CHAPTER 10

Toxicologic Decontamination

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This chapter explores multiple decontamination procedures. Other chapters in this text cover the use of specific antidotes, supportive care, and emergency management of the poisoned patient. Owners and staff should be advised to protect themselves from toxic exposure when decontaminating a patient; this principle is particularly true with dermal toxins and toxins that are easily volatilized. An important rule in toxicologic decontamination is not to contaminate the decontaminators. The aim of successful decontamination is to inhibit or cease further toxin absorption and enhance elimination from the body.

Ocular Decontamination

Contaminated eyes should be flushed with copious quantities of water or physiologic saline. The eyes should be flushed for a minimum of 15 minutes; often multiple flushings are required. Mild sedation or a short-acting anesthetic may be necessary to control the patient before flushing. As a first-aid measure, owners should be advised to flush contaminated eyes at least 15 minutes before transporting the patient to the veterinary facility for complete evaluation and specific medical intervention.

Dermal Decontamination

As already mentioned, it is paramount for the veterinarian and paraprofessional staff to protect themselves from dermal exposure when transporting or decontaminating the dermally exposed patient. Rubber gloves and aprons should be used by the attending staff. Toxic dermal exposures allow both transdermal absorption and oral exposure if the patient tries to clean itself by licking.

Long-haired patients may benefit from having the hair clipped before cleaning the skin. Washing with a mild soap or detergent usually removes most toxins. Several washings may be required. Oily substances can often be removed with commercial hand-cleaning degreaser compounds, such as Gojo or Goop. These compounds themselves may be toxic if ingested and should be removed by washing with warm water and soap. Hypothermia is possible, and the patient's overall physical status should be continually evaluated while decontamination procedures are ongoing.

Special care is necessary when decontaminating acid and caustic burns. Toxin-exposed skin should be copiously flushed with tepid water. The damaged tissue is easily traumatized, and extreme care should be taken not to add mechanical injury to the wound when removing the toxin from the skin.

Gastrointestinal Decontamination

Emesis

The effectiveness of vomiting and the percentage of the gastric contents recovered depend on several factors. These factors include the physical properties of the toxin ingested, the

time elapsed from toxin ingestion to induction of emesis, the volume of gastric contents, and the emetic agent used.

Emesis is contraindicated when the risk of aspirating vomitus is great. This includes situations in which the patient is stuporous, neurologically depressed, or unconscious or at risk of becoming so before the emetic can work. Additionally, if the animal is having seizures or is at serious risk of a seizure before the emetic works, the risk of aspiration is increased. No emetic should be employed when oil, gasoline, or other petroleum distillates (e.g., volatile compounds) have been ingested. Emesis is contraindicated if vomiting will re-expose the mouth, pharynx, larynx, or esophagus to additional injury from ingested caustic or corrosive compounds.

Studies have indicated that the more rapidly emesis is induced, the greater the percentage of recovery of gastric contents.[1-4] In one study in dogs, the maximum toxin recovery reported was 75% (range, 9% to 75%) with a mean recovery of 49% when emesis was induced within 11 to 30 minutes after toxin ingestion.[1] Generally, gastric recovery in dogs ranges from 17% to 62% if emesis is induced within 60 minutes after toxin ingestion.[1-4] In humans, mean toxin recovery achieved by emesis induction ranged from 21% to 60%.[5,6] In all these studies, the emetic was administered within 60 minutes after toxin ingestion.

Emesis has been successful in removing toxins when it is administered longer than 1 hour postingestion; however, it should be stressed that the volume recovered decreases significantly as time passes. Inducing vomiting more than 4 hours after toxin ingestion is generally of little value.

The usefulness of emetics also depends on the toxic compound ingested. If the toxin has strong antiemetic activity, emetics may be ineffective. As a general rule, if a specific emetic fails to induce vomiting after two doses, it will not be effective. For example, apomorphine, a potent emetic, works by stimulating the chemoreceptor trigger zone in dogs. However, it also directly depresses the emetic center, so if the initial dose fails to induce emesis, subsequent doses are not likely to be effective. Additionally, emetics themselves can be toxic if normal dosages are exceeded.

Emetics are usually classified into two groups—those used by the owner at home as a form of first aid and those used by the veterinarian in the medical facility. Home-use emetics include 7% ipecac syrup, 3% hydrogen peroxide (H_2O_2), liquid dishwashing detergent, and table salt (NaCl). Veterinary emetics include apomorphine, xylazine, and several other less commonly used compounds in cats (e.g., dexmedetomidine, cefazolin, hydromorphone, and midazolam).

I do not routinely advise home induction of emesis. Often the time spent by the owner finding the emetic and then catching and trying to dose the animal usually consumes more time than it would take to drive promptly to the veterinary facility. Generally, home-administered emetics usually succeed only in delaying prompt medical evaluation and specific therapeutic intervention. Additionally, home emetics may not be as successful as clinically available compounds. Owners often miscalculate the weight of the animal and can be either too passive or too aggressive in calculating the dose and administering the compound. The toxin ingested may cause mental depression before the home-administered emetic takes effect, leaving the patient with no airway protection. The risk of aspiration and other secondary side effects of available home emetics is significant. Generally, most owners can transport the animal to a veterinary facility within 30 to 45 minutes. Often the best first aid is rapid transport of the patient and toxin container to the nearest veterinary facility, where prompt induction of emesis is possible.

Home-Use Emetics

7% Syrup of Ipecac

Syrup of ipecac is derived from the dried root of *Cephaelis ipecacuanha,* which is indigenous to South America. The active alkaloids are emetine and cephaeline. This product should not be confused with ipecac fluid extract, which is 14 times stronger. Outdated products can be used, but may be less effective. The mechanism involved is direct gastric

irritation and stimulation of the chemoreceptor trigger zone. The dosage for dogs is 1 to 2 mL/kg orally (PO); the dosage for cats is 3.3 mL/kg. Some recommend that the cumulative dosage should not exceed 15 mL in either species. The dose can be repeated once. Effective vomiting should result within 10 to 30 minutes, although it can be delayed for up to 1 hour.

Problems with syrup of ipecac include difficulty in administering the compound to conscious patients because of its bitter taste, which is particularly repugnant to cats. Also, a lack of effectiveness in inducing emesis in 50% of small animal patients has been observed. Additionally, if the patient fails to vomit, the ipecac should be removed from the stomach by lavage because of its potential cardiotoxic arrhythmia-inducing action. In humans the most common complications related to ipecac administration are diarrhea, lethargy, depression, and prolonged vomiting. The use of the emetic is not routinely recommended.

3% Hydrogen Peroxide

Hydrogen peroxide is most effective if it is administered after a small meal. Recommended dosage in dogs and cats is 1 to 5 mL/kg PO (alternatively, 1 mL per pound), generally not to exceed 50 mL for dogs (although many veterinarians exceed this total dose with negligible complications reported) or 10 mL for cats. The mechanism of action is believed to be gastric irritation. There are no significant risks from H_2O_2 ingestion; however, there have been recent concerns regarding the use of this in cats inducing a hemorrhagic gastritis. Because of this, some toxicologists do not recommend the use of hydrogen peroxide as an emetic in cats; and if it is used, it is not recommended to repeat the dose. This risk, albeit low, appears to be even lower in dogs. However, the owner should be careful to prevent the patient from aspirating the hydrogen peroxide when it is being administered. If it is successful, 3% hydrogen peroxide usually induces emesis within 10 minutes. If it is unsuccessful, the dose can be repeated in dogs.

Sodium Chloride (Table Salt)

The recommended dosage in dogs and cats is 1 to 3 tsp PO. Sodium chloride acts as a direct gastric irritant. It usually induces emesis within 10 to 15 minutes. Several negative sequelae are possible with administration of sodium chloride, including hypernatremia, particularly in young animals, if emesis is not induced. Additionally, hematemesis can occur. In children oral sodium chloride administration has produced hypernatremia, cerebral edema, and convulsions. If emesis induction is unsuccessful, lavage should be considered to remove the excess salt. If emesis is successful, water should be available as needed. The use of table salt as an emetic in companion animals is actively discouraged because of the potential for inducing more harm than good.

Liquid Dishwashing Detergent (e.g., Palmolive, Dove, Ivory)

The recommended oral dosage in dogs and cats is 10 mL/kg body weight of a mixture of 3 tbsp detergent to 8 oz of water. Vomiting usually results within 20 minutes after administration. Detergents containing phosphate are most effective. The mechanism of action is primarily gastric irritation. In one human trial, liquid dishwashing detergent appeared to be safe, and emesis was initiated in 83% of patients compared with 97% of controls given ipecac.[7] The effectiveness of liquid detergent in the veterinary population has not been fully elucidated. These detergents should not be confused with caustic detergents, such as electric dishwasher soap or laundry detergent, which are alkaline and are extremely caustic and could induce serious injury to the upper gastrointestinal tract.

Veterinary Emetics

One advantage of "veterinary emetics" is the possibility of using them in conjunction with activated charcoal. Once emesis has been induced, the emetic can be readministered after the patient has received a dose of activated charcoal.

Apomorphine (6.25-Mg Tablets)

Apomorphine acts directly on the chemoreceptor trigger zone to induce emesis. Apomorphine is generally the emetic of choice in dogs because of its rapid onset and the ability to

reverse its action. Apomorphine is given at a dose of 0.02 to 0.04 mg/kg intravenous (IV) or intramuscular (IM). It can also be administered by placing it directly behind the eyelid in the subconjunctival sac. Diluting the pill with sterile water minimizes ocular irritation. Apomorphine solutions are not stable and must be made fresh before each administration. Vomiting usually ensues within 4 to 6 minutes. When used conjunctivally, the eye should be flushed copiously once vomiting occurs. Apomorphine can be used in cats but at the lower end of the dosage; however, it appears that apomorphine is much less efficacious in inducing emesis in cats (10% effective by one report) as compared with dogs. Adverse side effects can be reversed with naloxone (0.01 to 0.04 mg/kg IV) in both dogs and cats. Apomorphine administered subcutaneously often has a delayed onset of action, and the duration of action may be prolonged.

Apomorphine can induce central nervous system (CNS) excitability in patients intoxicated with snail bait (metaldehyde), and its use in these patients is not recommended. Apomorphine may also be contraindicated when further CNS depression will significantly alter the patient's condition. Rare adverse side effects include CNS and respiratory depression, excessive vomiting, and occasionally CNS stimulation. "Morphine mania" is also an adverse event that may occur in cats.

Xylazine

Xylazine can be used as an emetic in cats with limited effectiveness (approximately 60% effective by one report) at a dosage of 0.44 to 1 mg/kg IM or subcutaneous (SC). Xylazine has centrally acting α_2-agonist activity. If effective, this drug usually induces vomiting within 10 minutes. A potential adverse effect of xylazine is respiratory depression. The activity of this drug can be reversed with yohimbine, an α_2-antagonist, at a dosage of 0.1 mg/kg IV in both dogs and cats.

Others

It is generally noticed by many veterinarians that inducing vomiting in cats can be a challenge. Some clinicians have suggested the use of cefazolin (300 mg fast IV) or a combination of hydromorphone and midazolam (0.05 mg/kg + 0.2 mg/kg, respectively) as emetics for cats. One drug that seems to be emerging as an effective emetic in cats is dexmedetomidine, an α_2-receptor–specific agent. Many different dosage regimes have been suggested for cats (e.g., 1-2 mcg/kg IV; 40 mcg/kg IM), and the sedative effects can be successfully reversed with atipamezole.

Gastric Lavage

The purpose of gastric lavage is to remove ingested toxins from the stomach by irrigation. The procedure may be indicated if emesis induction is ineffective or contraindicated. The patient must be unconscious or lightly anesthetized, placed in right lateral recumbency. A cuffed endotracheal tube must be in place. A large stomach tube is passed into the stomach no farther caudal than the xiphoid process. The patient is placed in an inclined position with the head down at approximately a 20-degree angle. If the patient is tilted at too great an angle, the weight of the fluid-filled stomach on the diaphragm can impair respiration.

Water or physiologic saline is then instilled by gravity flow at a volume of 10 mL/kg body weight. Physiologic saline is the lavage fluid of choice in smaller patients, which are more prone to fluid and electrolyte abnormalities. Care should be taken to prevent overdistention of the stomach. The degree of gastric distention can be felt by placing a hand on the abdomen. Lavage fluid should be warm to slow gastric emptying and prevent hypothermia of the patient. Manual agitation of the stomach is performed while the lavage fluid is aspirated, and the procedure is repeated until the recovered lavage fluid is clear. Often a bilge or stomach pump is employed to churn the stomach contents and aid in recovering the lavage fluid. Copious amounts of lavage fluid are usually required (often 15 to 20 lavage cycles), and the practitioner should be prepared for disposal of the same volume. The fluid initially recovered should be saved for toxicologic examination. An activated charcoal suspension can be instilled before the stomach tube is kinked and removed.

An increasing trend is to administer activated charcoal before performing lavage to halt further absorption of the toxin. Removal of the toxin-charcoal complex becomes the goal of gastric lavage. Additional activated charcoal is then instilled after the lavage procedure has been completed.

Several studies of lavage have been performed in animals, and none has demonstrated substantial drug recovery, particularly if the procedure was delayed for more than 60 minutes after ingestion of the toxin.[1-3] When lavage was performed within 15 to 20 minutes of toxin ingestion, the mean recoveries were 38% and 29%, respectively. If lavage was delayed until 60 minutes after ingestion of the marker, the mean recoveries were 13% and 8.6%. In the majority of poisoned patients that present to the veterinary hospital nearly 60 minutes have already passed since toxin ingestion, and by the time the mechanics of setting up a lavage procedure are performed and lavage is started, this period has clearly passed. Therefore, the practical clinical success of this decontamination procedure is questionable.

It seems prudent to employ this technique if the ingested material is chunky, and large fragments of material can be expected to be recovered. However, chunky material larger than the diameter of the stomach tube will obviously not be retrieved. Additionally, lavage may be more effective in combating ingestions of toxins that delay gastric emptying, such as salicylates, anticholinergics, and cyclic antidepressants. Concretions may not be recovered. Other examples of anticipated poor recovery are toxins similar to iron tablets, which may adhere to the gastric lining, or large amounts of chocolate, which may melt into a significant ball of material that is difficult to retrieve.

The major complications of gastric lavage are aspiration pneumonia, laryngospasm, hypoxia, hypercapnia, fluid and electrolyte imbalances, and mechanical injury to the throat, esophagus, and stomach. Gastric lavage is contraindicated in patients with an unprotected airway, with ingestions of substances that carry a high risk of aspiration (e.g., hydrocarbons) or that are corrosive, with ingestion of sharp objects, with an underlying pathologic condition that increases the risk of hemorrhage or gastric perforation, and in patients that are postsurgical or have medical conditions that may be compromised by the lavage procedure.

Another version of this technique is enterogastric lavage. This entails gastric lavage combined with retrograde high enema. This technique requires placing a gastric tube and endotracheal tube and then instilling an enema solution until it passes from the stomach tube.

Whole-Bowel Irrigation

In whole-bowel irrigation (WBI), the gastrointestinal tract is cleaned by the enteral administration of large volumes of osmotically balanced polyethylene glycol electrolyte solution (PEG-ES) until a clear liquid stool is achieved. The goal is to physically remove the intraluminal contents of the gastrointestinal tract. The concentration of electrolytes and osmolality in the PEG-ES are balanced so that no net gain or loss of electrolytes or changes in water balance occur.

WBI has a theoretical value in a limited number of toxic ingestions. This technique is indicated in patients with ingestion of toxins that are in a sustained-release or enteric-coated form. It may possibly be of value in cases in which iron pills have been ingested.

Studies in dogs have shown that the average total body clearance of paraquat was increased from 5.67 L/hr to 13.2 L/hr with WBI, which removed 68.9% of the ingested dose.[8,9] Human volunteer studies have reflected mixed results. Studies with ampicillin, delayed-release aspirin, and sustained-release lithium demonstrated toxin reductions of 67%, 73%, and 67%, respectively.[10-15] However, in a study in which coffee beans were used as the marker, no significant improvement was demonstrated in the rate of removal from the gastrointestinal tract with WBI.[16]

WBI requires a nasogastric tube, a type of nasogastric feeding bag, a large quantity of PEG-ES, and an easy-to-clean location in the hospital. The nasogastric tube is necessary because the patient will not willingly consume an adequate volume of PEG-ES at a sufficient rate. Force feeding an adequate volume of PEG-ES is difficult to manage over a 24-hour period. If the patient is in an upright position, the incidence of emesis is decreased because

gravity aids in moving the solution into the intestinal tract. The solution is usually administered at a rate of 500 mL/hour. If vomiting occurs, it is best controlled with metoclopramide because of its antiemetic and gastric emptying effects. If this does not work, the flow rate of PEG-ES should be reduced by 50% for 60 minutes and then returned to the original infusion rate. This procedure should be continued until the fecal effluent is clear. The technique may take several hours.

WBI is contraindicated in patients with an obstructed or perforated bowel; ileus; significant gastrointestinal hemorrhage; hemodynamic instability; uncontrollable vomiting; and an unprotected, compromised airway. PEG-ES interactions with activated charcoal are unclear. The charcoal does not significantly alter the osmolality of the solution. The binding capacity of the charcoal may be decreased if it is administered concurrently with the PEG-ES. Complications of WBI generally include nausea and vomiting (particularly in patients receiving emetics before the irrigation procedure). Human patients occasionally complain of abdominal cramps and bloating.

Ion Exchange Resins, Cathartics, and Activated Charcoal

Ion Exchange Resins

Ion exchange compounds are used to bind the toxin to a carrier, thereby trapping it in the gastrointestinal tract until it is passed in the stool. Cholestyramine and activated charcoal are the two most commonly used compounds. Cholestyramine (Questran) and to a lesser extent colestipol (Colestid) are anion exchange resins that bind with lipoproteins and bile acids, thereby preventing intestinal absorption of toxic compounds taken up through these systems. Additionally, cholestyramine can interrupt enterohepatic recirculation of substances excreted through the bile. Some examples of susceptible compounds include phenobarbital, tetracycline, penicillin G, anticoagulants, thyroxine, chlorothiazide, digitalis, β-blockers, some pesticides, any highly lipophilic compound, and heat-stable *Escherichia coli* enterotoxin.

Cholestyramine is packaged as a powder, containing either 444 mg or 800 mg of dried cholestyramine resin per gram of powder. An oral suspension is made by mixing the powder with 60 to 180 mL of water, milk, fruit juice, or other noncarbonated beverage. The recommended dose is 50 to 75 mg/kg PO. Patients should be well hydrated during therapy. Cholestyramine has a wide margin of safety. The potential side effects include nausea, hypoproteinemia, constipation, steatorrhea, and loss of fat-soluble vitamins.

Cathartics

Another decontamination approach is the use of cathartic solutions to speed the transit time of the toxicant through the gastrointestinal tract, thereby decreasing the time allowed for toxin absorption. Saline cathartics work by altering the physical-chemical forces within the intestinal lumen. Osmotic forces increase the fluid volume within the gastrointestinal tract, which stimulates motility and speeds expulsion of the gastrointestinal contents. Cathartics are especially useful in aiding the elimination of ingested solid toxicants and enteric-coated or timed-release compounds.

The two primary types of osmotic cathartics administered to poisoned patients are saline solutions and saccharide solutions. Saline cathartics include sodium sulfate, magnesium sulfate, and magnesium citrate. The most common saccharide cathartic is sorbitol. Mineral oil (paraffin oil) is generally not recommended for use as a cathartic in dogs and cats. Mineral oil has a higher risk of pulmonary aspiration than other cathartics and is no longer recommended for use in organic compound ingestions or organochlorine insecticide intoxications. Mineral oil has been advocated in the past as an adsorbent for lipid-soluble toxins. Mineral oil should not be used concurrently with dioctyl sodium sulfosuccinate because emulsification could occur that could cause hepatic accumulation of the indigestible oil.

Animal studies that measured the effectiveness of cathartics used alone showed no statistically significant difference in toxin clearance between controls and treatment

groups.[17-20] These results are remarkable because the doses of cathartic administered were greater than the recommended therapeutic levels and the intervals between toxin ingestion and cathartic administration were less than those expected in an actual toxicosis.

The most rapid and potent cathartic is sorbitol, which is administered as a 70% solution at a dosage of 1 to 2 mL/kg. If catharsis is to be attempted it should be instituted within 60 minutes after toxin ingestion. Multiple doses can be dangerous and can intensify side effects, such as vomiting, nausea, abdominal cramps, dehydration, and possible hypotension. Many veterinarians use sorbitol-containing activated charcoal products, which appear to be very safe with few side effects (but may be less effective at binding toxin).

Contraindications to cathartic administration include a lack of bowel sounds, ingestion of a corrosive substance, presence of diarrhea, recent abdominal trauma, or intestinal obstruction or perforation. Additionally, patients with hypotension, volume depletion, or electrolyte abnormalities should not receive a cathartic. Based on current data, there are no definitive indications for the use of cathartics alone in the management of the poisoned patient.

Activated Charcoal

Activated charcoal, the most commonly used adsorbent, is often called the *universal antidote*. The large surface area of the charcoal adsorbs the toxicant, thus reducing or preventing systemic absorption (Box 10-1). The quality of the various charcoal products depends on the size and surface area per gram of charcoal. On average, 1 g of activated charcoal has approximately 1000 square meters of surface area. Activated charcoal is produced by heating wood pulp to 900° C and, then washing it with inorganic acids and drying it. The resulting small charcoal particles are "activated" with steam or strong acids. The final adsorptive surface contains several carbon moieties that adsorb compounds with varying degrees of affinity. In vitro adsorption to activated charcoal in aqueous solutions is a nonspecific process that reaches equilibrium in less than 30 minutes. Once equilibrium is reached, some desorption can occur. For this reason, some individuals advocate using activated charcoal in conjunction with a cathartic, most commonly sorbitol, to speed gastrointestinal transit and thereby decrease the time available for desorption to occur.

Activated charcoal has an undisputed role in the treatment of acutely poisoned patients. The earlier it is administered after toxin ingestion, the more effective it is. Activated charcoal is particularly effective against large nonpolar compounds. Neutral molecules are more adsorptive than ionized toxins. Activated charcoal interferes with endoscopic visualization. In human volunteers an average of 62 hours elapsed after administration before stools were no longer black.[21]

The dosage of activated charcoal to be used varies with the individual product and other contributing factors, such as the amount of food present in the gastrointestinal tract. However, the dose-response curve indicates that higher doses are more effective. If the volume of toxicant is known, the general rule is to administer the activated charcoal in a dose of 10 times the dose of toxicant. Activated charcoal tablets and capsules are not as effective as activated charcoal slurries.[22,23]

For animals, the recommended dose of powdered activated charcoal and water is 2 to 5 g/kg of body weight (1 g activated charcoal in 5 mL water). If sorbitol is added as a cathartic, it is given at a dose of 3 mg/kg and mixed with the activated charcoal. If the intoxicant is subject to significant enterohepatic recirculation, repeat administration of activated charcoal every 4 to 6 hours for 2 to 3 days may be indicated. When multiple doses of activated charcoal are used, it is important that the patient be adequately hydrated to prevent constipation (kaolin-containing products can enhance the probability of causing constipation). Additionally, repeat dosing with sorbitol may cause significant hypotension or hypovolemic shock, particularly in small patients or those with underlying cardiovascular instability.

As mentioned, cathartic use in conjunction with activated charcoal has been advocated. Several cathartics have been used; however, sorbitol seems to have the fewest drawbacks and enhances the flavor of the suspension. Other cathartics used are sodium sulfate (Glauber's salts) and magnesium sulfate (Epsom salts). Magnesium sulfate is not recommended

Box 10-1 Partial List of Compounds That Bind to Activated Charcoal

Acetaminophen	Meprobamate
Amphetamines	Methyl salicylate
Antibiotics	Methylene blue
Anticoagulant rodenticides	Morphine
Anthelmintics	Muscarine
Aspirin	Narcotics
Atropine	Nicotine
Barbiturates	Nortriptyline
Camphor	Organic iodine
Cantharides	Organic metal compounds
Carbamates	Organochlorine insecticides
Carbamazepine	Organophosphorus insecticides
Chlordane	Phenobarbital
Chloroquine	Phenothiazine
Chlorpheniramine	Phenylbutazone
Cocaine	Phenylpropanolamine hydrochloride
Diazepam	Phenytoin
2,4-D (dichlorophenoxy acetic acid)	Quinacrine hydrochloride
Digitalis	Quinidine
Digitoxin	Quinine
Ethylene glycol	Salicylamide
Fungicides	Salicylates
Hexachlorophene	Strychnine
Ipecac	Sulfonamides
Isoniazid	Theophylline
Mefenamic acid	Tricyclic antidepressants

in patients exhibiting CNS depression. Mineral oil should not be used in conjunction with activated charcoal because it coats the activated charcoal, blocking its ability to adsorb gastrointestinal toxicants.

Activated charcoal is contraindicated in patients with a compromised airway (without intubation), with hydrocarbon ingestion (because of an increased risk of aspiration), or with a gastrointestinal tract that is not anatomically intact. Activated charcoal should not be used in patients that have ingested caustic substances nor in those that are vomiting, having seizures, or are comatose unless endotracheal intubation has been accomplished to protect the compromised airway. The major complication of activated charcoal administration is emesis. Those patients receiving concurrent sorbitol cathartics are at higher risk of vomiting. In dogs administration of a sorbitol-containing solution in concentrations greater than 30% sorbitol frequently induces emesis.[19]

Care should be taken when using activated charcoal as it is very osmotically active and the potential exists to induce a clinically significant hypernatremia that might be difficult to control (more commonly seen in small breed animals, less than 30 lb). If possible a baseline sodium level should be obtained, and if the toxicant is known its ingredients should be reviewed. Hypernatremia can occur in patients with preexisting hypernatremia (e.g., dehydrated) or if the toxicant has excess sodium chloride (e.g., sodium bicarbonate, modeling clay, play dough) or osmotically active substances (e.g., gummy candies, sugar-free candies, sorbitol, paintballs, glycerol, bulk artificial sweeteners).

Cathartic and Activated Charcoal Combinations

Administration of a cathartic and activated charcoal combination has been advocated with the rationale that the charcoal binds the toxicant and the cathartic speeds expulsion of the

charcoal-toxicant moiety from the gastrointestinal tract before significant desorption of the toxicant from the activated charcoal occurs. Several manufacturers of activated charcoal supply it in a 10 to 20% sorbitol slurry solution. This combination is administered at a dose of 10 mL/kg. The earlier this combination is administered, the more effective it is. Administration of activated charcoal with a sorbitol cathartic may be beneficial even as late as 6 hours after toxin ingestion. Studies have shown that the addition of sorbitol to activated charcoal does not affect the adsorption of acetaminophen and actually increases the adsorption of aminophylline.[24,25] However, salicylate elimination was slowed with administration of an activated charcoal–sorbitol combination compared with charcoal use alone.

When treating patients that have ingested sustained-release or enteric-coated toxins or toxins with delayed absorption or marked hepatic biliary recirculation, "pulse" dosing of the activated charcoal is advocated (every 6 hours for 2 to 3 days). There is a risk of dehydration if all doses of activated charcoal are mixed with a cathartic. It is recommended that subsequent doses of activated charcoal not be mixed with a cathartic solution unless the clinician is confident that the patient's fluid balance will not be compromised.

Forced Diuresis

Forced diuresis is induced by administering a fluid overload and a diuretic concurrently. The patient must not be anuric or significantly oliguric. The object is to increase urine flow rates to 3 to 5 mL/kg/hr to force renal clearance of the toxicant. The most common diuretics employed are furosemide (5 mg/kg every 6 to 8 hours) and mannitol (1 to 2 g/kg IV every 6 hours). Intoxications that are most likely to respond to forced diuresis are those with a high level of renal excretion of the primary toxicant (e.g., bromide, lithium, amphetamine, phenobarbital, and salicylate). Protein-bound toxins are not cleared efficiently by diuresis.

The primary problems associated with forced diuresis include potential fluid overload and electrolyte abnormalities. The practitioner should monitor for hyponatremia, hypokalemia, water intoxication, pulmonary edema, and cerebral edema. Additionally, some toxins such as tricyclic antidepressants and many sedative-hypnotics increase the patient's susceptibility to interstitial pulmonary edema. Forced diuresis is contraindicated in these intoxications.

Ion Trapping

Ion trapping alters the urine pH to inhibit reabsorption of toxins across the renal tubular membranes. The principle behind this approach is to "trap" the toxin in its ionized form in the urine where it can be excreted. Most nonionized molecules diffuse across membranes because of their high lipid solubility (i.e., nonionic diffusion). Ionized molecules usually cannot diffuse across cellular lipid membranes. The toxic compounds most successfully trapped are weak acids and bases.

Ion trapping can be successful only in the presence of the following conditions: the compound is excreted predominantly unchanged through the kidneys, the compound is a weak electrolyte with a suitable pKa, and the toxicant is primarily distributed to the extracellular space and is not protein bound. Ion trapping is contraindicated when the toxicant has a large volume of distribution, is strongly protein bound, is highly lipid soluble, and is cleared primarily by tissue or hepatic metabolism. Any attempt to alter urine pH requires monitoring of baseline urine pH, serum sodium and potassium levels, and blood pressure. Urine pH should be reevaluated hourly and sodium and potassium levels every 1 to 2 hours as indicated by the patient's condition and trends in serial laboratory values.

Acid diuresis is most commonly employed in patients intoxicated with compounds such as amphetamines and phencyclidine. Acidification is achieved by the administration of ammonium chloride at a dose of 100 mg/kg every 12 hours PO. A problem with urinary acidification is that many intoxicated patients already have a preexisting metabolic acidosis. Acidification may also affect the excretion rates of other drugs being used to treat the patient.

Alkaline diuresis is achieved with sodium bicarbonate administered at a dose of 1 to 2 mEq/kg of body weight added to intravenous fluids infused over 6 hours. Complications of alkaline diuresis include volume overload, hypernatremia, decreased serum ionized calcium, decreased oxygen delivery to tissue, paradoxical CNS acidosis, and hypokalemia. Contraindications for alkaline diuresis include any existing metabolic alkalosis (especially if the patient is concurrently receiving furosemide), hypokalemia, or hypocalcemia.

Gastrotomy

Gastrotomy and actual physical removal of toxins may be necessary if these toxins are refractory to other methods of decontamination. One example is ingestion of iron tablets that adhere to the gastric lining and have to be physically scraped off the gastric wall.

Lipid Emulsion

Intravenous fat emulsions usually composed of triglycerides and phospholipids can be used therapeutically for some toxicities with fat-soluble compounds (e.g., pyrethrins, permethrin, ivermectin, ionophores, marijuana). These fat emulsions theoretically form chylomicron-like droplets in the patient's serum, which may act as a "sink" for highly lipid-soluble xenobiotics. Compounds pulled into this "sink" are then unavailable for binding at their sites of action or to their target organs. The use of lipid emulsion therapy is new and its effectiveness against a growing list of lipid-soluble toxic compounds is being studied. Authors of individual chapters in this text highlight the effectiveness of this therapy against a variety of toxic xenobiotics.

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