus, the 16th century Swiss alchemist and father of toxicology would say, "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy." In deciding how to manage a toxicity case, it is important to know how toxic the chemical is (i.e., ethylene glycol in antifreeze versus theobromine in milk chocolate) with which you are dealing, the dosage of the chemical ingested (i.e., puddle of antifreeze versus half a chocolate bar that has gone missing), and the weight of the animal (i.e., Maltese versus a Great Dane). The easiest parameter to determine is often the weight of the animal. The toxicity of the chemical can be found by utilizing any one of several sources, including VIN (Veterinary Information Network), NAPCC (National Animal Poison Control Center), VCA NWVS Intern Handbook, the packaging that contained the toxicant, the website for the product, or toxicology textbooks such as Peterson and Talcott's *Small Animal Toxicology* as well as Gwaltney-Brants and Poppenga's *Small Animal Toxicology Essentials*.

The most challenging part of the exposure assessment to identify is determining the largest possible amount of the toxicant to which the patient was exposed. The amount of potential toxin an animal has been exposed to can be determined by, for example, determining how many pills are left in a prescription pill or OTC medication bottle, how much of a packet of rodenticide is remaining, how much topical flea product was applied, or how much of a raisin-based trail mix is missing. When estimating, it is best to be conservative and base calculations on a "worse case" amount or scenario.

Emergency Database/Blood Samples

Once the diagnosis of toxin exposure or ingestion has been made and the patient has been stabilized, general treatment as well as specific antidotal treatment of the poisoned patient can begin. It is ideal to obtain blood and urine samples for clinical as well as toxicologic testing prior to any treatments being administered as some treatments (i.e., diazepam containing propylene glycol can interfere with ethylene glycol testing) may alter test results. If a patient is suspected of ingesting a toxin, baseline admitting bloodwork should contain CBC, chemistry panel and electrolytes. Other screening diagnostics that might be helpful include a coagulation panel (rodenticide toxicity), ECG (methylxanthine ingestion), radiographs (zinc toxicosis), ethylene glycol testing (antifreeze ingestion), and urinalysis (illicit drug screening).

Treatment of the Poisoned Patient: Antidotes

If a specific "culprit" toxin has been identified, clients as well as clinicians often seek the "Holy Grail"—that is, the specific antidote for a particular toxin. Unfortunately, there are several drawbacks to this approach:

- There is no "universal antidote."
- Very few antidotes exist for the majority of toxins in our world; treatment for most poisons is supportive and symptomatic.
- Relatively few companies make antidotes due to the high cost of production and the low profit margin to be made.
- Antidotes are often expensive, have a short expiration date, and are difficult to locate and access.
- Some antidotes are potentially toxic themselves, need precise care to deliver properly, and a patient must be monitored closely during time of administration.
- The majority of antidotes in small animal veterinary medicine are off-label use.
- Even when a specific antidote to a toxin exists, it is important to stress to the client that supportive care including decontamination is often equally important in treating their animal.

In general, antidotes fit into three basic categories:

1. *Chemical antidotes* are substances that directly act on a toxin to create harmless compounds.

    - Chelation: These substances bind a toxin (typically a metal) into a ring structure, increasing their water solubility and promoting excretion. An example of a chelator is deferoxamine, used in iron toxicosis.

    - Enzyme Inhibition: These substances work either by competitively or irreversibly inhibiting the formation of toxic metabolites. An example of enzyme inhibition is 4-methylpyrazole, used in ethylene glycol/antifreeze toxicosis.

    - Commercial antibodies: These are the antibodies extracted from a host animal's blood after a small amount of toxin has been injected. Example of an antibody antidote include antivenin (snakes and spiders) and antidigitoxin Fab fragments (plant cardiac glycosides and Bufo toads).

2. *Pharmacological antidotes* are substances that, either through binding to the toxin's receptor site or through binding to the toxin itself, minimize or eliminate the action of the toxin. Examples of a pharmacologic antidote include atropine (used for anticholinesterase pesticide toxicosis), hyperbaric oxygen (carbon monoxide poisoning) and naloxone (opiod intoxication).

3. *Functional antidotes* are substances that are used to the specific symptoms caused by a toxin. Examples of functional antidotes are cyproheptadine (used in Serotonin Syndrome), methocarbamol (used in slugbait toxicites), and pamidronate (cholecalciferol rat bait and Dovonex toxicity cases).

TOXIN	ANTIDOTE
Acetaminophen	Acetylcysteine or Sodium Sulfate
Alpha Adrenergic Agonists	Atipamezole
Amitraz	Tolazine or Yohimbine
Anticoagulant Rodenticides	Vitamin K1 (Phytondione)
Arsenic	Dimercaprol (BAL)
Benzodiazepines	Flumazenil
Carbon Monoxide	100% Oxygen
Cholecalciferol	Pamidronate Disodium or Calcitonin
Copper (inherited storage dogs)	D-Penicillamine or Trientene
Crimidine	Pyridoxine (vitamin B6)
Cyanide	Hydroxocobalamin OR Sodium Nitrite/Thiosulfate
Digitalis (digoxin)	Digoxin Immune Fab
Ethylene Glycol	4-Methylpyrazole or Ethanol
Heavy Metal (cadmium, zinc)	Calcium Disodium EDTA or D-Penicillamine
Heparin	Protamine
Iron	Deferoxamine
Lead	Calcium Disodium EDTA *with or without* Dimercaprol or D-Penicillamine OR Succimer (dogs)
Mercury (inorganic)	Calcium Disodium EDTA or D-Penicillamine OR DMSA
Mercury (organic)	Dimercaptosuccinic acid (experimental)
Mushrooms (Amanita)	Acetylcysteine
Naphthalene / Nitrates	Methylene Blue
Opiods	Naloxone
Organophosphates	Atropine (or glycopyrrolate) AND 2-PAM
Pyriminil	Nicotinamide
Sodium fluoroacetate 1080	Monoacetin, acetic acid, AND ethanol
Thallium	Diphenylthiocarbazone (dithizone)
Venom (snake, spider)	Antivenin
Xylazine	Yohimbine
Zinc	Calcium Disodium EDTA or D-Penicillamine
***Fat Soluble Drugs	Intravenous Lipid Solutions
local anesthetics, buproprion	
barbiturates,propranolol, verapamil,	
TCA's and avermectin	

Treatment of the Poisoned Pet: Decontamination

The aim of general decontamination is to prevent further toxin absorption, promote toxin excretion and to provide continuous supportive care and monitoring of the animal throughout the process. How the animal has been exposed to the toxin determines how to decrease or eliminate any further absorption.

Dermal Exposure

If the animal has been exposed via the skin, then both the skin and the GI tract are considered to be routes of exposure. Bathing often eliminates a large portion of the toxicant, and this can be achieved with either a mild shampoo designed for pets or a diluted, mild dishwashing detergent. Rubber gloves and protective clothing such as rubber aprons should be worn by veterinary staff who are bathing animals. If an animal has been heavily contaminated, then multiple baths may be necessary. Special care should be taken to lubricate eyes and to keep the animal warm and dry afterwards. Mild sedation should be considered for fractious animals so they do not traumatize themselves or their caregiver. In circumstances involving substances such as tree sap, tar, glues and adhesives, oil-based products— such as mineral oil, vegetable oil, mayonnaise, butter, or peanut butter—can be helpful to loosen "sticky" material from an animal's fur. Working the oil in and around the "gummy" toxin will make it stiffer and easier to remove with a brush or clipper. Bathe the animal thoroughly with a mild shampoo after removing as much as possible of the sticky substance.

Ocular Exposure

When an animal's eye has come into contact with caustic or toxic substances, they should be copiously flushed. Owners can be instructed to flush their pet's eyes (gently) either prior to transport or in transport to a veterinary facility. Fluids that can be used as flushing solutions include tepid water or sterile saline or eye wash solutions. Contact solutions containing cleaning agents should not be used. Eyes should be flushed from the medial canthus to the lateral canthus in order to avoid contaminating the other eye. If an eye has been extensively irritated by a toxic substance, it may require flushing for 20-30 minutes, and the animal may require sedation. After flushing, the eye should be evaluated for corneal ulcers with fluoroscein staining. After the staining process and subsequent flushing of the stain, a lubricating eye ointment should be instilled in the affected eye.

Inhalation Exposure

Animals can inhale toxic smoke, gasses, fumes, dust, particulates, and ash if they are exposed to contaminated areas or are trapped in a fire. Although challenging to determine, clinical signs such as singed whiskers, acrid-smelling haircoats, coughing or wheezing, or powder/dust on faces or bodies can be evidence that a pet has had an inhalation exposure. Removing the pet from the source of the inhaled toxicant is the first step in treatment; this is followed by administration of supplemental oxygen (if indicated) and supportive care. IV fluids, pain medications, bronchodilators and/or additional medications may be indicated based on the source of the exposure. Diagnostics including baseline and serial blood gas analysis and thoracic radiography are often helpful in monitoring response to therapy.

Oral/GI Exposure

Eliminating a toxin from the gastrointestinal tract typically involves:

1. Evacuation of the GI tract : emesis or gastric lavage
2. Administration of adsorbants
3. Administration of cathartics

1) Evacuation of the GI tract

Early emesis, within 2 hours of toxin ingestion, can remove 40-60% of ingested material and is the most effective means of emptying the stomach—more beneficial than gastric lavage. Past the 2 hour time period post-ingestion, inducing emesis is only indicated if the animal ingested a large amount of material. The sooner emesis is induced post ingestion, the greater chance here is for recovering a larger amount of toxin. If two doses of an emetic are administered and the

animal does not vomit, subsequent doses of that emetic will not be effective. At that point, the choice becomes either to try a different emetic or to administer activated charcoal.

Dogs, cats, ferrets, and potbelly pigs are companion animals that can be induced to vomit. Contraindications to emesis are listed in the section below:

Contraindications to emesis:

- Altered neurologic status: stupor, coma, seizures, extreme depression
- Respiratory distress
- Laryngeal paralysis
- Decreased gag reflex
- Cardiovascular disease
- Ingestion of sharp or irritating objects
- Ingestion of corrosive or caustic materials (i.e., acids, lye)
- Ingestion of petroleum distillates (i.e., gasoline)
- Rodents, rabbits, birds, horses, or ruminants
- Recent histories of abdominal surgery or potential for a gastric torsion
- Emesis should not be attempted if the animal has already vomited or is exhibiting clinical signs.

In general, emetics are classified into two categories based upon their mode of action:

- Local gastric irritation
- Activation of the central nervous system.

Of those that work by local gastric irritation, including salt solutions, liquid dishwashing detergent, mustard, syrup of ipecac, and 3% hydrogen peroxide, only hydrogen peroxide are is recommended by toxicologists as a safe and effective emetic. The remaining items on that list are unreliable in induction of vomiting and can cause SERIOUS side effects.

Syrup of ipecac acts through both by gastric irritation as well as activation of the chemoreceptor trigger zone (CRTZ). Once a common item to be found in most human households, it has been removed from the market for the following reasons: it is no longer recommended for children who ingest toxicants and it became a health hazard as bulimics who abuse ipecac develop heart damage from chronic use. It is also no longer recommended in veterinary patients for several reasons: (1) a general lack of effectiveness in inducing emesis in approximately 50% of small animal patients, (2) if vomiting was induced, it was often associated with a delay of 40 minutes to an hour before emesis occurred, (3) residual ipecac in the stomach of patients who failed to vomit had the potential to cause life-threatening cardiotoxic arrhythmias, and (4) its extremely bitter taste made it challenging to administer (especially to cats).

Central acting (aka “veterinary”) emetics that are routinely used for the induction of vomiting in dogs and cats include apomorphine and xylazine.

<u>Emesis: Canine</u>

The most reliable drug used for inducing emesis in dogs in the veterinary setting is apomorphine. It is the emetic of choice due to its rapid onset, ease of use, multiple modes of administration, and reversibility. The dosage varies slightly according to route of administration:

Intravenous @ 0.03 mg/kg
Intramuscular @ 0.04 mg/kg
Subcutaneous @ 0.08 mg/kg
Topical conjunctival @ 0.3 mg/kg

There has been, and continues to be, much debate as to the safest and most efficacious method of administering apomorphine. The protocol for apomorphine for many years was either to place a portion of a tablet (or alternatively dissolve a portion of a tablet in saline) and place this in a dog’s eye, rinsing it out once it had produced the desired effect

(vomiting of the toxin). However, in recent years, the use of IV apomorphine has been said to produce more consistent absorption (and thus more consistent emesis) and eliminate the potential for ocular irritation and ulcers. The potential adverse effects of sedation and protracted vomiting seen with apomorphohine can be reversed with naloxone (0.01 to 0.04 mg/kg). Apomorphine is contraindicated in animals with CNS or respiratory depression.

The locally acting gastric irritant hydrogen peroxide 3% is also still routinely used in veterinary clinics for induction of vomiting in dogs. The dosing recommendations and caveats in at-home use are the same: 0.5 to 2 mls/kg (and no greater than a total of 45 mls); it can be repeated in 10 minutes. If no vomiting is induced after two doses, NO FURTHER DOSES are recommended. Hydrogen peroxide is most effective if administered after a small meal. The complications and concern with using hydrogen peroxide include aspiration (from forceful oral administration), prolonged vomiting, hemorrhagic esophagitis and gastritis, and hematemesis. The potential for creating adverse effects with hydrogen peroxide can be minimized with appropriate dosing and careful administration.

Emesis: Feline:

Proving once again that "cats are an alien species," one of the greatest challenges facing a veterinarian is getting a cat to stop vomiting when they shouldn't and getting them to vomit when they should! Apomorphine is not as consistent in producing emesis in cats as it is in dogs. Studies have shown that while the dog's CRTZ is controlled largely by opioid receptors (which explains why they vomit predictably with apomorphine), the cat's CRTZ is largely controlled by alpha receptors (very little opioid involvement). In addition to lack of efficacy, apomorphine also has the potential to create a dysphoric "manic" state in felines and as such, is not routinely recommended. The centrally acting alpha agonist xylazine is the most frequently recommended agent for inducing emesis in cats; the recommended dosage is 0.44 mg/kg IM.

Typically, if xylazine induces vomiting, it will occur within 10-15 minutes after administration. The side effects of respiratory depression and excessive sedation from xylazine can be reversed. Toxicologists do not recommend using hydrogen peroxide in cats due to the potential for air embolus and the induction of esophageal and gastric erosions and hemorrhagic gastritis; there have been recorded fatalities. Several years ago, the ASPCA NAPCC did a survey to determine frequency with which emesis worked in cats: xylazine 60%, hydrogen peroxide 40%, and apomorphine 10%.

It is very important before attempting to induce vomiting with ANY substance in cats that the owners are informed, educated and understand the off-label use, the risks, the cost-benefit of removing a toxin versus attempting emesis, and potential adverse effects (even mild) involved.

One final note: The author's personal favorite advice was given by a VIN expert on how to induce vomiting in cats: *"Just place them on an expensive rug and wait."*

Another means of removing toxic material from the GI tract is gastric lavage. Gastric lavage is less effective than emesis, especially if the toxin was recently ingested, but it is an alternative if emesis was contraindicated or was ineffective. Gastric lavage is most effective if performed within the first hour post-toxin ingestion; recovery of the toxin has been shown to decrease to approximately 10-15% if delayed after this time period.

In order to perform gastric lavage, an animal must be anesthetized and an endotracheal tube placed. Because it is very important to prevent aspiration of stomach contents, the endotracheal tube should be properly placed and cuffed and a large bore stomach tube should be used. The stomach tube should be premeasured from the tip of the nose to the last rib. Warm water at a dose of 10 ml/kg body weight should be administered gently via a gravity flow or through a stomach tube. The use of physiologic saline is recommended for lavage fluid in smaller patients in order to minimize fluid and electrolyte abnormalities. After a few minutes, stomach contents and fluid are aspirated back and placed in a bucket for examination. This procedure should be repeated several times (upwards of 15-20 times is not unusual) until the lavage fluid is clear. Before removing the stomach tube, activated charcoal in suspension can be instilled in the stomach. Some clinicians instill activated charcoal *before* gastric lavage; the goal is to minimize further toxin absorption. Caution should be used with this procedure to avoid aspiration of activated charcoal. Before removing the stomach tube, "kink" it to prevent lavage fluid from leaking into the esophagus and oropharynx.

It is important to review potential adverse effects of gastric lavage with owners prior to performing this procedure. Complications can include aspiration of fluid/activated charcoal, traumatic injury to the upper GI tract (oropharynx, esophagus and stomach), and fluid and electrolyte imbalances.

2) Administration of Adsorbants

After emesis or gastric lavage has been performed, some toxin may still be present in the GI tract. Adsorption therapy, whereby a toxin is physically bound to an unabsorbable substance, is recommended after GI decontamination. Activated charcoal is a very effective adsorbant and the one most commonly used in veterinary medicine. Activated charcoal can be administered orally or via stomach tube following gastric lavage. The dosage recommended by toxicologists is 1-3 grams/kg of activated charcoal suspended with water (powdered charcoal mixed with water). For liquid Toxiban, the labeled dose is 10-20 ml/kg.

As with a large number of issues in toxicology, there is ongoing debate on the use of activated charcoal (when to use, how much to use, whether or not to use repeatedly, etc.). While there is not a complete consensus on activated charcoal, toxicologists in general recommend it in cases where serious, life-threatening signs can arise from the exposure.

The effectiveness of charcoal depends on the timing of administration and the toxicity for which it is being administered. In general, charcoal binds best to large organic molecules and to most pharmaceuticals and is most effective when administered early (within one hour) of toxin ingestion. Examples of compounds that bind to activated charcoal include OTC meds such as acetaminophen and aspirin; prescription meds such as antibiotics, TCA's and digitalis; illicit drugs such as morphine, amphetamines and cocaine; OP and carbamate insecticides; barbiturates; and baits such as metaldeyhyde, anticoagulant rodenticides and strychnine (to name just a few). An initial or "one-time" dose of activated charcoal often contains a cathartic such as sorbitol to aid in the removal of toxins. For toxins that undergo enterohepatic recirculation (i.e., the NSAID naproxen (74 hour half-life), ivermectin or moxidectin, bromethalin), extended-release formulations of some drugs, or very large ingestions of toxins, multiple doses of activated charcoal are indicated. Second and subsequent doses of activated charcoal should NOT contain sorbitol; this will reduce the risk of inducing hypernatremia. Repeated doses of activated charcoal should be given at half the initial volume and at intervals of 6-8 hours, depending on the toxin ingested. Animals receiving multiple doses of activated charcoal should be placed on intravenous fluids to maintain proper fluid and electrolyte balance.

Instances where activated charcoal is not recommended include the following: when it is not given in a timely manner, when it will not bind to a toxicant, when it has a chance for creating an adverse effect, or when it is contraindicated altogether. Activated charcoal does not bind well to heavy metals, inorganic molecules such as sodium chloride, nitrates, and chlorates, or to small organic molecules (e.g. ethanol, ethylene glycol, xylitol, etc.). Activated charcoal is not recommended for ingestions of caustic substances or hydrocarbons. It is not recommended for use in patients who do not have a functioning airway, an intact and functioning complete GI tract, who are comatose or having seizures, who are actively vomiting, or who are susceptible to electrolyte disturbances. Hypernatremia can occur with administration of activated charcoal—with or without sorbitol. Animals administered even a single dose of activated charcoal should be monitored closely for 4-6 hours (typical timeframe for development of hypernatremia) and either be placed on IV fluids or have free access to water.

3) Cathartics

A cathartic is a substance that enhances the movement of a toxin through the gastrointestinal system. There are several different types: bulk cathartics, saline cathartics and osmotic cathartics. High fiber products such as canned pumpkin, bread, or psyllium absorb water and aid in physically moving material through the GI tract. Sodium sulfate and magnesium sulfates (Epsom salts) stimulate motility of the gastrointestinal tract. The most commonly used cathartic is an osmotic cathartic: sorbitol. A commercially available suspension of 70% sorbitol is combined with activated charcoal to speed up removal of the toxicant-charcoal complex from the GI tract. While multiple doses of activated charcoal may be prescribed in scenarios where a toxin undergoes enterohepatic recirculation, only an initial single dose of sorbitol combined with activated charcoal is indicated. Multiple doses of cathartics can create excessive fluid loss and electrolyte disturbances.

Diagnosis, decontamination, and elimination of a toxin via administration of an antidote is often just the beginning of treatment; close monitoring and supportive care of the cardiovascular, respiratory, nervous and renal systems is key to providing a successful outcome for the poisoned patient.

Top Ten PNW Toxins

Lists of "most common intoxications" are often chosen by the following criteria: toxicity victims most often seen in emergency rooms across the US or toxicities most commonly reported to the NAPCC. With the vast numbers of poisons that exist in our world, it is most beneficial to our pets (and our brains) to focus on the ones that are relevant to the place we live on the planet. The toxins described below are a by-product of urban life. Our pets are exposed to food we eat, the drugs we use to medicate ourselves, and the chemicals we bring into our homes, garages, and yards to both beautify them and to rid them of "vermin." Because our "place in space" is smaller and our furry friends are underfoot, these items are more likely to poison a city-dwelling pet than one that goes farther afield.

- Rodenticides / Baits
- Pyrethrins
- Antidepressants / Serotonin Syndrome
- Pantry Poisons: Grapes/Raisins, Onions, Xylitol
- NSAIDS (human and veterinary)
- Chocolate/Caffeine/Methylxanthines
- Ethylene Glycol
- Marijuana
- Lilies (Bouquets of Terror)
- Tremorogenic toxicities: Compost and Metaldehyde

Anticoagulants Baits:

- Historically, the most popular chemicals used to control rodent populations have been "anticoagulants." Anticoagulant rodenticides competitively inhibit vitamin K epoxide reductase, the enzyme component necessary for the recycling of Vitamin K. This leads to a depletion of the Vitamin K dependent clotting factors II, VII, IX, and X. There is a typical delay in clinical signs of 3-7 days post-ingestion because of circulating clotting factors already produced and present in the body.

- Due to the resistance rodents developed to the first-generation short-acting compounds, most anticoagulant rodenticides found on shelves today are second-generation products. Typical active ingredients in these long-acting anticoagulant rodenticides include brodifacoum, bromadiolone, chlorophacinone, coumafuryl, and difethialone.

Diagnosis:

- Diagnosis of rodenticide toxicity is often made by a combination of factors: a witnessed ingestion, evidence in the form of a tampered rodenticide package, and/or suspect clinical signs including loss of color in the mouth and gums, weakness, and bleeding from the mouth, nose, urogenital area or from the anus. Bleeding into the thoracic cavity is frequently seen as dyspnea and/or coughing. These symptoms often show up several days after a pet has ingested the poison.

- Laboratory tests to confirm anticoagulant rodenticide toxicity include a PT (prothrombin time) and PIVKA (Proteins induced by Vitamin K Absence or Antagonists). A prolonged PT is an early detection test (within 72 hours of ingestion) as it will be elevated with any deficiency of Factor VII; PIVKA (Proteins Induced by Vitamin K Antagonism or Absence) is the measurement of the buildup of the inactive clotting factor precursors.

Clinical Signs:

- Pale mucous membranes, weakness, collapse, hemorrhage, lameness, swollen joints, dyspnea, coughing.

Prognosis:

- Good to Grave, depending on clinical signs and the initiation of early and aggressive treatment. Left untreated, pets with anticoagulant rodenticide ingestion will hemorrhage from virtually any site.

Treatment:

- If the rodenticide was ingested within a 2-3 hour time frame from presentation, vomiting is induced.
- Activated charcoal is administered to absorb any remaining toxin in the gastrointestinal tract.
- The patient should be started on vitamin K1 supplementation immediately and be continued on this medication for an average of 3-4 weeks. Vitamin K1 should not be given intravenously (possible anaphylaxis). The initial dose of Vitamin K1 is 5 mg/kg; this dose is then divided BID and given with a fatty meal (to enhance absorption) for the duration of treatment.
- Pet owners should be instructed to restrict activity of their pet until completion of Vitamin K1 therapy.
- Coagulation factors and PCV/TP should be rechecked in two days after Vitamin K therapy has been discontinued to make sure that all the effects of the rodenticide have been eliminated from the pet's system. If the PT remains elevated, then Vitamin K1 therapy should be continued.
- Patients in situations where treatment has been delayed may require hospitalization with IV fluid or oxygen support and may also require either a fresh whole blood or plasma transfusion to replace clotting factors. PT times should be monitored throughout therapy to determine if clotting factors are being produced.

Take home message with regard to all baits:

It is essential to determine what kind of "ratbait" an owner saw the animal ingesting. Always have them identify the box or package (don't rely on "I think he ate D-Con, Doc."), or call the exterminator service that put out the bait. Call poison control if you have any questions about the trade name of the product. Treat early and aggressively. Most importantly, like everything in medicine (as in life), a pint of client education is worth a dog's weight in treatment at the ER. Alerting owners to the presence of such powerful chemicals in their environment and the risk they pose to small children as well as pets reinforces our invaluable role as educators in the community.

Pyrethrin Toxicity

- Pyrethrins are chemical insecticides used to kill fleas on cats and dogs and the most common source of pyrethrin toxicity is through spot-on products, although a variety of sprays, shampoos, dips and mousses are available. Many pyrethrin products are readily available through grocery, discount or pet stores.
- Pyrethrins are derived from the chrysanthemum plant; pyrethroids are synthetic pyrethrin derivatives (e.g. resmethrin, allethrin, and permethrin).
- Pyrethrins alter the activity of sodium ion channels of nerves reversibly prolonging sodium conductance producing increased depolarization after potentials resulting in repetitive nerve firing.

- Toxicities can occur with pyrethrins by several means:
    - excess application of appropriate pyrethrin product
    - application of inappropriate product (i.e., the placement on cats of "spot-on" products labeled "for use on dogs-only")
    - adverse reaction to application of pyrethrin (Cats are exquisitely sensitive to pyrethrins)
    - ingestion of topical pyrethrin product

- While most pyrethrin products contain small amounts of pyrethroids and are considered safe for pets, "spot-on" products can contain 45-65% pyrethroids. These products are intended for canine use only; the consumer misuse of flea products placed on cats that are labeled "for use on dogs only" accounts for a large portion of the cases that present with tremors, twitching and hypersalivation.

Diagnosis:

- Diagnosis is typically made by a combination of clinical signs and history of exposure to pyrethrin products.

Prognosis:

- Although untreated seizures can be fatal, the prognosis for pyrethrin toxicosis is typically good with early treatment and supportive care. Most cases recover within a 24-hour period, although owners should be warned that fine muscle tremors may continue for several days.

Clinical signs:

- Clinical signs commonly seen with pyrethrin toxicosis include hypersalivation, muscle tremors, ataxia, anorexia, hyperexcitability, hyperthermia, vomiting, and diarrhea.

Treatment:

- Owners of pets who are showing symptoms after a flea product has been applied should bathe the patient with dish soap (i.e., Dawn) to remove the product from the skin. The patient should then be wrapped in a warm towel or blanket and brought to a veterinary hospital for further treatment.

- If alert and not neurologically impaired, oral administration of activated charcoal with a cathartic can be useful within the first two hours after pyrethrin application/ingestion. Repeat doses of activated charcoal are not indicated due to little or no enterophepatic recirculation of pyrethrins.

- IV catheter placement and fluid support.

- For management of muscle tremors:
    - Methocarbamol
- For management of seizure activity:
    - Diazepam: Consider CRI of diazepam if repeated doses needed to control seizures
    -
- Severe cases should be monitored at a 24-hour veterinary facility and may require moderate to advanced levels of nursing care and medical intervention to control seizures.

- Owners should always bring the flea product packaging to the veterinary office with the pet when they suspect pyrethrin toxicity.

Antidepressants/Serotonin Syndrome

"Serotonin Syndrome" is a disorder that can be caused by the ingestion of drugs or combinations of drugs that increase serotonin availability. Just a few of the drugs that fall into these categories are the tricyclic antidepressants such as amitryptiline and clomipramine as well as amphetamines, cocaine, buspirone, fentanyl, meperidine, and dextromethorphan. Serotonin Syndrome can develop after therapeutic use, overdose, or when two or more drugs which increase serotonin availability by different mechanisms are used simultaneously.

Diagnosis:

- No specific test can confirm Serotonin Syndrome, and there is no specific antidote.
- History of exposure in combination with suspect clinical signs.

Prognosis:

- Good to grave, depending on the type of drug ingested, the amount ingested and onset of treatment
- Medications with "pure" serotonergic action often have a good prognosis with early/aggressive therapy (i.e., 5-HTP)
- Medications with multiple actions/effects in addition to serotonergic action have more uncertain prognoses (i.e., TCA, amphetamines)

Clinical signs:

- Clinical signs that result from over-stimulation of serotonin receptors include mental status changes (i.e., confusion, incoordination) in addition to vomiting, difficulty breathing, drooling, seizures, arrhythmias, and tremors.
- Clinical signs can appear within minutes of ingestion and can progress to coma and death within a few hours.

Treatment:

- If ingestion within 2 hours, induce emesis.
- Administer activated charcoal:
- Place IV catheter and administer IV fluids to support blood pressure and blood flow to the kidneys.
- Manage Neuromuscular signs:
    - Seizures/Tremors:
        - Diazepam, Phenothiazines, Chlorpromazine, Acepromazine
        - Phenobarbital for refractive seizures
    - Muscle rigidity/Hyperthermia
        - If rigidity/tremors persist after sedation, consider methocarbamol
- Monitor/Manage Cardiovascular Issues
    - Typically improve with IV fluids sedatives and serotonin antagonists.
- Monitor/Manage GI signs (nausea, vomiting, diarrhea)
    - IV fluids
    - Antiemetics:Maropitant (Cerenia)
- Provide Serotonin Antagonist:

    - Cyproheptadine, an antihistamine that possesses serotonin antagonistic properties, has been used with success in human patients.
        - If unable to give orally; crush and give rectally
    - Chlorpromazine (as discussed in above treatments)

- It is important to closely monitor electrolytes, glucose, renal values, CPK and ECG.

Raisins/Grapes:

- Fresh grapes and grape pressings left over from wine-making have been shown as the cause in canine kidney failure in reports made to the National Animal Poison Control Center (NAPCC) over the past decade. Problems have occurred from dogs ingesting from 0.1 ounce per kilogram of body weight to 8 ounces per kilogram, showing a potential for a dog getting big trouble from eating very little.
- There are still many unknowns when it comes to grape/raisin toxicity: the toxic mechanism, how to test if it is present, why some dogs go into renal failure and some don't, and if it is an idiosyncratic reaction. What is known is the lowest amount causing ARF (0.7 oz/kg of grapes and 0.11 oz/g raisins). Typically, a raisin weighs 0.3 to 0.5 grams.

Diagnosis:

- The diagnosis is made through history of exposure, clinical signs and bloodwork. There is no specific test available for grape toxicity

Prognosis:

- If early and aggressive treatment is given, pets will often make a full recovery; severe oliguria or anuria has a poor prognosis.

Clinical Signs:

- Some dogs may be unaffected after exposure, some dogs may be affected by a few grapes, and other dogs may eat large quantities with no signs.
- After ingesting grape products, clinical signs seen in the first 24-36 hours in cases reported to NAPCC involved vomiting, diarrhea, anorexia, lethargy and abdominal pain; dogs often vomited within 6 hours of ingestion.
- In 36-72 hours of ingestion, 50 percent of the dogs developed anuric or oliguric renal failure and died. Within one to three days of eating the grape products, over half of the prior case reports showed evidence of kidney failure.

Treatment:

- There is no specific antidote for grape or raisin toxicity, and symptomatic and supportive care is recommended.
- Emesis should be induced if ingestion occurred within the first 8 hours.
- This should be followed by a single dose of activated charcoal.
- Baseline renal values should be obtained at admission.
- IV fluid therapy should be administered at 2X maintenance for 48 hours to keep the tubules clear.
- If renal values normal at 48 hours, wean off fluids, recheck renal values at 72 hours and send patient home if normal.
    - If ARF develops, specific and supportive treatment should be instituted
- Consider hemodialysis or peritoneal dialysis in severe cases where fluid diuresis in addition to medical therapy has failed to promote urine output.

Points to Ponder:

- When a dog has ingested grapes or raisins and the owner believes that the dosage falls below the dose for ARF, NAPCC recommends giving the owner the option to treat with emesis/AC and fluid diuresis. If they choose to decline any treatment and take the dog home, recommend they watch the dog closely for 24 hours. If the dog vomits (the first and most consistent sign of issues with grape/raisin toxicity), strongly advise that they return for IV fluid diuresis and hospitalization.

- In hospital, signs of renal toxicity from grape/raisin toxicity include changes in urine (granular casts, glucosuria) at approximately 18-24 hours and BUN/Creatinine elevations at 24-48 hours post ingestion.

- As opposed to EG toxicosis (which irreparably damages the basement membrane of the kidney), the basement membrane of the proximal renal tubular cells remains intact with grape/raisin toxicity. If an animal has ARF from grape/raisin toxicity, they potentially can have regrowth of damaged kidney cells if they are kept well hydrated and producing urine.

Xylitol

- Xylitol is a five-carbon sugar alcohol—a close cousin to sorbitol and mannitol. Because xylitol is unable to be fermented by oral bacteria to create tooth decay and has 40% fewer calories than sugar, dentists, diabetics and weight-watchers alike have pushed for its incorporation into candies, baked goods, puddings, oral rinses, toothpastes, and most prominently, sugar-free gum.

- Due to differences in xylitol metabolism, dogs ingesting this substance show not only hypoglycemia (from a large release of insulin) but also liver failure and coagulopathies. The two current mechanisms proposed for the injury to hepatocytes include either depletion of ATP via xylitol metabolites or the production of reactive and damaging oxygen species. Clotting dysfunction is often seen as a result of massive hepatic damage.

- As for the toxicity level for xylitol in dogs, hypoglycemia has been seen to occur at doses around 0.1 gram per kg and hepatic failure at 1.4-2.0 grams per kg. Using these formulas, if one major brand of pudding contains 7 grams of pudding in each container, then the ingestion of one container can cause hypoglycemia in a 50 kg dog and liver failure in a 12-pound dog. Due to the varied and often unknown amounts of xylitol in chewing gums, 2 to 3 sticks consumed by a 10 kg dog can be considered a toxic ingestion.

- As for other products where the amount of xylitol is listed on the label, any ingestion greater than 0.1 grams/kg should be seen and treated.

Diagnosis:

- Based on witnessed or suspected ingestion in combination with clinical signs and bloodwork.

Clinical Signs:

- Clinical signs include weakness (hypoglycemia), lethargy, vomiting, and ataxia which may progress to recumbancy, stupor, seizures.

Prognosis:

- Good to guarded depending on amount ingested, clinical signs, and overall underlying health of animal. When hepatic failure and coagulopathy are seen, prognosis becomes guarded.

Treatment:

- General decontamination measures including inducing emesis in alert animals: activated charcoal administration is not indicated as xylitol is a very small molecule and not bound by charcoal.

- Blood drawn and submitted for stat blood glucose, PCV/TS, electrolytes, and baseline liver values. Also recommend establishing baseline complete CBC/CHEM. If liver values are elevated or become elevated on rechecked bloodwork, recommend monitoring coagulation parameters.

- IV catheter and place on IV fluids containing dextrose.

- Glucose should be monitored every 2-4 hours for the first 24 hours: once blood glucose has normalized, recommend continuing fluids and monitoring the blood glucose and liver values off dextrose drips for 12-24 hours.

- For dogs with a known ingestion at or above hepatic necrosis levels, the initial treatment course outlined above is recommended in addition to frequent monitoring (Q6-8 hours) phosphorous, liver enzymes, and clotting status for at least 72 hours post-ingestion.

- Drugs that support liver function including S-adenosyl-L-methionine, silymarin, vitamin E, n-acetylcysteine, and ursodiol (as long as no evidence of biliary obstruction exists) could be beneficial and should be considered in cases with elevated hepatic enzymes.

- Antibiotics may be indicated in severe liver impairment:
    - Clavamox).Clavulanic acid OR Amoxicillin combined with Metronidazole

- For cases of protracted nausea and vomiting, consider antiemetics such as Maropitant (Cerenia)

- Consider GI protectants:
    - Famotidine (Pepcid)
    - Sucralfate (Carafate) *Separate from other medication by 1 hour to allow for absorption of other meds*

- Plasma or whole blood transfusions may be needed in cases of severe coagulopathy.

- If the ALT and bilirubin are measured at 72 hours and are normal or if there are only mild ALT changes, then the dog is likely "out of the woods." Increasing ALT, hyperbilirubinemia, hyperphosphatemia, or persistently elevated PT/PTT are bad prognostic indicators.

- *It is important to avoid drugs, or alter the dosage prescribed, for drugs that are primarily metabolized by the liver.*

Anti-inflammatories : Acetaminophen, Ibuprofen, Aspirin

- NSAIDs are commonly found in human medicine cabinets and as such, owners will often reach for them when they believe their pet is in pain. Besides inappropriate use of human NSAIDs or overdose of veterinary products, NSAIDs combined with patient risk factors or the combination of NSAIDs can cause clinical signs.

- Non-steroidal anti-inflammatory drugs reduce the production of prostaglandins and/or thromboxane. This makes them very good for decreasing inflammation and very bad for stomach lining and renal blood flow.

- While there are medications that help the liver metabolize acetaminophen, there are no specific antidotes for either acetaminophen or ibuprofen toxicosis.

Acetminophen Toxicosis:

- Acetaminophen is an active ingredient in many OTC meds such as Excedrin, Tylenol, Dristan, Anacin, Allerest, Sinutabs, Nyquil, etc. It is also an ingredient in prescription medications such as oxycodone (Percocet) and hydrocodone (Vicodin).

- Acetaminophen's mechanism of toxicity is through glutathione depletion in tissues, liver or RBC oxidative damage and through the direct binding of metabolites. The result of acetaminophen toxicity is hepatic damage or methemoglobinemia (dogs); hemolytic anemia/methemoglobinemia, hepatic, renal (cats).

Diagnosis:

- Witnessed or suspected ingestion of acetaminophen or acetaminophen-containing products.
- Compatible clinical signs with exposure history.
- Testing for serum levels available in some human hospitals/labs.

Clinical Signs:

- Lethargy, anorexia, hypersalivation, facial edema, icterus or brown mucous membranes, cyanosis, vomiting, dyspnea, tachycardia, weakness, depression.

Prognosis:

- Good to Grave depending on amount ingested and clinical signs.
    - Toxic Dose for Dogs: 100-150 mg/ kg
    - Toxic Dose for Cats: as low at 10 mg/ kg; consider any exposure to acetaminophen toxic to cats and recommend treatment.

**Cats have an increased sensitivity to acetaminophen relative to dogs and humans due to

a. Inability to conjugate glucuronic acid products with acetaminophen
b. Susceptibility of feline erythrocytes to methemoglobin formation

Treatment**:**

- Ask owner what product animal ingested, the strength of tablet, and time of ingestion.

- Calculate total dose and dose/kg.

- Draw blood for STAT PCV/TS and Critical Care Panel. Submit blood for CBC/Chemistry.

- If it is unknown whether ingestion occurred, a red top tube can be drawn and submitted for acetaminophen levels to a local hospital. The information will NOT tell if a level is toxic but will help determine if the animal ingested or absorbed any acetaminophen. ANY amount in a cat is toxic. Best to get blood submitted within 4 hours post ingestion; valid up to 12 hours but not worthwhile after 16 hours.

- If recent ingestion (within the last 4-6 hours) and animal is alert and not in respiratory distress, induce emesis and administer activated charcoal. This dose can be repeated every 4-6 hours for two doses if large amount acetaminophen ingested.

Remember: multiple doses of activated charcoal should NOT contain cathartics after the first dose due to the risk for dehydration and secondary hyponatremia.

- IV isotonic fluids at twice maintenance.

- If severely cyanotic, administer oxygen therapy
  *Extremely important not to stress cats—limit physical restraint and activity.

- N-Acetylcysteine ("Mucomyst")

- Ascorbic Acid (Vitamin C) treatments. Vitamin C is an antioxidant/free radical scavenger that aids in the conversion of methoxyhemoglobin to oxyhemoglobin; more important to use in cats.

- SAMe (S-adenosylmethionine—a sulfur donor that helps to replenish body stores of glutathione)

- Cimetidine (Tagamet) and famotidine (Pepcid) NOT routinely recommended for use in cats or dogs that have ingested toxic doses of acetaminophen as they inhibit the enzyme (NAT) that helps to detoxify acetaminophen metabolites.

- If patient is severely anemic and clinically affected (dyspneic), red blood cell transfusions may be indicated to increase oxygen carrying capacity of the blood.

Points to Ponder:

Animals with mild methemoglobinemia or mild hepatic injury often recover in 3-5 days; severe hepatotoxicity carries a poor prognosis.

Aspirin (Salicylates)

- Aspirin is the most common source of salicylates; salicylates can also be found in oil of wintergreen, Pepto-Bismol, Percodan, and BenGay.

- The therapeutic dose of aspirin in dogs is 10-20 mg/kg BID.

- The therapeutic dose in cats is 10-20 mg/kg q 48 hours. Cats are deficient in glucuronyl transferase and therefore have a prolonged excretion rate. For example, a dose of 25 mg/kg in a cat has an elimination half-life of almost 45 hours.

- The toxic dose in dogs 100 mg/kg (may see GI signs with 25 mg/kg), cats 50 mg/kg (also slower clearance from the body in cats: aspirin half-life is 7.5 hours in dogs compared to 44.6 hours in cats).

- Aspirin has multiple mechanisms of toxicity. It inhibits cyclooxygenase, reducing the synthesis of prostoglandins and thromboxanes (irreversibly inhibiting cyclooxygenase in platelets for their lifespan of 5-7 days). In addition to its anti-prostaglandin effect and inhibition of platelet aggregation, aspirin uncouples oxidative phosphorylation. This latter effect can cause an increase in oxygen consumption and carbon dioxide production—causing acid-base disturbances and potential multiple organ dysfunction

- Salicylates are metabolized primarily by the liver; young dogs with immature enzyme systems and cats (who have deficiencies in the glucuronyl transferase) are more susceptible to aspirin toxicity.

Diagnosis:

- Diagnosis is made through a combination of exposure (often deliberate dosing), clinical signs, and bloodwork findings.
- Blood salicylate levels can be measured, but levels won't always correlate with clinical signs.
- Evidence on radiographs (peptobismol tablets).

Prognosis:

- Recovery from salicylate toxicity is possible in most cases with aggressive treatment; early recognition/treatment is best.

Clinical signs:

- Minor exposure can have side effects of GI upset, GI ulceration/bleeding, or platelet dysfunction. CNS signs can range from altered mental state to coma, seizures, noncardiogenic pulmonary edema (tachypnea to dyspnea). Other generalized signs include vomiting, hematemesis, diarrhea, melena, lethargy, anorexia, dehydration, weakness.

Treatment:

- If the patient has recently ingested aspirin, then GI decontamination is recommended (emesis, gastric lavage, charcoal). Multiple doses of activated charcoal may be needed if large doses of salicylate were ingested or they were sustained release/enteric-coated).

- There is no primary antidote; treatment is aimed at supportive care and to optimize excretion via urine alkalinization.

- Prevent GI bleeding with GI protectants—the use of sucralfate in combination with H2 blockers (famotidine) or proton pump inhibitors.

- Misoprostal has also been shown to be helpful in preventing aspirin induced ulcers.

- Administration of IV fluids at maintenance rate can aid in excretion; monitor electrolytes to prevent hypokalemia. Consider u-catheter to monitor urine output.

- If significant bleeding has occurred, blood transfusions may be indicated

Ibuprofen Toxicity

- Ibuprofen is an active ingredient in many OTC meds such as Motrin, Advil, Mediprin, Nuprin, and Motrin IB.

- Ibuprofen mechanism of toxicity is through its inhibition of cyclooxygenase and a subsequent decrease in prostaglandin synthesis. When prostaglandins are inhibited, the kidneys suffer from excessive vasoconstriction, and there is a loss of the prostaglandin's cytoprotective effect on the stomach wall lining.

Diagnosis:

- Witnessed or suspected ingestion of ibuprofen; clinical signs and/or bloodwork in combination with clinical signs and exposure history.

Clinical Signs:

- Clinical signs include vomiting, anorexia, depression, diarrhea, ataxia, hematemesis, melena, tachypnea, oliguria, coma, seizures, respiratory depression.

Prognosis:

- Good to Grave depending on amount ingested, time elapsed, and clinical signs.

- Toxic Dose for Dogs: 50 to 100 mg/kg—GI ulceration / hemorrhage
  175 mg/kg—Acute renal failure
  400 mg/kg—Neurologic signs
  600 mg/kg—LD 50
- Toxic Dose for Cats: 50 mg/ kg

- Lower doses result in clinical signs of GI irritation and hemorrhage. Ingestion of greater than 300 mg/kg for both cats and dogs can result in signs of acute renal failure in addition to GI irritation—renal signs may occur as early as 12 hours after exposure or as late as 3 to 5 days. In animals ingesting more than 600 mg/kg clinical signs include respiratory depression, stupor, coma, seizures, and death. ***

- Consult chart at end of protocol for toxic doses of other NSAIDS.

****Remember that animals with pre-existing renal insufficiency can have problems at lower doses.*

Treatment:

- Ask the owner what product animal ingested, the mg dosage of the product, how many tablets (if they can try to estimate from the bottle's contents), and the time of ingestion.

- Calculate the total dose and dose/kg.

- Draw blood for state blood gas and Critical Care Panel; submit blood for CBC/ Chem panel

- If exposure suspected BUT not known, can consider submitting red top tube for ibuprofen level to regional laboratory for ibuprofen level.

- If recent ingestion (within 2-4 hours) and animal is alert, induce emesis and administer activated charcoal. Administer repeat doses of activated charcoal Q 4 to 6 hours for 24 hours due to enterohepatic circulation

***Remember—multiple doses of activated charcoal should NOT contain cathartics (sorbitol) after the first dose due to risk for dehydration and secondary hypernatremia. (Use the BLUE label)*

- IV isotonic crystalloids such as Norm R or LRS @ 2-3 times maintenance.

- GI Protectants:
  - Misoprostil, Famotidine, Sucralfate
- If vomiting, consider antiemetic: Cerenia: Dogs and Cats

- If significant bleeding has occurred due to large amount of NSAID ingestion, blood transfusions may be indicated.

- If renal values are normal at 48 hours, wean off fluids over 24 hours and repeat renal values. If the renal values are elevated, then continue on fluids until normal. If renal values normal, send pet home on sucralfate and famotidine for 10-14 days.

Chocolate Toxicosis

- Chocolate, caffeine stimulant tablets, headache medications, coffee and coffee beans, herbal weight loss supplements and energy boosters containing guarana, cocoa powder, and cocoa bean mulch all contain theobromine and caffeine.

- Depending on the amount an animal ingests, these chemicals affect the central nervous system, the heart, the kidneys, the musculoskeletal system and the gastrointestinal tract.
- Dogs do not metabolize and excrete theobromine as well as humans.

Diagnosis:

- Diagnosis of chocolate or caffeine toxicity is most often made by the combination of compatible history and clinical signs. Supporting evidence can come from stomach contents (i.e., presence of chocolate in vomitus).

Prognosis:

- Good to guarded depending on amount ingested, clinical signs and overall underlying health of animal.
    - Potentially toxic/ lethal dose for dogs is 100-250 mg/ kg of theobromine.
    - Typically, doses of 20-40 mg/kg of theobromine causes vomiting and diarrhea whereas doses higher than 40 mg/kg cause more severe clinical signs.
    - This equals approximately 2 ounces milk chocolate/kg body weight or 0.2 ounces baking chocolate/kg body weight

Clinical Signs:

- Typical signs seen with chocolate or caffeine ingestion include restlessness, hyperactivity, vomiting and diarrhea, tachycardia, weakness, diuresis, tremors, seizures, incontinence, diarrhea.

Treatment:

- Obtain full history from owner as per amount, type, and time of chocolate ingestion.

- Weigh dog and determine amount of theobromine/kg ingested:

    - Consult one of the following to calculate the potential toxicity of the chocolate ingested by your patient. Each option requires that you enter the canine's body weight in addition to the type of chocolate and amount of chocolate ingested.
        - The chocolate toxicity table at the end of the chocolate protocol.
        - Type into "VIN" or your computer search engine: "Chocolate Toxicity Calculator."
        - Use a Chocolate Toxicity iPhone App.

- There is no specific antidote for chocolate or caffeine toxicosis and treatment is symptomatic. It is important to prevent further absorption, hasten elimination, and maintain basic life support.

- If dose is within toxic range *or* if animal is showing clinical signs, recommend hospitalization, treatment, and monitoring to owner.

- If the patient is comatose or convulsing, gastric lavage should be performed—otherwise, vomiting should be induced as soon as possible. Because the half-life of theobromine is long and because chocolate tends to stay in a solid or semiplastic state in the stomach, repeated doses of activated charcoal should be administered every 4 hours for up to 72 hours after ingestion.

- Start IV fluids at twice the maintenance levels and obtain blood for a critical care panel. If the dog has a history of pancreatitis or if the dog has had persistent vomiting or abdominal pain since ingestion, draw blood and submit to lab for CBC/CHEM with lipase/amylase.

- The EKG should be closely monitored and if indicated, VPCs and tachycardia can be controlled with lidocaine or metoprolol (*Propranolol delays excretion of methylxanthines so use metoprolol succinate or tartrate).

- If the patient is seizuring, diazepam is the initial drug of choice followed by barbiturate therapy.

- Fluid therapy, close continuous monitoring of cardiac and neurologic status and supportive care are essential to a successful outcome. It may take up to four days for the theobromine to clear the pet's body.

Points to Ponder:

With so many forms of chocolate on the market, it can be challenging to determine whether or not your patient's dietary indiscretion could do them serious harm. The lethal dose of caffeine: 110-200 mg/kg (dog), 80-150 mg/kg (cat); lethal dose theobromine: 250-500 mg/kg (dog)—doses much lower than this may cause clinical signs. The darker the chocolate, the higher the content of theobromine, and thus the greater potential for serious problems in our pets.

Chocolate Toxicity Table: submitted by Stephanie Coryell on VIN

Milk Chocolate **(60 - 66 mg/oz or 2.12 - 2.33 mg/gram of methylxanthines)**

Weight		**Mild Reaction**		**Moderate to Severe Reaction**	
5 lbs	2.27 kg	0.75 oz	21.26 gm	1.5 oz	42.53 gm
10 lbs	4.5 kg	1.5 oz	42.53 gm	3.0 oz	85.05 gm
20 lbs	9.1 kg	3.0 oz	85.05 gm	6.0 oz	170.10 gm
30 lbs	13.6 kg	4.5 oz	127.58 gm	9.0 oz	255.15 gm
40 lbs	18.2 kg	6.0 oz	170.10 gm	12.0 oz	340.20 gm
50 lbs	22.7 kg	7.5 oz	212.63 gm	15.0 oz	425.25 gm
60 lbs	27.3 kg	9.0 oz	255.15 gm	18.0 oz	510.30 gm
70 lbs	31.8 kg	10.5 oz	297.68 gm	21.0 oz	595.35 gm
80 lbs	36.4 kg	12.0 oz	340.20 gm	24.0 oz	680.40 gm

Semi Sweet Chocolate **(150 mg/oz or 5.29 mg/gram of methylxanthines)**

Weight		**Mild Reaction**		**Moderate to Severe Reaction**	
5 lbs	2.27 kg	0.3 oz	8.51 gm	0.6 oz	17.01 gm
10 lbs	4.5 kg	0.6 oz	17.01 gm	1.2 oz	34.02 gm
20 lbs	9.1 kg	1.2 oz	34.02 gm	2.4 oz	68.04 gm
30 lbs	13.6 kg	1.9 oz	53.87 gm	3.6 oz	102.06 gm
40 lbs	18.2 kg	2.1 oz	59.54 gm	4.2 oz	119.07 gm
50 lbs	22.7 kg	2.5 oz	70.88 gm	5.0 oz	141.75 gm
60 lbs	27.3 kg	3.8 oz	107.73 gm	7.6 oz	215.46 gm
70 lbs	31.8 kg	4.2 oz	119.07 gm	8.5 oz	240.98 gm
80 lbs	36.4 kg	4.8 oz	136.08 gm	9.6 oz	272.16 gm

Baking Chocolate **(~450 mg/oz or 15.87 mg/gm of methylxanthines)**

Weight		**Mild Reaction**		**Moderate to Severe Reaction**	
5 lbs	2.27 kg	0.1 oz	2.84 gm	0.2 oz	5.67 gm
10 lbs	4.5 kg	0.2 oz	5.67 gm	0.4 oz	11.34 gm
20 lbs	9.1 kg	0.4 oz	11.34 gm	0.8 oz	22.68 gm

30 lbs	13.6 kg	0.6 oz	17.01 gm	1.2 oz	34.05 gm
40 lbs	18.2 kg	0.8 oz	22.68 gm	1.6 oz	45.36 gm
50 lbs	22.7 kg	1.0 oz	28.35 gm	2.0 oz	56.70 gm
60 lbs	27.3 kg	1.2 oz	34.05 gm	2.4 oz	68.04 gm
70 lbs	31.8 kg	1.4 oz	39.69 gm	2.8 oz	79.38 gm
80 lbs	36.4 kg	1.6 oz	45.36 gm	3.2 oz	90.72 gm
NOTES:					
* Mild reactions may be seen at ~ 20 mg/kg					
* Moderate to severe reactions may be seen at doses over 40 mg/kg					
* Cardiotoxicity may be seen at ~ 50 mg/kg					
* Seizures are possible at doses over 60 mg/kg					
* Any dose over 40 - 45 mg/kg should be considered potentially life-threatening					
* 100 mg/kg is the LD50, meaning that at this dose half of the animals will die; animals can die from exposures well below the LD50					

Marijuana

- Marijuana's active ingredient is delta 9-tetrahydrocannabinol (THC); THC targets the brain, interacting with central neurotransmitters (norepinephrine, dopamine, serotonin, acetylcholine) and binding to receptors in the cerebellum and frontal cortex.
- THC is absorbed rapidly and eliminated slowly; forewarn owners that signs (and treatment required) can potentially last several days.
- Most cases of marijuana ingestion involve dogs; it is rarely reported in cats.

Diagnosis:

- Diagnosis is typically made based on suspicious clinical signs combined with history of exposure.
- Owners will rarely volunteer a history of illicit drug ingestion; more commonly the history involves the dog "getting into something" at a park, party, or friend's house.
- These cases typically do not present before clinical signs are evident when the owner notes that their pet is acting strange, is depressed, or exhibits an acute onset of ataxia.
- THC can be detected on a urine drug screen.
    - There are no veterinary approved ones at this time.
    - Human ones haven't really been validated
        - More likely false negative than false positive
        - Anecdotal reports: veterinarians have had luck with the Multi-Drug Screen Test from Medical Disposable of PR

Prognosis:

- Prognosis post marijuana ingestion is usually good as THC has a large therapeutic index: a reported minimal lethal oral dose of d-THC in the dog is > 3 g.

Clinical Signs:

- Clinical signs of marijuana ingestion include depression, ataxia, tremors, weakness, vomiting, tachycardia/ bradycardia, seizures, coma.

Treatment:

- If the ingestion was recent (witnessed or suspected within 30 minutes) and the patient is alert, induce emesis.
- After clinical signs of intoxication appear, induction of vomiting is difficult due to the nausea control properties of THC.
- Administer activated charcoal and give repeated doses every 8 hours for the first 24 hours due to extensive enterohepatic circulation.
- Administer IV fluids at a maintenance rate
- Placing the marijuana intoxicated animal in quiet and dark environment helps mediate agitation.
- If the patient requires medication to alleviate anxiety, administer diazepam at low doses.

Points to Ponder:
THC is absorbed rapidly and eliminated slowly; forewarn owners that signs (and treatment required) can potentially last several days.

Ethylene Glycol / Antifreeze

- Ethylene Glycol is a colorless, odorless, sweet tasting liquid used primarily as an antifreeze and windshield de-icing agent.
- Antifreeze in small doses can cause severe damage: approximately 3 tablespoons can be lethal to a 10 kg dog.
- The most profound consequence of ethylene glycol poisoning is acute renal failure. Antifreeze poisoning can cause metabolic acidosis, acid-base imbalances, central nervous system dysfunction, and gastrointestinal irritation that results in vomiting, nausea, and weakness.

Diagnosis:

- Witnessed ingestion of antifreeze.
- Access to antifreeze (or other ethylene glycol containing product such as windshield de-icing agents, photo developing solutions, brake fluid/motor oil, and some inks, paints and stains) and showing clinical signs as outlined below.
- Analysis of a blood sample either using an in-veterinary clinic kit or by sending out testing to a local human hospital.
- Also, urine samples can be evaluated for monohydrate calcium oxalate crystals. Ethylene glycol is rapidly metabolized and is not detectable in serum or urine 48 to 72 hours after ingestion.

Clinical Signs:

- Antifreeze poisoning can cause metabolic acidosis, acid-base imbalances, central nervous system dysfunction and gastrointestinal irritation which result in vomiting, nausea, and weakness.
- 30 minutes -12 hours post-ingestion: vomiting, hypothermic, ataxic/knuckling “drunken” behavior.
- 12 hours-36 hours post-ingestion: above clinical signs but dogs may appear “recovered”; cats remain depressed and may already exhibit renal failure.
- 36-72 hours post-ingestion: anorexia, depression, coma, seizures, renal failure.

Prognosis:

- VERY TIME DEPENDENT—Important to communicate to owners on the phone/on admit that ethylene glycol toxicity has a high potential for lethal outcome and to stress that time is of the essence in diagnosis and treatment.
- When dogs are treated aggressively and with 4-methylpyrazole within 5 hours of EG ingestion (cats = 3 hours), then prognosis for recovery is good/excellent.

- Once azotemia is present, prognosis is very guarded; oliguria or anuria carry a very poor to grave prognosis. In these patients, peritoneal dialysis or hemodialysis can offer a chance for recovery if owners commit to this long term (and often expensive) therapy.

Azotemia = poor-grave prognosis

Treatment:

- Place an IV catheter and obtain blood for the following tests:
  - Ethylene Glycol test
  - STAT blood gas, electrolytes, calcium, creatinine, osmolality, PCV/TS
  - CBC, Chemistry Panel, and U/A and submit to lab:
    *cats can be azotemic by 12 hours, dogs by 36-48 hours

Ethylene glycol and ethanol are small molecules which are poorly bound by activated charcoal. However, activated charcoal is still recommended as it might provide some benefit in detoxifying cases not yet affected with vomiting or CNS signs.

- Obtain urine for urinalysis:
  - Calcium oxalate crystals can possibly be observed at 3 hours post-ingestion in the cat and 6 hours post-ingestion in the dog.
  - Place an indwelling urinary catheter and monitor fluid ins and outs.

- Begin replacement fluid therapy with a balanced electrolyte solution @ 2-3 times maintenance
- **Choose antidotal therapy, ethanol or 4-methylpyrazole, and administer before toxic metabolites are generated by the liver!**

In addition to receiving an antidote, animals who have ingested ethylene glycol benefit from supportive care including the correction of fluid, acid base, and electrolyte imbalances, gastric protectants, and renal specific therapy if indicated.:

- If pH of <7.1 or HC03 of <11, consider sodium bicarbonate administration.
- Give B-complex vitamins in the daily fluids—1 to 2 mls/liter.

- If <1ml/kg/hr of urine production: Place central line for CVP measurements. Give crystalloids to increase CVP by 5 cm H2O (if initial CVP not too high).
  - If urine output not increasing with increase in crystalloids, consider administering lasix, dopamine or mannitol.

- If responding but requiring extended treatment, consider PN.

- Consider antiemetics.

- Consider dialysis for animals who are 24 hours or more post-ingestion, who are unresponsive to fluid therapy alone, or who are azotemic, oliguric or anuric. *Consider this ONLY if owners are highly motivated and understand poor prognosis and high financial cost involved.
- *Can also consider dialysis to remove ethylene glycol in the early phases of toxicity—if the animal is already azotemic, if the ethylene glycol blood level was high, and if the owners understand the substantial financial commitment.*

Points to Ponder:

- Ethylene Glycol poisoning is the second most common cause of fatal poisoning in animals according to the American Association of Poison Control Centers.
- Although there is a very high potential for lethal results, patients' lives can be saved by early recognition of exposure and quick administration of an antidote

- Due to concern for CNS depression, respiratory suppression and acidosis, NAPCC recommends 7% CRI > 20% bolus method for ethanol administration. If bolus method is chosen, monitor patient closely for respiratory suppression, vomiting, aspiration.

- Recommend recheck ethylene glycol test post-treatment; discontinue antidotal therapy once test is negative or blood EG level is <50 mg/dL.

- Recommended that an in-line filter be used for the IV use of either ethanol or 4-MP.

- The client should be informed of the use of a non-pharmaceutical product (ethanol) and give their informed consent.

- The fluid of choice to use with the ethanol is 5% dextrose in 0.9% NaCl.

Lily Toxicosis:

- Several members of the Lily family including Easter Lilies (*Lillium longiflorum)*, daylilies (*Hemerocallis)*, tiger lilies *(Lilium tigrinum*), and oriental and Asiatic lilies *(Lilium speciosum* and *Lilium lancifolium*) have been reported to cause kidney failure in cats.

- The exact water soluble toxic principle of the lily, as well as the toxic dose for cats, is unknown.

- All parts of the plant are toxic, including the stems, stamen, leaves, petals, and pollen. Even a very small amount ingested by a cat (a bite of a leaf or a lick of pollen off the fur) can be fatal.

- The lily's toxin targets the renal tubular epithelium of the cat.

- If there is any question as to whether a cat has ingested any part of a lily, the owner should be instructed to have the cat seen by a veterinarian immediately.

Diagnosis:

- Diagnosis is based on witnessed or potential exposure and clinical signs. There is no laboratory test currently available for definitive diagnosis of lily toxicosis.

Prognosis:

- Early and aggressive treatment (within 18 hours of exposure) typically yields a good prognosis. If cats are symptomatic with anuric renal failure, prognosis is guarded to poor.

Clinical Signs:

- The clinical signs of cats with lily intoxication include salivation, vomiting, anorexia, dehydration and depression. Cats typically develop acute renal failure within 24 to 72 hours post-exposure.
- Within 12 hours of exposure, elevations in creatinine, BUN, phosphorous and potassium are detectable. Within 24 hours of exposure, proteinuria, glucosuria and isosthenuria and the presence of casts are often seen.

Treatment:

- If the ingestion was recent (witnessed or suspected within 4 hours), induce emesis.
- Administer activated charcoal with cathartic—30 minutes after emesis induced.
- Start IV fluids at twice the maintenance levels and obtain blood for baseline renal values.
- Renal values should be repeated at 24 and again at 48 hours post exposure.
- Antiemetics and GI protectants.

Points to ponder:
As in grape/raisin toxicity, the lily nephrotoxin does NOT induce an irreparable injury to the renal tubular basement membrane and the regeneration of damaged tubules is possible. In severe cases of polyuric or oliguric renal failure, continued diuresis can help maintain kidney function until tubular regeneration occurs (10-14 days or longer). Although peritoneal or hemodialysis can be considered once anuric renal failure develops, prognosis for recovery becomes poor.

TREMOROGENIC TOXICITIES: Mycotoxins and Metaldehyde

Metaldehyde Toxicosis:

Metaldehyde is a molluscicide that comes in liquid, pellet, granular or powder forms and is the most popular form of slugbait available. It works by destroying the mucus-producing system of slugs which reduces their digestion and mobility. It is toxic to animals by dermal exposure, inhalation and—most severely—by ingestion. Although the there are several mechanisms by which metaldehyde is toxic to animals, the main clinical signs are likely a result of its alteration of neurotransmitter levels (GABA) and decreased concentrations of serotonin (5-HTP) in the brain.

Because of its toxic effects, the World health organization classifies metaldehyde as a Class II moderately hazardous pesticide. The EPA classifies metaldehyde as a "Restricted Use" pesticide because of its potential long and short term effects on wildlife. The label *"This pesticide may be fatal to dogs or other pets if eaten. Keep pets out of treated areas."* must be placed on the front panel of all metaldehyde products. Despite these warnings and industry standards, most pet owners either don't read the often small warning label or don't comprehend the damage metaldehyde can do to their companion animals

These baits are often pelleted and sweet-tasting which make them appetizing to dogs as well as slugs and snails. A very small amount of metaldehyde, less than a teaspoon per 10 pounds of body weight, can cause poisoning in dogs.

Diagnosis**:**

- Witnessed or suspected ingestion of slugbait.

- There is no specific test for metaldehyde toxicity and the diagnosis is made based on possible exposure to slug bait combined with the characteristic twitching appearance of the patient.

Prognosis:

- Good to Grave: the toxic dose for dogs ranges from 200-600 mg/kg and 207 mg/kg in cats.

- Depending on the amount of toxin ingested and severity of signs, untreated animals can die from respiratory paralysis within hours to days. Liver failure may also occur in 2-3 days after exposure.

- Prognosis is good with thorough decontamination of metaldehyde from the gastrointestinal tract followed by early and aggressive treatment.

Clinical Signs:

- Metaldehyde affects the brain and clinical signs can occur rapidly—often within one hour of ingestion. Early clinical signs include tachycardia, anxiety, and restlessness; these are often followed by followed by vomiting, hypersalivation, ataxia, tremors, hyperthermia, tachypnea, opisthotnous, and tonic convulsions. Symptoms can progress to blindness, liver failure, respiratory failure, and even death.

Treatment:

- Treatment goals are to decontaminate the patient, stabilize vital signs (especially hyperthermia associated with tremors), control tremors and seizures, and address metabolic acidosis.

- If the ingestion was recent (witnessed or suspected within 4 hours) and the patient is alert, induce emesis and administer activated charcoal. If the animal is severely altered neurologically, consider gastric lavage and activated charcoal via stomach tube.

- IV catheter should be placed, IV fluids administered at twice maintenance, and blood work submitted for stat critical care panel (*consider full CBC/Chemistry depending on status/history of the patient).

- Administer muscle relaxants for tremors: methocarbamol (Robaxin)

- Administer anxiolytic/anticonvulsants: Diazepam or Midazolam

- If seizures are persistent and poorly controlled with above medications, consider either phenobarbitol or pentobarbital IV to effect. * Concern with using barbiturates is that they can interfere with the metabolism of metaldehyde (* they compete for the enzymes that degrade acetaldehyde, a metabolite of metaldehyde). Avoid using barbiturates unless the seizures can not be controlled with diazepam.

- Frequent enemas have been shown to hasten the elimination of metaldehyde and to decrease its reabsorption from the colon.

- If in respiratory failure from slugbait *or* if a deep plane of anesthesia is needed to control tremors/seizures, intubate and ventilate.

Points to Ponder:
This scenario can be avoided altogether if non-toxic alternatives to metaldehyde are used: some are commercially available and some are home made. Crushing eggshells or oyster shells and placing them around the perimeter of specific garden pots or plants creates a barrier effect for the vulnerable soft underside of slugs. Beer or yeast placed in small tins sink in the ground at plant level entices the slugs and entraps them—just remember to remove and rebait the traps every 1-2 days. Commercially available slug traps that are more visually appealing (colored discs / animal shapes) can also be purchased at nurseries and garden stores. Copper bars or wires used as edging around plants or pots is a natural albeit expensive repellant to slugs and snails. Once ingested, Iron phosphate pellets (found under commercial names "Sluggo," "Escargot," or "Worry-Free") causes snails to cease feeding and die within 3 to 6 days. That bait, just like metaldehyde, needs to be reapplied after a heavy rain and/or approximately every 2 weeks.

Mycotoxins

While mycotoxin ingestion is typically thought to be a problem of the free-roaming dog in suburbia or the countryside, household pets who have access to spoiled food are just as likely to become "garbage hound" victims. Moldy nuts, grains, pasta, and dairy foods are common sources of tremorogenic mycotoxins. While over 20 different mold toxins have been shown to produce compounds that are capable of producing muscle tremors in animals, the penicillium species are the most likely culprits in food poisonings of pets. Penitrem-A and roquefortine C are the most common neurotoxins that can

induce produce varying degrees of muscle tremors, ataxia, and seizures that can last for several hours to several days. The mechanism of each tremorgenic mycotoxin has not been determined but many investigators propose that they center on creating a reduction of central inhibitory neurotransmission; penitrem A is known to antagonize the production of the inhibitory neurotransmitter glycine in mice.

Diagnosis:

- Diagnosis is based on witnessed ingestion or history of potential exposure in combination with characteristic twitching or tremors. Definitive diagnosis of mycotoxin ingestion is rarely performed for several reasons: (1) There is no specific blood test, (2) Mass spectrometry of the vomitus and gut contents is expensive and takes several days to complete, and (3) As there is no specific antidote for tremorgenic mycotoxicosis, treatment is supportive and not dependent on a particular toxin ingested.

Prognosis:

- Typically good, especially if decontamination is early and thorough

Clinical Signs:

- The typical clinical signs produced by tremorgenic mycotoxins including anxiety, restlessness, hypersalivation, and mild to severe whole-body muscle tremors.

Treatment:

- Treatment for mycotoxin poisoning is essentially the same as for metaldehyde toxicosis: early and aggressive decontamination of the patient, stabilize vital signs (especially hyperthermia associated with tremors), control tremors and seizures and address metabolic acidosis.

Points to Ponder:
Due to the extensive enterohepatic recirculation that occurs with the mycotoxin penitrem-A, owners should be informed that treatment may be prolonged.

Cyanobacteria

Another name for this is "Blue-Green Algae". Do not let dogs swim/play or ingest standing water with algae blooms. Some of the algae in this type of water contain potent cyanotoxins. These toxins affect the nervous system and liver and can be rapidly fatal. Clinical signs include vomiting, weakness, diarrhea, muscle rigidity, tremors, seizures and paralysis. There is no antidote so the best treatment is prevention! It is best to avoid all stagnant water and any water with a "green film" on top.

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