

Toxicology

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According to the old saying, “curiosity killed the cat.” Usually, this is just a metaphor used to describe the ill effects of being nosy. But in many cases, it can actually be true. One of the instances when this phrase is true occurs when curious cats are exposed to poisons. Exposure may occur by way of the oral, cutaneous, or inhalation routes, although most feline toxicoses result from ingestion. Cats may chew on poisonous plants, ingest chemicals spilled on their fur, or swallow poisons in food or water. Sometimes cats are exposed to poisons through inappropriate administration by their owners. Many of these situations have the potential of being fatal without proper and prompt treatment.

Although toxicoses are not as common in cats as in dogs, they still accounted for 10% of the calls to a pet poison helpline.¹⁹ Most veterinarians report that pyrethrin-permethrin and plant intoxications are the most common toxicoses seen in cats.¹⁷ Cats are deficient in their ability to metabolize certain compounds, leading to poor detoxification and excretion of many chemicals and drugs. Cats are more sensitive to adverse drug reactions than most companion animals for several reasons. Cats are deficient in glucuronyl transferase activity, an enzyme that conjugates many chemicals. Moreover, the feline red blood cell is more susceptible to oxidative damage than that of other species, resulting in Heinz body formation and methemoglobinemia.

According to Paracelsus, the father of toxicology, everything is toxic—the dose makes the poison. That is especially true with cats. Although there are thousands of potential poisons for cats, the ones that are of special note include permethrin topical spot-ons, members of

the *Lilium* and *Hemerocallis* genera of plants, acetaminophen, and ethylene glycol (EG). This chapter focuses on these toxicants and several others that can be dangerous for cats. A list of online resources for toxicology information is provided in Box 31-1.

PESTICIDES

Cats may have exposure to pesticides either through accidental ingestion of inappropriately stored products or through malicious poisonings.

Snail and Slug Baits

Metaldehyde is a polymer of acetaldehyde and is often used as snail and slug bait. Commercial products are available in various forms, such as granules, pellets, and liquids, and are designed to be used in and around gardens (Figure 31-1). Toxicosis is more common in dogs, which are more likely to ingest bait in a garden or from an improperly stored container. The minimum lethal dose of metaldehyde in cats is not known; however, in dogs it is 100 mg/kg.⁴² Serious adverse effects occur at much lower doses. Although the mechanism of action of metaldehyde is not known, its effects are well established. Both metaldehyde and its metabolite, acetaldehyde, will distribute widely in the body and cross the blood-brain barrier.

After ingestion affected animals have signs of tachycardia, nervousness, sensitivity to light and noise, panting, drooling, ataxia, hyperthermia, tremors, and

BOX 31-1**Online Resources for Toxicology Information**

American Association for the Prevention of Cruelty to Animals, Animal Poison Control Center:

<http://www.aspca.org/apcc>

Pet Poison Helpline: <http://www.petpoisonhelpline.com>

American Board of Veterinary Toxicology:

<http://www.abvt.org>

National Pesticide Information Center:

<http://npic.orst.edu>

Environmental Protection Agency: <http://www.epa.gov>

American Association of Poison Control Centers:

<http://www.aapcc.org>



FIGURE 31-1 Pelleted slug and snail bait is often placed under plant foliage in gardens, either scattered loosely or in small traps. Nontoxic slug and snail bait containing ferric phosphate is readily available.

seizures.⁴² Onset of clinical signs in dogs generally occurs within 3 to 5 hours of ingestion but can occur in as soon as 30 minutes. Clinical signs can last for up to 5 days but will lessen over 12 to 72 hours with appropriate treatment.⁵¹ Metabolic acidosis often occurs with toxicity, and in some cases hepatic failure may occur within 2 or 3 days after exposure.⁴²

The diagnosis of metaldehyde toxicosis is based on history of exposure and associated clinical signs. If necessary, serum and urine can be assayed for acetaldehyde. Lesions on necropsy are generally nonspecific.

Treatment includes early decontamination, supportive care, and seizure control. Emesis can be induced in appropriate patients, or gastric lavage may be used under sedation or anesthesia. Activated charcoal may help inhibit metaldehyde absorption. Methocarbamol has been used successfully to control tremors and seizures from metaldehyde toxicosis in dogs (Table 31-1).⁴² Other options for seizure control include diazepam,

barbiturates, and inhalant anesthesia. Affected animals should be monitored for metabolic acidosis and hyperthermia. Intravenous fluid therapy is indicated to combat hyperthermia and dehydration. Metabolic acidosis may be treated with bicarbonate if necessary. A warm, quiet, comfortable environment helps lessen anxiety and nervousness. Treatment should continue until clinical signs are resolved, which may take several days.

Fly Bait

Methomyl is an extremely toxic carbamate insecticide that is found in certain fly baits. The mechanism of action of carbamates is through the inhibition of both acetylcholinesterases and pseudocholinesterases.³ Almost immediately after ingestion, seizures and pulmonary edema occur. The signs occur rapidly and are so severe that most cases are fatal.³ Atropine, a cholinergic agent, is antidotal for methomyl toxicity.³ In addition, seizure control is recommended. Because the clinical signs occur so quickly, decontamination is usually not an option.

RODENTICIDES

Rodenticides are designed to kill rats, mice, gophers, and other rodents. Cats may be exposed to rodenticides either through accidental ingestion of the bait or through eating poisoned rodents. Sometimes people mix rodenticides with foods such as tuna or peanut butter, inadvertently luring pets as well as rodents to the bait. The most commonly reported toxicoses are caused by anticoagulant rodenticides, bromethalin, cholecalciferol, strychnine, and zinc phosphide.³¹ In many cases diagnosis is based on a history of exposure and compatible clinical signs. In some cases laboratory testing is necessary to establish the diagnosis, especially when an accurate history is not available. Response to treatment may also be a valuable indicator. Although antidotes are not available for all rodenticides, decontamination and symptomatic and supportive treatments are important.

Emetics may be administered to appropriate patients (e.g., those without seizures, depression, or coma) if exposure occurred within the previous 1 to 2 hours. Activated charcoal is administered as an adsorbent and may be combined with a cathartic.

Anticoagulant Rodenticides

Anticoagulant rodenticides include the short-acting warfarin and long-acting chemicals such as pindone, diphacinone, difethialone, chlorophacinone, brodifacoum, and bromadiolone. They are readily available in many formats, including pellets and powders, from several sources, including feed stores and home and

TABLE 31-1 Selected Drugs Useful in the Treatment of Toxicoses in the Cat

Drug	Indication	Dose
Acetylcysteine	Acetaminophen toxicity	140 mg/kg PO for initial dose, then 70 mg/kg PO every 4 hours for 3-5 treatments
Activated charcoal	Adsorbent for ingested toxicants	2-5 g/kg PO; slurry made with 1 g per 5-10 mL water
Ascorbic acid	Acetaminophen toxicity	20-30 mg/kg, PO, every 6 hours
Atropine	OP, carbamate toxicity	0.2-0.5 mg/kg; $\frac{1}{4}$ given IV, remainder IM or SC; every 4-8 hours as needed
Cimetidine	Acetaminophen toxicity	5-10 mg/kg PO or IV, every 6 to 8 hours
Dapsone	Brown recluse spider envenomation	1 mg/kg PO, once daily for 14 days
Diazepam	Control of seizures	0.25-0.5 mg/kg IV or rectally; repeat as needed
Ethanol (20%): add 250 mL 100% ethanol to 1 L crystalloid fluids	Ethylene glycol toxicity	5 mL/kg, CRI over 1 hour; every 6 hours for 5 treatments, then every 8 hours for 4 treatments
Kaolin/pectin	Gastrointestinal protectant	1-2 mL/kg PO, every 6 hours
Methocarbamol	Control of tremors, muscle fasciculations	55-200 mg/kg IV or PO, every 8 hours; maximum 330 mg/kg/day
Misoprostol	Gastric protectant, NSAID toxicity	1-3 µg/kg PO, every 12 hours
Pamidronate	Cholecalciferol toxicity	1.3-2 mg/kg IV, diluted with saline and given over 2 hours
Phenobarbital	Control of seizures	2-6 mg/kg IV bolus, repeat up to 2 times at 20-minute intervals
Pralidoxime chloride (2-PAM)	OP toxicity (not for carbamate toxicity)	10-15 mg/kg IM or SC, every 8-12 hours
Sodium sulfate	Cathartic	250 mg/kg PO
Sorbitol (70% solution)	Cathartic	1-2 mL/kg PO
Sucralfate	Oral, esophageal, gastric, duodenal ulceration	0.25-0.5 g/cat PO, every 8 to 12 hours
Vitamin K ₁	Anticoagulant rodenticide toxicity	3-5 mg/kg PO or SC, every 8 to 12 hours with food
For Induction of Emesis*	Comments	Dose
Apomorphine	Dissolve 6 mg tablet in water or saline; flush conjunctival sac after emesis; antagonized with yohimbine (0.1 mg/kg, IV or 0.5 mg/kg SC or IM)	0.04 mg/kg IV or 0.25 mg/kg, conjunctival sac
Hydrogen peroxide (3%)	Take care to avoid aspiration	2 mL/kg PO; maximum 10 mL/cat
Xylazine	May cause respiratory depression, reversed with yohimbine (0.1 mg/kg IV or 0.5 mg/kg SC or IM)	0.44-1.1 mg/kg IM

PO, By mouth; OP, organophosphate; IV, intravenously; IM, intramuscularly; SC, subcutaneously; CRI, constant-rate infusion; NSAID, nonsteroidal antiinflammatory drug.

*Emesis should not be induced in patients that have ingested corrosive or caustic substances or substances that may cause aspiration pneumonia. In addition, induction of emesis is contraindicated in animals with decreased consciousness or those that have or are likely to have seizures.

garden stores. One of the first rodenticides marketed was warfarin, but resistance rapidly developed in target species, so newer generation compounds have been developed. Anticoagulants act by blocking the recycling of vitamin K₁ in the liver, which results in a

coagulopathy. Dysfunctional forms of clotting factors II, VII, IX, and X are released into circulation.³¹

Clinical signs of an anticoagulant toxicity include ecchymoses, petechiae, frank hemorrhage, pale mucous membranes, weakness, exercise intolerance, lameness,

dyspnea, coughing, and swollen joints. Early signs may be vague, such as lethargy and anorexia.²¹ The most common clinical presentation is acute onset of dyspnea caused by bleeding into the thoracic cavity.²⁴ Other presentations include otic bleeding, hematoma, melena, and hematochezia.²¹ Sudden death without preceding clinical signs is also possible. Clinical signs are seen several days after the bait is ingested because of the time needed to completely block the coagulation pathways. The duration of action, and thus the length of treatment required, is highly variable, ranging from 14 days to several weeks, depending on the chemical involved.²⁴

Commonly used coagulation tests include bleeding time, activated clotting time (ACT), prothrombin time (PT), and activated partial thromboplastin time (APTT). ACT and APTT measure the intrinsic clotting cascade. PT measures the extrinsic coagulation pathway. Anticoagulant rodenticides affect both the extrinsic and intrinsic pathways. When vitamin K is depleted, the first clotting factor to be affected is factor VII of the extrinsic coagulation pathway. In early cases of toxicoses (36 to 72 hours after ingestion), the PT will be prolonged, but the animal will still appear clinically normal because the other pathways are functioning.³⁰ However, after 72 hours factor IX becomes depleted and shuts down the intrinsic pathway, prolonging other coagulation tests, at which time hemorrhage is possible.

The PIVKA (protein induced in vitamin K antagonism) test (Thrombotest; Axis-Shield PoC, Oslo, Norway) is a newer diagnostic tool for anticoagulant rodenticide toxicosis. It evaluates the extrinsic and common pathways.²² However, if the PT is prolonged, the PIVKA test adds no further information. PIVKA times are also prolonged in any vitamin K₁-responsive coagulopathy.

Decontamination is helpful only with early recognition of ingestion. Vomiting may be induced if ingestion occurred within the previous 4 hours.²⁴ Activated charcoal may be useful if a significant amount of chemical has been ingested. Other treatment is aimed at providing functional clotting factors. PT and PIVKA are monitored at baseline and 48 and 72 hours later.²⁴ Because PT elevates before clinical signs occur, it is a useful indicator of when vitamin K₁ therapy is indicated. Testing for both PT and PIVKA must be performed before vitamin K₁ is administered to prevent false-negative results.

In some cases initial treatment may require transfusion of fresh frozen plasma or whole blood to supply clotting factors.²¹ Oral vitamin K₁ (phytonadione) therapy is antidotal for anticoagulant rodenticides. Injectable vitamin K₁ is not recommended because of the risk of anaphylactic reactions. Vitamin K₃ is contraindicated because it is not effective and may induce hemolytic anemia.³⁰ Treatment should continue as long as necessary, depending on the type of rodenticide (e.g., 14 days for warfarin, 21 days for bromadiolone, 30 days for other compounds). Confirming a normal PT 48 to 72

hours after the last dose of vitamin K₁ can ensure that it is not needed further. The prognosis is generally good if the toxicity is recognized and treated early.

Bromethalin

Bromethalin has been sold since the 1980s and is typically available in grain-based pellet form. Bromethalin is an uncoupler of oxidative phosphorylation and causes a reduction of adenosine-5'-triphosphate (ATP), decreasing nerve impulse conduction.¹² After ingestion absorption is rapid and peak plasma levels are reached in a few hours.³⁰ Clinical signs can occur at any time from 24 hours after ingestion to 2 weeks later and include muscle tremors, seizures, hyperexcitability, forelimb extensor rigidity, ataxia, depression, loss of vocalization, paresis, paralysis, and death.^{11,12,30} Low-dose exposure causes slow development of clinical signs, starting with hindlimb ataxia and paresis, with hindlimb paralysis following.¹¹ Affected animals also show decreased conscious proprioception, loss of deep pain, and upper motor neuron bladder paralysis.¹¹ The most common postmortem lesions include cerebral and spinal cord edema and a spongy appearance to the cerebellum.¹²

Diagnosis is based on history of exposure and compatible clinical signs. Because there is no antidote to this rodenticide and the clinical effects can be extremely severe, early aggressive decontamination is critical. If ingestion occurred recently (within 2 hours of presentation), emetics, activated charcoal, and a cathartic should be administered.¹¹ Activated charcoal may be required every 4 to 8 hours for at least 3 days. Cats with cerebral edema may be treated with mannitol (250 mg/kg intravenously, every 6 hours) and dexamethasone (2 mg/kg intravenously, every 6 hours).¹¹ Seizures may be managed with diazepam or phenobarbital. Unfortunately, treatment of such severely affected animals is often futile. Mildly affected animals may recover in 1 to 2 weeks. More severely affected animals may require prolonged nutritional support and nursing care.

Cholecalciferol

Cholecalciferol (vitamin D₃) is metabolized in the liver to calcifediol (25-hydroxycholecalciferol). Calcifediol is then metabolized by the kidney to calcitriol (1,25-dihydroxycholecalciferol). Cholecalciferol increases intestinal absorption of calcium, stimulates bone resorption, and enhances renal tubular reabsorption of calcium. Toxic ingestion results in hypercalcemia, which can lead to renal failure, cardiovascular abnormalities, and tissue mineralization. Plasma phosphorus and calcium increase within 72 hours of ingestion. The (calcium × phosphorus) product may exceed 130 mg²/dL² (10.5 mmol²/L²), well above the level at which soft tissue mineralization occurs.²⁹ Other laboratory abnormalities include

increased blood urea nitrogen (BUN) and creatinine, hyperkalemia, acidosis, and decreased urine specific gravity.³⁰

Clinical signs usually occur 18 to 36 hours after ingestion and include vomiting, diarrhea, inappetence, depression, polyuria, polydipsia, and cardiac arrhythmia.^{29,30} With high doses renal failure results from the deposition of calcium in the kidney and occurs in 24 to 48 hours.²⁹ Death is often due to acute renal failure, and animals that survive may have permanent loss of renal function and other abnormalities. Cholecalciferol is highly lipid soluble and is eliminated slowly from the body.²⁹ Clinical signs, and therefore duration of treatment, may last for weeks.

Diagnosis is based on a history of exposure and compatible clinical signs. Other causes of hypercalcemia must be ruled out. Assessment of serum parathyroid hormone, parathyroid hormone-related polypeptide, and 25-hydroxycholecalciferol levels may be helpful in the differential diagnosis.

Decontamination is recommended with early exposures. Emesis may be induced in appropriate patients. Activated charcoal should be administered concurrently with a cathartic. Baseline and serial monitoring of serum BUN, creatinine, phosphorus, and calcium is necessary. In cats that develop clinical signs or changes in laboratory parameters, diuresis with intravenous 0.9% saline is indicated. Furosemide is added to the therapy once the cat is hydrated to increase renal calcium excretion.²⁹ Oral prednisone may be used to decrease serum calcium by decreasing bone resorption, decreasing intestinal absorption, and increasing renal excretion.

Severely affected cats or those that relapse after initial therapy may require treatment with a bisphosphonate. Pamidronate (see Table 31-1) inhibits osteoclastic bone resorption and has been used successfully to treat exposures in combination with fluid therapy and supportive care.²⁹ Once calcium levels are normal, they should be monitored daily for 4 days. Retreatment may be required.

Strychnine

Strychnine is an alkaloid derived from the nux vomica tree that is used to kill rodents and also other pests, including coyotes.³⁰ Strychnine is considered a restricted pesticide in many states. It is often, but not always, found as red-colored grain-based pellets. Cats are less commonly affected than dogs but may be poisoned by accidental or malicious exposure. Strychnine is a glycine antagonist in the central nervous system (CNS) and results in excessive neuronal activity, causing muscle spasms and severe convulsions.⁵² The lethal dose in cats is 2 mg/kg.⁵² Early signs of toxicity (within minutes) include apprehension and stiffness, progressing to tonic extensor rigidity, especially in response to stimulation (light, sound, touch).³⁰ Clinical signs affect the face, neck,

and limb muscles first.⁵² Convulsions with opisthotonus can appear quickly. Death may occur as a result of hypoxia from impaired respiration as soon as 10 minutes after ingestion or up to 24 to 48 hours later.⁵² The differential diagnosis includes a wide variety of possibilities, including rabies and other intoxications.

Diagnosis is based on a history of exposure, compatible clinical signs, and strychnine testing (on urine, tissues, or stomach contents). If exposure is recent and the patient's status is stable, activated charcoal may be administered to reduce further absorption. Emetics should be used with care insofar as they may precipitate violent muscle or convulsive activity.⁵² It may be safer to perform gastric lavage on a sedated or anesthetized patient. Seizure control in most cases is difficult but may include the use of methocarbamol, propofol, or barbiturates. Diazepam is usually not recommended because its efficacy for strychnine-induced seizures is variable.³⁰

Respiration should be monitored closely and mechanical ventilation initiated if severe respiratory depression occurs. Supportive care includes intravenous fluid therapy and provision of a quiet, dark environment. Most poisoned animals require hospitalization for 24 to 72 hours. Patients presented late in the progression of the disease are at higher risk of death.

Zinc Phosphide

Zinc phosphide is commonly found in mole and gopher baits at concentrations up to 5% and is highly toxic.¹ It is used for vermin control in areas where rodents have become resistant to other chemical control methods and is usually a restricted-use pesticide. Dogs and cats are the species most likely to suffer accidental ingestion and toxicosis. Cats that eat very recently poisoned rodents may also be at risk of toxicity from zinc phosphide still in the gastrointestinal tract of the target animal.¹ For most species a lethal dose is 20 to 40 mg/kg.¹

After ingestion, phosphide is converted to phosphine gas by stomach acid.¹ Released phosphine gas causes severe irritation to the pulmonary tissues, which results in respiratory distress and death occurring secondary to respiratory failure. Clinical signs are typically seen in 15 minutes to 4 hours, depending on when the animal last ingested a meal.¹ Early signs of toxicosis include anorexia and depression, followed by rapid and deep respirations.¹ Vomiting is common and often contains blood.

Treatment includes early decontamination (induction of emesis or gastric lavage, activated charcoal with a cathartic) and supportive care for the associated clinical effects (e.g., acidosis, respiratory compromise, depression). Some animals suffer liver failure.¹ There is no specific antidote for zinc phosphide. Because the conversion of zinc phosphide to phosphine gas is enhanced with gastric acidity, treatment with antacids is highly recommended.

The phosphine gas emitted from the affected animal is a human health hazard and can be dangerous to hospital personnel even at levels that cannot be detected by smell. Therefore precautions such as adequate ventilation should be taken to protect staff members.¹

INSECTICIDES

Insecticides are primarily used in and around cats for flea control. They can be used as sprays, powders, flea collars, dips, and spot-on treatments. When used according to the label directions, most insecticides can be used safely around cats. In the United States these products are regulated as pesticides by the Environmental Protection Agency (EPA). Most incidents reported to the EPA are minor, but major incidents, including death, have occurred. Serious adverse effects are most likely to occur when products labeled for dogs—often bearing a name similar to that of a product labeled for cats—are inappropriately or mistakenly applied to cats, especially products containing permethrin. Problems may also occur when products are not applied according to label directions, or are applied to ill cats. In addition, cats have been affected by exposure to treated dogs. It may be prudent to keep cats away from dogs immediately after treatment with spot-on products. Adverse effects should be reported to the product manufacturer. In the United States veterinarians should also report incidents to the National Pesticide Information Center and the EPA (see Box 31-1).

Pyrethrins and Pyrethroids

Pyrethrins are naturally derived from chrysanthemum flowers, whereas pyrethroids are synthetic analogs. These compounds modify the sodium channels in nervous tissue and muscle cell membranes, causing repetitive discharging of the cell and clinical signs of neurotoxicity. Most insecticidal products labeled for use in cats contain low levels of pyrethrin and, if used appropriately, are relatively safe for cats.³⁷

Permethrin is derived from a combination of esters that are extracted from dried chrysanthemum flowers and is further classified as a type I pyrethroid.³⁷ This insecticide is used in spot-on flea treatments for dogs but is contraindicated in cats because of the high risk of toxicity. Cautionary labeling on canine products may not be visible enough or adequate to prevent inappropriate use, and risk awareness among pet owners may be low. Cats are highly sensitive to the effects of permethrin, probably because of their deficiency of hepatic glucuronyltransferase.⁶ Permethrin toxicity is one of the most commonly reported feline toxicities.^{28,50}

Cats are most commonly exposed to concentrated permethrin compounds inappropriately or accidentally

through exposure to topical flea products intended for canine use only. These spot-on products can contain 45% to 65% permethrin or more. The most commonly seen clinical signs include tremors, muscle fasciculations, ear twitching, facial twitching, hyperesthesia, ataxia, ptalism, pyrexia, mydriasis, and seizures.⁶ Clinical signs may occur with exposure to only a few drops of the concentrated solution and may occur within a few hours or several days. In general, clinical signs will continue for 24 to 72 hours but may last up to 7 days.^{37,50} Death occurs in about 10% of cases.⁵⁰

Diagnosis of permethrin toxicity is based on a history of recent exposure and typical clinical signs. Differential diagnoses include other causes of seizures and tremors. Treatment should focus on seizure control, decontamination, and supportive care (Box 31-2). With recent exposures to permethrin, the cat should be bathed completely using lukewarm water and mild hand dishwashing detergent or shampoo to remove any residual product. Hot water should not be used because it may increase dermal perfusion and uptake of permethrin. Seizures and tremors typically respond to methocarbamol (see Table 31-1).³⁷ Other options for seizure control include propofol, barbiturates, diazepam, and inhalant anesthetics. In addition, supportive care, including maintaining normal body temperature, supplying intravenous fluids, and providing nutritional support, is needed.

Cholinesterase Inhibitors

Cholinesterase inhibitors include carbamates and organophosphates. These compounds have been widely used in agricultural and veterinary medicine for decades. Cats may be exposed accidentally or by inappropriate use of products. Organophosphates are very toxic to cats and not recommended in this species. Carbamates (e.g., carbaryl) are less toxic and are found in several insecticides marketed for use in cats in various formulations (e.g., shampoos, powders, collars). These compounds bind to and inhibit cholinesterases, causing excess accumulation of acetylcholine and resulting in cholinergic excitation and muscarinic and nicotinic signs.²⁷ Organophosphates have higher binding affinity than carbamates and are often called *irreversible inhibitors*.

Clinical signs result from overstimulation of the cholinergic nervous system, as well as skeletal muscle and the CNS, and appear within minutes to hours after exposure.⁵³ Clinical signs of toxicity include classic muscarinic signs often referred to as *SLUDGE signs*: salivation, lacrimation, urination, defecation, gastrointestinal upset, and emesis.²⁷ Nicotinic signs include ataxia, weakness, tremors, and muscle fasciculations. Cholinesterase inhibitors can also cause seizures, increased bronchial secretions, pulmonary edema, and bradycardia.²⁷

Diagnosis is based on history of exposure and compatible clinical signs. To confirm exposure to a

BOX 31-2**Recommendations for Treatment of Permethrin Toxicosis* in the Cat****Veterinary Treatment Plan:****1. Seizure control**

- Diazepam: 0.25-0.5 mg/kg IV (can also be given rectally), repeat as needed every 3-5 min
- Midazolam: 0.3 mg/kg IV/IM, repeat as needed every 3-5 min

If ongoing seizures after benzodiazepine bolus $\times 2$, then consider:

- Propofol: 4-6 mg/kg slow IV as a bolus, then 0.05-0.3 mg/kg per min IV as a CRI
- Alfaxalone CD: 2-3 mg/kg slow IV bolus
- Phenobarbitone: 2-4 mg/kg slow IV, diluted 1:10 with 0.9% NaCl. Repeat as needed every 2 h, total dose should not exceed 20 mg/kg/day

2. Muscle fasciculation control. Note that the aim is not to completely anaesthetize the patient but to decrease the severity of clinical signs.

- Methocarbamol (if available): 55-200 mg/kg IV/PO every 8 hours, up to a maximum dose of 330 mg/kg per day
- Midazolam: 0.2 mg/kg/hour IV as a CRI
- Propofol: 0.05-0.3 mg/kg per min IV as a CRI

3. Ensure patent airway. Swab/suction pharynx if patient is hypersalivating. Provide oxygen support if needed (maintain SpO₂ >95%).**4. Skin decontamination.** Warm bath with mild detergent, towel, warm blow dry.**5. Temperature monitoring and control.** Maintain body temperature 38°-39° C.**6. IV crystalloids.** Aim for 1.5 \times maintenance rates. Monitor packed cell volume/total plasma protein, electrolytes every 12 hours; check urine specific gravity when available.**7. Ocular lubrication.** Every 4 h (e.g., Lacrilube/Opticin)**8. Bladder expression or urethral catheterization:** Every 6-8 h (lower motor neuron [LMN] bladder).**9. Quiet, darkened environment.****10. Maintain sterna recumbency,** head slightly elevated, turn hind legs every 6 h.**11. Prevent self-grooming.** Apply Elizabethan collar once mobility begins to improve.

IV, Intravenously; *IM*, intramuscularly; *CRI*, constant-rate infusion; *PO*, by mouth.

*Source: Protocols, Animal Referral Hospital, Sydney, NSW, Australia. From: Boland LA, Angles JM: Feline permethrin toxicity: retrospective study of 42 cases, *J Feline Med Surg* 12:61, 2010.

be detected in stomach contents and tissues. Changes on complete blood cell count, serum chemistry panel, and urinalysis are generally nonspecific.⁵³

Patients affected by organophosphate or carbamate toxicity may deteriorate quickly so treatment must be initiated as soon as possible. Respiratory failure is the main cause of death, so artificial respiration may be required. Treatment also includes control of seizures. The specific antidote for organophosphate toxicity is pralidoxime chloride (2-PAM; Protopam, Ayerst Laboratories), which releases cholinesterase from the organophosphate. 2-PAM will control nicotinic signs and is most effective when given as soon as possible after exposure (preferably within 24 to 48 hours). Clinical improvement should occur within 3 to 4 days, and treatment is continued as long as needed. This drug is usually not recommended for carbamate toxicity.⁵³

Although atropine is often considered an antidote to cholinesterase inhibitors insofar as it blocks the effects of the excess acetylcholine at the neuromuscular junction, it should be used with caution. If muscarinic signs are present, a test dose (0.02 mg/kg intravenously) can be given to determine if the signs are due to organophosphate or carbamate toxicity versus other causes. If the heart rate increases and the pupils dilate in response to the test dose, the clinical signs are probably not due to organophosphate or carbamate toxicity. This is because the atropine dose required to resolve clinical signs caused by insecticide toxicity is about 10 times the pre-anesthetic dose of the drug. If insecticide toxicity is confirmed, atropine can be administered to control muscarinic signs (see Table 31-1). The dose is adjusted by monitoring response, especially heart rate and secretion production.

Activated charcoal may be used to bind insecticide in the gastrointestinal tract, and bathing with soap and water can be used for cats with dermal exposure to prevent further absorption. Additional treatments may include methocarbamol, diazepam, or phenobarbital to control seizures and muscle tremors.⁵³ Good supportive and nursing care, including intravenous fluid therapy and nutritional management, is essential.

Other Topical Spot-On Products

There is limited published information regarding adverse effects of most topical spot-on products approved for use in cats, although they appear to be generally safe.³⁹ Most products are applied to the area between the shoulder blades and are intended for use every 30 days. Oral exposure to topical products may result in excessive drooling and gastrointestinal upset owing to the bitter taste. Hypersensitivity skin reactions may occur with any topical product, and signs are expected to resolve with bathing and supportive care (e.g., topical corticosteroids, antihistamines).

cholinesterase inhibitor, whole blood, serum, or plasma cholinesterase enzyme activity (ACHE test) can be evaluated through an accredited veterinary laboratory. The diagnosis is confirmed if the cholinesterase activity is less than 25% of normal. In addition, the insecticide can

Imidacloprid

Imidacloprid is a chloronicotinyl nitroguanide insecticidal agent that is used as a spot-on product labeled to kill fleas (but not ticks) in dogs and cats. It is marketed as Advantage Topical Solution (Bayer HealthCare, Shawnee Mission, Kansas). In combination with moxidectin, it is marketed as Advantage Multi and Advocate (Bayer HealthCare). When applied topically, it spreads rapidly over the cat's skin by translocation but is not absorbed systemically. Imidacloprid acts by blocking the nicotinic pathways, which results in a buildup of acetylcholine at the neuromuscular junction, causing impairment of normal nerve function and death of the insect.²⁰ Imidacloprid is also found in combination with permethrin in dog-only products (K9 Advantix, Bayer HealthCare), which can be dangerous if used accidentally or inappropriately on a cat because of the permethrin.

Imidacloprid products formulated for use on cats have a low order of toxicity. The manufacturer recommends caution when used in debilitated, elderly, pregnant, or nursing animals and kittens younger than 4 months of age. There is one report of a cat with a thymoma that developed dermatosis (erythema multiforme) and systemic signs shortly after being treated with imidacloprid.¹⁴ A combination of paraneoplastic syndrome and drug reaction may have caused the cat's clinical signs.

Oral exposure to imidacloprid through grooming may cause mild and self-limiting drooling or retching as a result of the bitter taste of the product.^{20,39} Imidacloprid poisoning would be expected to produce nicotinic signs.³⁹ Dermal hypersensitivity reactions from topical application should be treated by bathing with a non-insecticidal shampoo and symptomatic care (e.g., antihistamines, hydrocortisone). Ocular exposure is treated with lavage.

Fipronil

Fipronil is a phenylpyrazole antiparasitic agent introduced in the United States in 1996 as a flea and tick control product. It is marketed for veterinary use as Frontline Top Spot and Frontline Spray (Merial; Duluth, Georgia). It may be found in combination with methoprene, an insect growth regulator (Frontline Plus, Merial). Fipronil is classified as a gamma-aminobutyric acid (GABA) agonist, and it causes its effects on insects by disrupting CNS activity.²⁰ After topical application the product spreads over the skin by translocation and collects in skin oils and hair follicles.

Fipronil products for veterinary use have a low order of toxicity by dermal, oral, and inhalation exposure.²⁰ Oral exposure may cause mild and self-limiting drooling and vomiting. A dermal hypersensitivity reaction may occur within hours of application of topically applied products.⁵⁸ Affected cats should be bathed in a

non-insecticidal shampoo and treated symptomatically (e.g., antihistamines, hydrocortisone). Ocular exposure causes mild reactions that are treated with lavage.

Selamectin

Selamectin is a semisynthetic avermectin developed specifically for broad-spectrum use in dogs and cats and was introduced in the United States in 2000. It is marketed as the spot-on product Revolution (Pfizer Animal Health). Selamectin causes parasite death through neuromuscular paralysis by increasing permeability in neuronal chloride channels.²⁰ Mammals have less sensitive chloride channels and so are less affected.

Topical selamectin is labeled for use in cats against fleas (*Ctenocephalides felis*), ear mites (*Otodectes cynotis*), hookworm (*Ancylostoma tubaeforme*), and roundworm (*Toxocara cati*). It is also approved as a preventive for heartworm disease. Although it is applied topically, it is absorbed systemically, with peak plasma levels occurring approximately 15 hours after application in cats.³⁴ Selamectin is distributed selectively to the sebaceous glands, where it acts against external parasites. According to studies, adverse effects of selamectin are rare.²⁰ The most common adverse effect is minor skin irritation (redness, irritation) or transient alopecia at the site of application.⁵⁸ Other possible adverse effects include diarrhea, vomiting, muscle tremors, anorexia, pruritus/urticaria, erythema, lethargy, salivation, and tachypnea. Accidental oral ingestion through grooming causes self-limiting salivation and intermittent vomiting in cats. There is no specific antidote, and treatment is symptomatic and supportive.

POISONOUS PLANTS

Lilies

Several species of the *Lilium* genus, including Easter lily (*Lilium longiflorum*), tiger lily (*Lilium lancifolium*, formerly *tigrinum*), Asian lily (*Lilium asiatica*), Stargazer lily (*Lilium auratum*), and others have been shown to cause acute kidney failure in cats, characterized by acute tubular necrosis.^{40,48,54} Also, some species of daylily (*Hemerocallis* genus) are potentially dangerous for cats.¹⁵ These flowering plants are common in gardens, as potted houseplants, and as cut flowers (Figures 31-2, 31-3, and 31-4).

Exposure to any part of the plant, including the pollen, can lead to toxicosis. Even consuming less than one leaf can lead to severe consequences.²³ Cats appear to be the only species in which this poisoning occurs, and the mechanism of action and toxic ingredient is not known, although it appears to be water soluble and most concentrated in the flowers.²³ The case fatality rate is often high, and surviving cats may suffer permanent renal damage.⁴⁸



FIGURE 31-2 **A**, Easter lilies (*Lilium longiflorum*) are popular houseplants in the spring. **B**, All parts of the plant are highly toxic. (Courtesy Dr. Vicki Thayer.)

Clinical signs typically develop within 12 hours of ingestion (but may be delayed for up to 5 days) and include vomiting, anorexia, depression, polyuria, and polydipsia (Table 31-2).¹⁶ Less frequent signs include disorientation, ataxia, face and paw edema, head pressing, and seizures.⁵ The initial vomiting often subsides in 4 to 6 hours, leading to a false impression that the cat is recovering from an innocuous problem.¹⁶ Renal failure develops within 24 to 96 hours of ingestion.⁵⁴ Laboratory abnormalities include azotemia (with creatinine disproportionately increased compared with BUN), glycosuria,



FIGURE 31-3 Stargazer lilies (*Lilium auratum*) are also highly toxic to cats and are found as garden plants and as cut flowers. (Courtesy Dr. Edward Javinsky.)

TABLE 31-2 Onset and Duration of Common Clinical Signs and Changes with Lily Toxicosis in Cats

Clinical Sign/ Parameter	Onset	Duration from Onset
Vomiting	0-3 h	4-6 h
Salivation	0-3 h	4-6 h
Anorexia	0-3 h	Throughout syndrome
Depression	0-3 h	Throughout syndrome
Proteinuria	12-24 h	Until anuria develops
Urinary casts	12-24 h	Until anuria develops
Glucosuria	12-24 h	Until anuria develops
Isosthenuria	12-24 h	Until anuria develops
Polyuria	12-30 h	12-24 h
Dehydration	18-30 h	Until corrected
Serum chemistry changes	>24 h	Until corrected
Vomiting reoccurs	30-72 h	Through remainder of syndrome
Anuria	24-48 h	Through remainder of syndrome
Weakness	36-72 h	Through remainder of syndrome
Recumbency	48-72 h	Through remainder of syndrome
Death	3-7 days	

From Hall J: Lily. In Plumlee KH, editor: *Clinical veterinary toxicology*, St Louis, 2004, Mosby, p 434.



A



B

FIGURE 31-4 A and B, Day lilies (*Hemerocallis* spp.) are common garden plants available in thousands of varieties that are toxic to cats. (A courtesy Tori-Rose Javinsky.)

proteinuria, isosthenuria, and tubular epithelial urinary casts.^{16,23,54} No crystalluria occurs with lily toxicity.¹⁶ Increases in liver enzymes may occur late in the course of illness.¹⁶ The differential diagnosis includes EG toxicity, nonsteroidal antiinflammatory drug (NSAID) toxicity, and chronic renal disease.

With recent exposures decontamination followed by fluid diuresis at twice the maintenance rate with lactated Ringer's solution for a minimum of 48 hours is recommended.⁴⁰ If ingestion occurred less than 2 hours before presentation and no clinical signs have appeared, emesis can be induced (even if the cat has already vomited), followed by activated charcoal with a cathartic.²³ Dehydration is an important component in the development of renal damage, so initiation of fluid therapy is necessary to prevent permanent damage. Ideally, a central venous catheter and closed urine collection system should be placed. Referral to a 24-hour treatment facility



FIGURE 31-5 Azalea is an ornamental shrub that contains highly toxic grayanotoxins. (Courtesy Dr. Vicki Thayer.)

may be necessary. Baseline laboratory data (particularly serum chemistries and urinalysis) should be obtained. Renal function should be monitored for 2 to 3 days or more. Delayed treatment, even by 18 hours, can result in irreversible renal failure, leading to death or euthanasia.⁵⁴ Cats that receive prompt and aggressive treatment have a good prognosis. Cats that become oliguric or anuric have a poor prognosis but may respond to peritoneal dialysis. Cats that do not receive treatment will die in 3 to 7 days.⁵⁴

Rhododendron Species

Rhododendron species (azalea, rhododendron, rosebay) contain grayanotoxin glycosides, which affect sodium channels in cell membranes, leading to neurologic, gastrointestinal, and cardiovascular dysfunction (Figures 31-5 and 31-6).¹⁸ Grayanotoxins are found in all parts of the plant, including the flowers and nectar, and as few as two leaves may cause serious poisonings. Clinical signs include vomiting, diarrhea, abdominal pain, weakness, depression, cardiac arrhythmias, hypotension, shock, cardiopulmonary arrest, pulmonary edema, dyspnea, lethargy, seizures, and death.¹⁸

Decontamination and cardiovascular support are recommended after exposure. There is no specific antidote. Early induction of emesis followed by activated charcoal with a cathartic is important. Electrocardiography should be used to monitor heart rate and rhythm, and blood pressure should also be monitored. Cardiovascular support may require aggressive intravenous fluid therapy and sodium channel-blocking drugs (e.g., quinidine, procainamide, atropine) may be useful for some patients.¹⁸

Plants Containing Cardiac Glycosides

Hundreds of cardiac glycosides have been identified in plants; the most commonly known is digitalis, which has



FIGURE 31-6 Rhododendrons are popular ornamental shrubs. Grayanotoxins are found in every part of the plant. (Courtesy Dr. Vicki Thayer.)

been used medicinally in human and veterinary medicine for many years. All parts of cardiac glycoside-containing plants are toxic, and even small amounts can cause significant clinical signs, including death. Examples of cardiac glycoside-containing plants include oleander (*Nerium oleander*), lily-of-the-valley (*Convallaria majalis*), foxglove (*Digitalis purpurea*), certain milkweeds (*Asclepias* spp.), and squill (*Urginea maritima*).¹⁸

Cardiac glycosides inhibit the sodium-potassium ATPase pump and slow cardiac electrical conductivity.¹⁸ Onset of clinical signs varies with the species of plant ingested but may occur within hours of ingestion and last for several days. Clinical signs are related to the cardiovascular system and the gastrointestinal tract and include vomiting, abdominal pain, bradycardia, ventricular arrhythmias, and even sudden death.¹⁸ Diagnosis is based on identification of the plant ingested.

Treatment includes early induction of emesis, followed by activated charcoal with a cathartic.^{13,18} Monitoring with electrocardiography is recommended for at least 24 hours. Further treatment varies with the specific cardiac abnormalities detected. Prognosis is poor without early and aggressive intervention.¹⁸

Castor Bean Plant

The castor bean plant (*Ricinus communis*) is used as a decorative plant, and oil extracted from the seeds is used in industry and medicine. The toxic principle is ricin,² which is one of the most potent toxins known, and it is often linked to bioterrorism. All parts of the castor bean plant are toxic, but the seeds contain the highest concentration of ricin and are most commonly associated with poisoning.² Damage to the seed coat (usually by chewing) is required to allow the ricin to be available for absorption. Castor bean oil should not contain ricin if it is

properly manufactured.² Another decorative plant containing a similar phytotoxin (abrin) is the rosary pea or jequirity bean (*Abutilon precatorius*).¹⁸ It is used in Latin America to make jewelry for tourists and is also grown in Florida and Hawaii.

Clinical signs appear within hours of ingestion and may last for up to 5 days. Ricin is a cellular toxin, and its major effects are on the intestinal mucosa.² Initial clinical signs include vomiting, diarrhea, and abdominal pain, progressing to hemorrhagic gastroenteritis, seizures, and cerebral edema.¹⁸ There is no specific antidote, and treatment consists of decontamination and supportive care. Prognosis is poor once clinical signs appear.¹⁸

Cycad Palms (Cycas, Zamia Species)

Cycad palms and similar ornamental plants are generally found in tropical to subtropical climates but may also be grown as houseplants in more temperate climates. Cycasin is considered to be the toxic principle that is responsible for the hepatotoxicity and gastrointestinal signs generally seen with toxicosis.^{18,59} Most parts of the plant are toxic, but the seeds contain a higher concentration of cycasin. Clinical signs include vomiting and diarrhea, lethargy, depression, liver failure, coagulopathy, and death. Neurologic signs, such as weakness, ataxia, seizures, and coma, may also be seen.¹⁸ There is no specific antidote. Treatment includes early decontamination and supportive care.

Plants Containing Insoluble Calcium Oxalates

Many plants, including peace lily (*Spathiphyllum* spp.), calla lily (*Zantedeschia* spp.) (Figure 31-7), *Philodendron* spp. (Figure 31-8), and *Dieffenbachia* spp. (Figure 31-9) contain insoluble calcium oxalate crystals.²⁶ These crystals can cause mechanical irritation of the oral cavity. The clinical signs seen with ingestion of these plants include oral pain, difficulty swallowing, hypersalivation, swelling of the oral cavity, vomiting, depression, and inappetence.²⁶ Clinical signs are temporary and rarely severe and usually respond to supportive care, such as rinsing the mouth with water and offering a small quantity of milk or yogurt. Oral swelling can be treated with an antihistamine, and a protectant such as kaolin/pectin can reduce gastrointestinal irritation.²⁶

HOUSEHOLD HAZARDS

Ant and Roach Baits

Ant and roach baits are common objects found in households. They are also referred to as hotels, traps, or stations. The insecticides used most commonly in these



FIGURE 31-7 The calla lily (*Zantedeschia* spp.) is not a true lily but is toxic to cats on account of the presence of calcium oxalates found in all parts of the plant.



FIGURE 31-8 Philodendrons are tropicals that are common ornamental houseplants. The leaves contain insoluble calcium oxalates, which can cause oral irritation.

baits are present only in small amounts and include chlorpyrifos, sulfluramid, fipronil, avermectin, boric acid, and hydramethylnon. The baits usually contain inert ingredients such as peanut butter, breadcrumbs, sugars, and fats, which could be attractive to pets. Exposures to these types of baits usually do not require decontamination or treatment. Most often, if signs are seen at all, they are mild in nature and self-limiting and



FIGURE 31-9 Dieffenbachia (dumb cane) belongs to the tropical *Araceae* family and also contains insoluble calcium oxalates.

are usually attributed to the inert ingredients instead of the active ingredient. However, cats that ingest baits containing chlorpyrifos, an organophosphate, may require decontamination and treatment (as discussed previously).

Silica Gel Packets

Silica gel is used as a desiccant and often comes in paper packets or plastic cylinders. These products are used to absorb moisture with leather, medication, and some food packaging. Silica is considered to be chemically inert if ingested. However, if large amounts are ingested, it is possible to see signs of gastrointestinal upset, such as nausea, vomiting, and inappetence. Additional problems could occur if the silica gel was used as a desiccant in medication packaging because the silica may have absorbed qualities of the medication.

Liquid Potpourri and Essential Oils

Liquid potpourri is used as a fragrance, often heated over a candle or other heat source (Figure 31-10). Cats may be exposed by ingesting the oil directly from the container or licking oil from fur or feet. Liquid potpourri is made with essential oils, sometimes in combination with cationic detergents, both of which can be harmful.³⁵ Essential oils can cause mucous membrane and gastrointestinal irritation, CNS depression, and dermal hypersensitivity and irritation. Severe clinical signs can be seen with potpourri products that contain cationic detergents, which can be corrosive to the skin and mucous membranes. Dermal exposure to cationic detergents can result in erythema, edema, intense pain, and ulceration. Ingestion of cationic detergents can lead to tissue necrosis and inflammation of the mouth, esophagus, and



FIGURE 31-10 Liquid potpourri is heated in an electric appliance or over a candle as a room fragrance. It contains essential oils and cationic detergents that are highly toxic to cats if ingested. (*Courtesy Dr. Vicki Thayer.*)

stomach.^{35,49} Additional clinical signs include hyperthermia, tachypnea, ptalism, and lethargy.⁴⁹ Cationic detergents can also be found in some household cleaners, disinfectants, sanitizers, and fabric softeners.

If the exposure is detected promptly, dilution of the chemicals with milk or water may be attempted.²⁸ Induction of emesis is avoided, as is the use of activated charcoal.²⁸ Sulcralfate as a slurry can be used to coat and protect oral lesions. Pain management is indicated for cats with ulcerations. Supportive care, including nutritional support (sometimes through a nasogastric or esophageal feeding tube) and intravenous fluids, may be required for several days.

Melaleuca oil is another essential oil derived from the leaves of the Australian tea tree (*Melaleuca alternifolia*). Melaleuca oil products are sold as topical treatments for skin problems, as insect repellants, and for many other uses. Unfortunately, it is a myth that melaleuca oil is nontoxic for dogs and cats; toxicities are not uncommon, especially when 100% oil is used topically for parasite control. The toxic components are cyclic hydrocarbons and terpenes.⁷

Melaleuca oil is rapidly absorbed from the skin and gastrointestinal tract.⁷ Cats are probably at higher risk of toxicity than dogs because they have a limited ability to perform hepatic glucuronidation of terpenes.⁷ Clinical signs of toxicity include weakness, ataxia, muscle tremors, depression, vomiting, diarrhea, and hypothermia.^{25,53} Elevation in liver enzymes and even liver failure may occur.²⁵ Onset of clinical signs is 2 to 8 hours, and duration is 1 to 2 days.^{7,53}

There is no antidote for tea tree oil toxicity, and treatment consists of decontamination and supportive care (e.g., intravenous fluids, maintenance of body temperature). Removal of the oil from the skin can be

accomplished by bathing in a mild shampoo or detergent. If ingestion has occurred as a result of grooming, activated charcoal and a cathartic may be used. Induction of emesis is contraindicated.²⁵ Most affected cats recover over a 2- to 3-day period.⁵³

Pennies

United States pennies minted after 1982 are composed of copper plating around a zinc core.³⁸ One penny contains approximately 2440 mg of zinc, and poisoning has been reported as a result of ingesting just one coin.³⁸ Zinc can affect the renal, hepatic, and the hematopoietic tissues. Zinc can also cause hemolytic anemia, which could lead to hemoglobinemia, hemoglobinuria, or both.³⁸ Surgical removal of the penny is recommended over chelation therapy.³⁸ Supportive care, including gastrointestinal protectants, fluid diuresis, and blood transfusions, may be needed.³⁸

Ethylene Glycol

EG is a hydrocarbon derivative.⁴¹ Most commercial anti-freeze preparations contain between 95% to 97% EG and are usually mixed 50:50 when used in automobile radiators. Cats may be exposed accidentally through ingestion of radiator leaks or product spills. Unfortunately, the taste appears to be attractive to cats. EG may also be added to toilets in winter months to prevent pipe freezing, and cats that drink water out of the toilets may be exposed. The minimum lethal dose of undiluted EG is approximately 1.4 mL/kg in cats, so even small ingestions can be dangerous.^{41,47}

EG is rapidly absorbed and can be measured in the blood within 30 minutes. EG is metabolized in the liver to more toxic compounds—to glycoaldehyde by alcohol dehydrogenase, and then glycoaldehyde is oxidized to glycolic acid and glyoxylic acid.⁴¹ Glyoxylic acid is primarily converted to oxalic acid; calcium is bound to oxalic acid, resulting in calcium oxalate crystal formation, which is deposited in the kidneys. A wide range of tissue toxicities is seen with EG toxicity (gastric irritation, CNS dysfunction, metabolic acidosis, and renal failure).⁵⁵

In most cases of EG poisonings, the animal begins vomiting within the first few hours. In 1 to 6 hours, signs of depression, ataxia and knuckling, weakness, tachypnea, hypothermia, polyuria, and polydipsia are seen.^{47,55} Cats are less likely to exhibit polydipsia than dogs.⁵⁵ By 18 to 36 hours, acute oliguric renal failure occurs. The kidneys may be swollen and painful.⁵⁵ Other signs seen with EG toxicosis include seizures, coma, and death.^{41,47}

Common laboratory changes include increased anion gap, hyperosmolality, increased BUN and creatinine, hypocalcemia, isosthenuria, and calcium oxalate crystaluria.^{22,47} Quantitative EG tests can be run through

human diagnostic laboratories on an urgent basis for diagnosis as early as 30 minutes after ingestion.⁴¹ Insofar as the minimum toxic blood level is not known in cats, any detection of EG would necessitate treatment.

In some cases it is helpful to examine the cat's oral cavity, face, paws, vomitus, and urine with a Wood's lamp for fluorescence.⁵⁵ This is because many (but not all) antifreeze solutions contain sodium fluorescein. Detection of fluorescence in a cat with compatible clinical signs may help confirm the diagnosis, but failure to detect fluorescence does not eliminate the possibility of EG toxicity.

In most cases of feline exposure to EG, decontamination steps are not helpful because it is absorbed too quickly. Emesis may be helpful only if it occurs within 30 minutes of exposure, and activated charcoal may be helpful only if given within 1 hour of ingestion.⁴¹ Activated charcoal should not be administered if ethanol treatment is planned because it will inhibit absorption.⁵⁵

The drug 4-methylpyrazole (4-MP) is an alcohol dehydrogenase inhibitor that inhibits conversion of EG to its more toxic metabolites. However, 4-MP has poor efficacy in cats when used at the canine dose and currently is not recommended,^{10,47} although further research to identify the optimal dose may improve outcomes.⁹ Intravenous ethanol is the treatment of choice in cats (see Table 31-1), despite its drawbacks (CNS depression, short-half life). Ethanol is the preferred substrate for alcohol dehydrogenase and is used to inhibit EG metabolism. Ethanol is most effective if started within 12 hours of ingestion.⁴⁷ Intravenous ethanol may have to be continued for up to 72 hours to ensure complete EG elimination. Supportive treatment, including intravenous fluid therapy, control of hyperkalemia and acidosis, diuresis, and nutritional support, is an important part of therapy. Prognosis is poor to guarded with most cases of EG poisoning in cats because most patients are presented for treatment too late in the course of the disease. The earlier treatment is initiated (preferably within 3 hours of ingestion), the better the chance of recovery.

ANIMAL HAZARDS

Bufo Toad

Cane or marine toads (*Bufo marinus*) are native to Central and South America and have been introduced throughout Oceania (including Australia) and the Caribbean. Colorado River toads (*Bufo alvarius*) are found in several U.S. states, including Florida, Texas, and Colorado. These toads are highly toxic. The skin of the Bufo toads contains a potent neurotoxin and cardiotoxin called *bufotoxin*.⁴⁵ When a cat has oral contact with this toad, the bitter taste initially causes hypersalivation and vomiting, which may help in self-decontamination. With

ingestion of bufotoxin, clinical signs can include weakness, cardiac arrhythmias, seizures, nystagmus, coma, and death.⁴⁵

Diagnosis is based on a history of exposure and compatible clinical signs. The mouth of a cat suspected of oral exposure to *Bufo* toads should be flushed thoroughly with water to remove any residue and the patient treated supportively for neurologic and cardiac abnormalities. Seizures may be treated with diazepam, and marked bradycardia with atropine. Most animals recover if treated early.

Poisonous Snakes

There are two types of poisonous snakes in North America: those that produce myotoxins, and those that produce neurotoxins. Pit vipers (crotalids) include rattlesnakes, copperheads, and water moccasins. Pit vipers are widespread in the United States and account for 99% of all snakebites.³² Crotalid venom causes a disseminated intravascular coagulopathy-like syndrome but also has effects on many body systems because of the complex mix of toxins produced by the various snakes.^{8,32} Clinical signs include severe localized pain at the site of the bite wound, salivation, weakness, hypotension, and bleeding. Onset of clinical signs may occur several hours after the bite occurred.

Coral snakes (elapids) are less commonly involved in envenomation because they must chew on their victim to inject venom, and their venom potency is low.³² Elapid venom affects the CNS, and clinical signs do not occur for more than 10 hours after contact.⁸ It may take up to 2 weeks for the venom to be cleared.³² In cats clinical signs include ascending flaccid quadriplegia, loss of cutaneous nociception, depression, and hypothermia. Death is typically caused by respiratory collapse.

According to studies, specific antivenins can increase the chances of a full recovery but only if they are used before moderate to severe signs are seen.³² Unfortunately, most snake bites in cats are not witnessed, and most envenomation may not be identified until the cat becomes symptomatic. In addition to antivenins, cases of snake bites should be treated symptomatically and supportively according to the clinical signs and affected body systems. Prognosis is poor with severe clinical signs.

Poisonous Spiders

There are two types of poisonous spiders in North America. Just as with snake bites, most spider bites are not witnessed, and until clinical signs are seen or the bite wound is visualized, the spider bite may not be known. The venom of the widow spiders (*Latrodectus* spp.) contains potent neurotoxins including alpha-latrotoxin, which causes a massive release of neurotransmitters,

including acetylcholine, norepinephrine, and dopamine.⁴⁴ Unlike the brown recluse bite, there is minimal tissue reaction at the site of the bite of the widow spiders. Diagnosis usually depends on onset of systemic clinical signs, which begin within 8 hours of envenomation.⁸ The bite causes severe generalized pain, which results in the cat becoming extremely vocal and agitated. Hypersalivation, vomiting, diarrhea, and tremors are also commonly seen. Later stages result in muscle paralysis and death caused by cardiopulmonary collapse.⁴⁴ Supportive and symptomatic therapy is required for stabilization, including pain management.^{8,44} The most effective treatment is the slow intravenous administration of antivenin (*Latrodectus* antivenin). The product acts rapidly, with resolution of clinical signs within 30 minutes.⁸

Because most bites by the brown recluse or violin spider (*Loxosceles* spp.) go unnoticed, the cat is usually presented with a severe wound that is several days old. *Loxosceles* venom contains necrotizing enzymes, and the lesions produced by them can persist for months.⁸ One portion of the venom, the enzyme sphingomyelinase, may cause systemic effects, including hemolysis, hyperthermia, and nausea.⁸ Although there is no specific antivenin for this spider bite, dapsone has been recommended to inhibit the influx of neutrophils and thus vasculitis at the site of the bite (see Table 31-1).^{8,44} Dapsone should be administered within 36 hours of the envenomation.⁴⁴

DRUGS

Cats may be exposed to dangerous medications either through accidental exposure or through inappropriate use by uninformed owners.

Acetaminophen

Acetaminophen (also known as paracetamol) is a derivative of P-aminophenol that has analgesic and antipyretic activity. Acetaminophen inhibits the effects of pyrogens by blocking prostaglandin synthesis and also by inhibiting cyclooxygenase (COX), which results in an increased pain threshold.³⁶ It is a common ingredient found in many human pain-relief products and cold and flu remedies and is available in more than 200 prescription and nonprescription formulations.³⁶ Cats are typically exposed when well-intentioned but uninformed owners administer the drug without consulting a veterinarian.

Although acetaminophen can be used safely in canine patients at a dose of 10 mg/kg every 12 hours, there is no safe dose for cats.³⁶ In fact, a single dose of 10 mg/kg has produced signs of poisoning in cats.³⁶ The reason for feline sensitivity to acetaminophen is based on the limited glucuronyl transferase activity in this species.

Glucuronidation and sulfation pathways become saturated, and cellular glutathione stores are depleted. This results in the production of a highly reactive metabolite, *N*-acetyl-parabenzenquinoneimine (NAPQI), which causes hepatocyte damage and also oxidative stress to red blood cells that leads to hemolysis and methemoglobinemia.³⁶

The major adverse effects of acetaminophen toxicosis are related to methemoglobin and hemolysis. Clinical signs of acetaminophen poisoning in cats include vomiting, lethargy, facial and paw edema, brown-colored mucous membranes, dyspnea, and death.⁴³ Centrilobular or diffuse hepatic necrosis is also a possible complication but less common in cats than in dogs.^{36,43}

With recent exposures to acetaminophen, decontamination through emesis and activated charcoal should take place. However, before administering activated charcoal, a preliminary dose of *N*-acetyl cysteine (NAC) should be given (see Table 31-1). NAC binds reactive toxic metabolites and is a glutathione precursor.⁴³ Treatment is most effective if started within 8 hours of ingestion.⁴³ Activated charcoal can then be given 2 hours later to help prevent the adsorption of NAC. NAC therapy and supportive care are indicated with any exposure. Cimetidine or ranitidine may be included as part of the treatment to reduce the metabolism of acetaminophen.⁴³ There is also some evidence that silymarin⁴ and s-adenosylmethionine⁵⁷ may protect liver tissue against oxidative damage in cats with acetaminophen toxicity.

Ibuprofen and Other Nonsteroidal Antiinflammatory Drugs

The NSAIDs reduce inflammation by inhibiting the COX enzyme system. NSAID toxicity affects the gastrointestinal, renal, hepatic, and CNS through a variety of effects on COX enzymes. Most cats are poisoned through inappropriate administration by owners.

Ibuprofen is a substituted phenylalkanoic acid with nonsteroidal antiinflammatory, antipyretic, and analgesic properties. According to studies of acute ingestion of ibuprofen in dogs, vomiting, diarrhea, nausea, anorexia, gastric ulceration, and abdominal pain can be seen with doses of 50 to 125 mg/kg. These signs in combination with renal damage can be seen at doses at or above 175 mg/kg. At doses at or above 400 mg/kg, CNS effects such as seizure, ataxia, and coma may occur.³⁶ Cats are considered to be twice as sensitive as dogs because they have a limited glucuronyl-conjugating capacity.³⁶ Other NSAIDs may have similar toxicity, but limited published data are available.

The most common signs of NSAID toxicoses include anorexia, nausea, vomiting, lethargy, diarrhea, melena,

ataxia, polyuria, and polydipsia. Postmortem lesions associated with NSAID toxicoses include perforations, erosion, ulceration, and hemorrhage of the gastrointestinal tract.³⁶

The primary goal of treatment is to prevent or treat gastric ulceration, renal failure, CNS effects, and possibly hepatic effects. There is no specific antidote available. Prognosis is good if the animal is treated promptly and appropriately. Delay in treatment can decrease survival potential with large exposures. Fluid diuresis for 24 to 48 hours is recommended when ibuprofen doses approach or exceed approximately 75 mg/kg in cats.³⁶ Peritoneal dialysis may be necessary if unresponsive oliguric or anuric renal failure develops.

Misoprostol (Cytotec, Pfizer) may be helpful for treating or preventing gastric ulceration caused by ibuprofen.³⁶ Sucralfate can be used to bind to erosions and ulcers and protect them from exposure to gastric acid, bile acids, and pepsin.³⁶ H₂ blockers or proton pump inhibitors may also be helpful.

Gastric protection is recommended for at least 5 to 7 days. When renal failure is possible, BUN, creatinine, and urine specific gravity should be monitored closely. A baseline level and then rechecks at 36, 48, and 72 hours are recommended.³⁶ The animal should also be monitored for acidosis and electrolyte shifts during treatment. Symptomatic treatment for gastric signs and renal failure should be provided until the animal fully recovers.

Aspirin

Aspirin (acetylsalicylic acid) is the salicylate ester of acetic acid and is derived from phenol. Salicylates are commonly used as analgesics, and they also have antipyretic and antiinflammatory properties. In addition to its antiprostaglandin effect, aspirin uncouples oxidative phosphorylation and can cause an increase in oxygen consumption and carbon dioxide production. Aspirin can also inhibit platelet aggregation. Unlike ibuprofen, renal injury is uncommon in acute aspirin toxicosis, although hepatic injury has occasionally been reported.

The therapeutic dose in cats is 10 to 20 mg/kg every 48 hours.³⁴ Cats are deficient in glucuronyl transferase and therefore have a prolonged excretion rate and are more susceptible to toxicity.⁴³ For example, a dose of 25 mg/kg in a cat has an elimination half-life of almost 45 hours.³⁴ Caution is necessary for doses of more than 30 mg/kg, especially in geriatric cats, kittens, and cats with preexisting renal or hepatic disease.

Signs of aspirin toxicosis may include fever, hyperventilation, vomiting, melena, abdominal pain, seizures, and coma.⁴³ Clinical laboratory abnormalities may include elevations in liver enzymes, respiratory alkalosis, metabolic acidosis, and increased bleeding time.⁴³ There is no specific antidote, so treatment is symptomatic and

supportive, including decontamination, intravenous fluid therapy to maintain renal function, and management of acidosis. Gastrointestinal protection with sucralfate or cimetidine may prevent further damage to the mucosa but may be required for up to 2 weeks.⁴³

5-Fluorouracil

5-Fluorouracil (5-FU) is a fluorinated pyrimidine antagonist, which acts as an antineoplastic antimetabolite.⁷ It is used in human patients to treat solar and actinic keratoses and some superficial skin tumors. Topical fluorouracil is available as 1% or 5% cream (Efudex, Valeant Pharmaceuticals). 5-FU can inhibit RNA processing and functioning as well as DNA synthesis and repair. The toxicity effects of 5-FU, as with other antineoplastic agents, occur mainly through destruction of rapidly dividing cell lines such as bone marrow stem cells and the epithelial layer of the intestinal crypts.⁷ The drug is no longer recommended for topical treatment of squamous cell carcinoma in cats because it can cause severe toxicity and death. Cats are very sensitive to the effects of 5-FU, and even a few licks may cause life-threatening toxicity.⁷

Early effects seen with 5-FU include generalized grand mal seizures, tremors, vomiting, and ataxia.⁷ Cardiac arrhythmia, respiratory distress, and hemorrhagic gastroenteritis are also seen. Clinical signs develop within 1 hour and are usually life threatening. Death often occurs within 6 to 16 hours after exposure. Induction of emesis is usually not recommended, given the rapid onset of toxicity. Thorough gastric lavage may be the preferred method of decontamination, followed by activated charcoal.⁷ Seizures and tremors are often poorly responsive to treatment with diazepam, and other options, such as barbiturates, propofol, or gas anesthesia, must be used. Further treatment may include blood transfusion, intravenous fluid therapy, gastrointestinal protectants, analgesics, and broad-spectrum antibiotics. In those cats that survive the initial effects, bone marrow suppression may take 2 to 3 weeks to resolve.⁷

Isoniazid

Isoniazid (INH) is a medication used to treat tuberculosis and has a very narrow margin of safety.⁵⁶ The LD₅₀ is estimated to be about 50 mg/kg in dogs. Isoniazid is available as an elixir, injection, syrup, and tablets (in strengths of 50, 100, and 300 mg). INH causes a decreased level of gamma-aminobutyric acid in the brain, and it also depletes the CNS of pyridoxine, the precursor of the coenzyme pyridoxal phosphate, which is necessary for the activity of the enzyme glutamic acid decarboxylase.⁵⁶ Overdoses produce life-threatening signs, including seizures, acidosis, and coma. Pyridoxine (vitamin B₆) is a direct agonist of INH.⁵⁶

PRINCIPLES OF TREATMENT

When facing a potential poisoning exposure, the veterinarian must first evaluate the situation. It is very important to obtain as much information as possible about the exposure. *Who, what, when, and how* are key questions to ask your client. First, who was exposed? Second, what is the patient's signalment? Next, to what was the animal exposed? Finally, when did the exposure occur, and how? Obtaining the medical history and the exposure history is very important and can affect the way the patient is treated. Important elements of the toxicologic history are found in [Box 31-3](#).

The initial examination of the patient should be performed quickly, with as little stress as possible, and should include assessment of the respiratory rate, capillary refill time, mucous membrane color, heart rate, and body temperature. Examination of a cat that is unconscious, in shock, seizing, or in distress must be conducted simultaneously with stabilization measures. If the cat is stable, a full history of the cat and the exposure should be taken, followed by a systematic physical

BOX 31-3

Important Elements in Taking a Toxicologic History

1. Listen to the client, avoiding bias or preconceptions.
2. Observe the animal.
3. Identify and treat immediate life-threatening problems (e.g., seizures, arrhythmias); do not wait for confirmation of poisoning to initiate supportive therapy.
4. Identify the animal's home environment; determine whether other animals or children could be involved.
5. Identify any current medications, preexisting disease conditions, or pertinent previous medical history.
6. Investigate the history of the exposure: how long ago, what toxin, what concentration, how much? What toxins/poisons could be in the home environment?
7. Identify the poison if possible: Estimate the mg/kg dose, and determine the worst-case scenario.
8. Establish a timeline for the exposure and onset of clinical signs: Is the animal getting better, deteriorating, or showing no clinical signs?
9. Establish a minimum database.
10. Always treat the patient, not the poison.

Modified from Fitzgerald K: Taking a toxicological history. In Peterson M, Talcott P, editors: *Small animal toxicology*, ed 2, St Louis, 2006, Saunders Elsevier, pp 39-46.

examination. Patterns of clinical signs may be suggestive for certain drugs or toxins ([Table 31-3](#)).

In cases of contact with chemicals or medicines, the veterinarian should ask the pet owner to bring in the packaging information to confirm product contents. For example, a pet owner may say that the cat was given a "baby aspirin" when in fact it was actually an aspirin-free formulation containing acetaminophen. The treatment and prognosis for acetaminophen are quite different than those for aspirin.

Stabilization of Vital Functions

Stabilizing the cat is always a priority, and the ABCs should always be followed (i.e., airway, breathing, circulation). A patent airway should be established and artificial respiration given if the animal is dyspneic. The cardiovascular system should be monitored closely, preferably with an electrocardiographic monitor, and any cardiovascular abnormality should be corrected. If cardiac arrest occurs, cardiopulmonary resuscitation should be attempted. The placement of an indwelling intravenous catheter may be necessary for the administration of medications and intravenous fluids. Anticonvulsant therapy should be given if the animal has tremors or seizures (see [Table 31-1](#)), and attention should be paid to hypothermia or hyperthermia.

Once the cat is stable, metabolic derangements should be assessed and a management plan formulated. The minimum database for a suspected toxicosis includes a complete blood cell count, serum electrolytes, serum chemistries (especially glucose, BUN, creatinine, calcium), and urinalysis. Depending on the presenting signs and the suspected toxin, other laboratory tests may be necessary, such as coagulation testing, electrocardiography, blood gases, and radiographs of the chest and abdomen.

Decontamination

Preventing absorption of the toxicant through decontamination is important for treating a toxicosis. It is also important that the members of the veterinary staff protect themselves from exposure while treating affected animals by using protective gear such as gloves, safety glasses, and aprons. After dermal exposure to a toxicant, the cat should be bathed with a mild liquid hand dish detergent or a non-insecticidal shampoo, then thoroughly rinsed and dried. It may be necessary to repeat the procedure. Care should be taken when bathing debilitated or unstable animals. Oily substances can be removed with commercial hand-cleaning degreasers (avoiding those containing citrus), but care should be taken to wash the animal afterward with warm water and soap to remove the degreasing product. It may help to clip the hair coat of longhaired cats. The veterinarian

TABLE 31-3 Clinical Signs Associated with Selected Toxins in Cats

Toxin	Vital Signs	Mentation	Clinical Signs and Findings
Acetaminophen	Normal in early stages	Normal	Anorexia, vomiting, facial and paw edema, brown mucous membranes, dyspnea
Ethylene glycol	Tachypnea	Lethargy to coma	Vomiting, depression, ataxia, seizures, hypothermia
Metaldehyde	Hyperthermia, tachypnea, tachycardia	Lethargy to coma	Salivation, ataxia, tremors, seizures
Organophosphates and carbamates	Changes in blood pressure, heart rate, respiration	Lethargy to coma	Vomiting, diarrhea, salivation, tremors, seizures, ataxia
Pyrethrins/pyrethroids	Variable	Lethargy to coma	Vomiting, diarrhea, salivation, tremors, ataxia, seizures, mydriasis
Salicylates (aspirin)	Hyperthermia, tachycardia	Agitation, then lethargy to coma	Anorexia, vomiting, melena, abdominal pain, seizures

Modified from Fitzgerald K: Establishing a minimum database in small animal poisonings. In Peterson M, Talcott P, editors: *Small animal toxicology*, ed 2, St Louis, 2006, Saunders Elsevier, pp 61-73.

should monitor the skin for redness, swelling, or pain. Skin exposed to caustic substances should be flushed thoroughly with tepid water and handled gently to avoid mechanical injury. It is important to note that most dermal exposures in cats also lead to oral exposure through grooming.

For ocular exposures a minimum of 20 to 30 minutes of irrigation with tepid tap water, lactated Ringer's solution, or physiologic saline is recommended.⁴⁶ Fractious animals or those experiencing pain may require sedation. If the substance is corrosive, the veterinarian should examine the eyes for evidence of corneal ulceration. In cases involving corneal damage, the cat may require follow-up examinations or consultations with a veterinary ophthalmologist.

Inducing emesis can help remove toxicants from recent ingestions. Cats do have the ability to vomit; however, the length of time since ingestion, the cat's age, its previous medical history, and the type of poison can affect the decision to attempt emesis. Any cat with a history of cardiovascular abnormalities, epilepsy, recent abdominal surgery, or severe debilitation is not a candidate for emesis induction.⁴⁶ Emesis should not be induced in an animal that is severely depressed or in a coma because doing so could lead to aspiration. On the other hand, inducing vomiting in a hyperactive animal could trigger a seizure. Moreover, inducing vomiting in a cat that has already vomited is usually not recommended.

Another factor affecting the decision to induce emesis is the nature of the substance ingested. Emesis is contraindicated for corrosive materials such as cationic detergents, acids, and alkali. Induction of vomiting is not recommended with corrosives because of re-exposure of the esophageal tissues to the corrosive material. Instead, dilution with milk or water is the recommended initial

treatment. Emesis is also contraindicated when a hydrocarbon has been ingested, with the main concern being aspiration. Some examples of hydrocarbon-containing products are tar, lubrication oils, fuel oil, kerosene, mineral spirits, and gasoline.

Establishing the time of exposure is important because emesis will be useful only if induced soon after the exposure (typically within 4 hours) and if food or liquid is present in the stomach. Several substances have been used as emetics but are generally unreliable and not recommended (e.g., dry salt, salt water, liquid dishwashing soap).⁴⁶ Other substances, such as syrup of ipecac, have been used, but the safest and most reliable emetics are hydrogen peroxide and apomorphine (see Table 31-1).

Hydrogen peroxide solution (3%), which can be purchased over the counter, has been shown to be an effective emetic for cats. Hydrogen peroxide can be administered with an eyedropper or a syringe by owners at home, under the direction of a veterinarian. Hydrogen peroxide causes mild gastric mucosal irritation, resulting in emesis. Typically, vomiting occurs within 15 minutes. If not, 3% hydrogen peroxide can be repeated one additional time at the same dose. Apomorphine can be used in cats to induce emesis; however, caution should be exercised because some cats will experience a paradoxical hyperactivity reaction with opioids. It is also not as reliable an emetic in cats as in dogs.

In cases when emesis is contraindicated but emptying stomach contents is essential, a gastric lavage under general anesthesia and using a cuffed endotracheal tube may be considered. A stomach tube is passed no further than the xiphoid process, and the patient is positioned with the head lower than the chest at approximately a 20-degree angle. Warm saline (10 mL/kg) is instilled, and manual agitation of the stomach is performed while

the fluid is allowed to drain by gravity or is aspirated.³³ The procedure is repeated (often 10 to 20 times) until the lavage fluid is clear. The veterinarian should avoid overdistention of the stomach and occlude the end of the tube before removal. Complications of gastric lavage include aspiration pneumonia, laryngospasm, hypoxia, mechanical injury, and fluid and electrolyte imbalances.³³ Careful technique and patient selection can help minimize these risks.

Activated charcoal is a nonspecific adsorbent that binds to many substances through weak chemical forces and prevents their absorption. It is ineffective for corrosive substances; hydrocarbons; and most heavy metals, such as iron, lead, mercury, and arsenic.⁴⁶ Activated charcoal is available commercially in liquid, powder, and granular forms. The granular or powder form of activated charcoal can be mixed with water to facilitate administration. Activated charcoal can be administered orally with a syringe or by gastric tube (see Table 31-1). It is advisable to wait 1 to 2 hours after attempting emesis before administering activated charcoal to minimize the risk of aspiration through vomiting. However, waiting for more than 2 hours after most exposures will reduce the benefit. For toxins that undergo enterohepatic circulation, repeated doses of activated charcoal every 6 to 8 hours over several days may be beneficial.⁴⁶

When gastric lavage is performed, activated charcoal may be administered before the lavage to slow absorption of the toxin and administered again after the lavage has been completed.³³ Contraindications to the use of activated charcoal include significant risk of aspiration, severe vomiting, and gastrointestinal perforation.⁴⁶

Cathartics increase the clearing of intestinal contents. Two types are readily available: saline solutions (e.g., sodium sulfate [Glauber's salt], magnesium sulfate [Epsom salt]) and saccharide solutions (e.g., sorbitol). Mineral oil should never be used as a cathartic because of the high risk of aspiration and interference with charcoal adsorption.⁴⁶ Cathartics are used to enhance the elimination of activated charcoal and adsorbed toxicant. Premixed products that contain both activated charcoal and a cathartic (usually sorbitol) are available as commercial preparations, or cathartics can be added to solutions of activated charcoal. Veterinarians are advised to administer activated charcoal with a cathartic to facilitate removal of the charcoal-bound substance, unless the cat already has diarrhea or is dehydrated. Increasing the transit time of the activated charcoal through the gastrointestinal system will decrease the chances that the bonds between the charcoal and poison can weaken and allow the poison to be released. If activated charcoal is administered in multiple doses, the cathartic should be included only with the first dose to prevent fluid and electrolyte imbalances. Contraindications to the use of cathartics include ingestion of a corrosive substance,

hypotension, dehydration, electrolyte abnormalities, abdominal trauma, and intestinal damage such as obstruction or perforation.³³

Supportive Care

The cat's clinical signs should be managed with symptomatic and supportive care. The appropriate antidote or antagonist should be administered, if one exists. Monitoring of the acid-base balance, serum chemistry profile, hydration, or electrolytes may be necessary, depending on the potential effects of the toxicant involved. Some toxicants, such as iron, copper, acetaminophen, and arsenic, are hepatotoxic, whereas others, such as estrogen, lead, and antineoplastic medications, can cause anemia.

Diuresis may be beneficial with exposures to nephrotoxic agents or to enhance elimination of the poison. Examples of nephrotoxic agents that could benefit from diuresis include EG, zinc, mercury, oxalic acids, NSAIDs, diquat herbicide, and aminoglycoside antibiotics. Adverse effects associated with diuresis include pulmonary edema, cerebral edema, metabolic acidosis or alkalosis, and water intoxication; therefore close monitoring is necessary.

Supportive care should continue until the patient fully recovers. When the cat is released to the owner, the veterinary staff should take the time to explain what signs should be monitored for and how medication or treatment should be given and schedule follow-up visits as needed.

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