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TOXICITY IN PLANTS
The list of substances that can cause hypoglycemia in dogs varies widely, including the sago palm tree.

MANAGEMENT STRATEGIES

Top 10 Toxicologic Causes of Hypoglycemia in Dogs

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Welcome to Practical Toxicology, brought to you in partnership between Today's Veterinary Practice and the ASPCA Animal Poison Control Center (APCC) (aspcapro.org/poison). This column provides practical clinical information about diagnosing and treating pets that have been exposed to potentially harmful substances.

The APCC:
- Provides 24-hour diagnostic and treatment recommendations by specially trained veterinary toxicologists
- Protects and improves animal lives through toxicology education, consulting services, and case data review
- Developed and maintains AnTox, an animal toxicology database system that identifies and characterizes toxic effects of substances in animals
- Works closely with human poison control centers to provide animal poisoning information
- Offers extensive veterinary toxicology consulting to organizations in industry, government, and agriculture.

If treating a patient that requires emergency care for poisoning, call the APCC at **888-426-4435**.

Five minutes before your clinic closes, a dog arrives laterally recumbent, unresponsive, and profoundly hypoglycemic. The differential diagnoses that run through your mind include insulinoma, hypoadrenocorticism, liver disease, and sepsis, among many others. When reviewing the DAMNITV differential diagnosis scheme (Degenerative, Anomalous, Metabolic, Neoplastic or Nutritional, Inflammatory, Traumatic or Toxic, Vascular), you focus on the "T" (for toxic) and think of numerous substances that can cause hypoglycemia. However, most of them may be encountered only rarely or occur only theoretically. This article is intended to help you rule out the rare causes and focus on those that should be on your short list: the top 10. After you have stabilized the patient, discussing these substances with the client may help pinpoint the cause of hypoglycemia.

We start with the least likely and work our way to the most likely.

10. BACLOFEN

Description: Baclofen is a centrally acting skeletal muscle relaxant.

Clinical signs: Ingestion of baclofen may cause vomiting, hypersalivation, agitation, ataxia, vocalization, mydriasis, depression, recumbency,

hypothermia, hypotension or hypertension, tremors, seizures, coma, and respiratory arrest.

Margin of safety: Narrow. The therapeutic dose for dogs has been listed as 1 to 2 mg/kg PO q8h.[1] However, clinical signs, such as ataxia and recumbency, have occurred after receipt of 0.5 mg/kg. Seizures, loss of gag reflex, and coma have occurred after receipt of 1 to 2 mg/kg.[2]

Mechanism for hypoglycemia: Direct and indirect. The exact mechanism is unknown, but hypoglycemia may result from suppression of glucagon release.[3] In dogs with severe tremor or seizure activity, increased use of glucose could lead to hypoglycemia.

Treatment Tip Be ready to intubate. Dogs often lose their gag reflex early in the course of intoxication and need respiratory support. Baclofen can cause flaccid paralysis of the diaphragm.[4]

9. METALDEHYDE

Description: Metaldehyde is a common active ingredient in molluscicides.

Clinical signs: Ingestion of metaldehyde most often results in seizures, hypersalivation, vomiting, diarrhea, hyperesthesia, tremors, twitching, ataxia, hyperthermia, tachycardia, nystagmus, acidosis, cyanosis, and death.[5] Liver failure may also occur 2 to 3 days later.[2]

Margin of safety: Narrow. Any exposure is cause for concern.

Mechanism for hypoglycemia: Direct and indirect. Severe muscle activity can lead to hypoglycemia from increased metabolic use of glucose. Liver failure can also cause hypoglycemia.

Treatment Tip Metaldehyde exposure is anecdotally described as a "shake and bake" toxic syndrome, and its treatment typically requires methocarbamol and benzodiazepines to control tremors. Hyperthermia, rhabdomyolysis, acidosis, and disseminated intravascular coagulation can result.

8. METHYLXANTHINES

Description: Methylxanthines include caffeine, theobromine, and theophylline. This broad category encompasses chocolate, diet pills, caffeinated beverages, bronchodilators (e.g., aminophylline, theophylline), and more.

Clinical signs: Methylxanthine toxicosis can cause panting, pacing, restlessness, tachycardia, hypertension, hyperthermia, arrhythmias, tremors, and seizures. Vomiting, diarrhea, polyuria, and polydipsia are also common.

Margin of safety: Variable. The margin of safety for chocolate is wide, depending on the type of chocolate (**TABLE 1**); the margin of safety for medications such as theophylline is narrower.

Mechanism for hypoglycemia: Direct and indirect. Methylxanthines may cause increased insulin release and insulin sensitivity.[6] In severely affected dogs, hypoglycemia may result from increased metabolic use of glucose.

Treatment Tip Dogs that have ingested large amounts of chocolate are often hemoconcentrated. The osmotically active environment puts them at high risk for hypernatremia or other electrolyte imbalances. For this reason, activated charcoal should be used with caution. It is not needed in all dogs that have ingested chocolate.

7. SAGO PALM

Description: Sago palms are decorative plants commonly found outdoors in warm climates, but they can be kept indoors in any region. They are also known as cycads. The genera of concern are *Microzamia*, *Zamia*, and *Cycas*.

TABLE 1 Average Methylxanthine Concentration of Chocolates

TYPE	METHYLXANTHINE CONCENTRATION, MG/OZ
White chocolate	1.1
Milk chocolate	65
Dark/semisweet chocolate	165
Baker's chocolate (unsweetened)	400
Dry cocoa powder	790

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Clinical signs: Sago palm toxicosis can cause vomiting and diarrhea (with or without blood), lethargy, depression, dehydration, anorexia, ascites, abdominal pain, icterus, tremors, ataxia, seizures, coma, and death.[7] Liver failure, coagulopathies, and thrombocytopenia may also be seen, but their onset may be delayed by 2 to 3 days.[7]

Margin of safety: Narrow. All parts of the plant are toxic, but the most toxic parts are the seeds. Any exposure is a concern.

Mechanism for hypoglycemia: Direct and indirect. The toxins decrease the activity of mitochondria, adenosine triphosphate, and glucose-6-phosphatase, thereby decreasing gluconeogenesis and glycogenolysis.[8] Hypoglycemia may also be associated with liver failure and/or sepsis.[9]

Treatment Tip The key action to take is aggressive decontamination. For dogs brought to you early, while still asymptomatic, the best course of action is inducing emesis, followed by giving multiple doses of activated charcoal. Cholestyramine may also be used in some cases to decrease enterohepatic recirculation. Cholestyramine is a powdered bile acid sequestrant that is widely available at human pharmacies. It binds bile and, by default, the toxins already bound to the bile. Cholestyramine should be given with food and also aids in elimination of cholecalciferol, amatoxin, and some nonsteroidal anti-inflammatory drugs (NSAIDs) (see numbers 3 and 4 below).

6. ZINC AND ALUMINUM PHOSPHIDE

Description: Zinc phosphide is an agent commonly used for mole and gopher control; aluminum phosphide is used to fumigate grain stores for insect and rodent control. Clinical signs can develop from inhalation of aluminum phosphide. After ingestion of zinc phosphide bait, phosphine gas is released when the product is exposed to the acidic environment of the stomach. The phosphine gas is rapidly absorbed via inhalation during eructation or across the gastric mucosa. The hydrolyzed phosphine causes significant oxidative damage throughout the body. Zinc toxicosis is not expected.[10]

Clinical signs: Zinc or aluminum phosphide exposures cause vomiting, hypersalivation, tremors, respiratory distress, ataxia, weakness, hyperesthesia, and seizures and, in some cases, may progress quickly to death. It is common for patients to have a strong odor to their breath, often described as a pungent garlic smell. Neurologic signs can develop soon after ingestion. Liver and kidney damage may occur days to weeks after ingestion.[2]

Margin of safety: Narrow. Any exposure has the potential to cause clinical signs.

Mechanism for hypoglycemia: Direct and indirect. Impairment of glycogenolysis and gluconeogenesis[11] can lead to hypoglycemia. Adrenal gland injury and low levels of cortisol may also play a role.[12] Animals experiencing severe seizure activity may become hypoglycemic because of increased metabolic use of glucose.

Treatment Tips

- As soon as possible, administer 1 tablespoon of aluminum hydroxide or magnesium hydroxide per 20 pounds of body weight. Doing so will increase the pH of the stomach and decrease the amount of phosphine gas released. If possible, avoid inducing emesis with hydrogen peroxide; ideally, emesis should be induced with apomorphine while the animal is outdoors.
- Phosphine gas is toxic to all living creatures. Clients should drive to the clinic with the windows down, in case the dog vomits in the car. Any questions regarding humans (clients and/or veterinary hospital employees) who may have been exposed to phosphine gas should be immediately directed to Human Poison Control (1-800-222-1222).

5. SYMPATHOMIMETICS

Description: Sympathomimetics directly or indirectly cause an increase in catecholamines at the neuronal junction.[13] Examples of sympathomimetics include amphetamines, prescription medications for attention-deficit/hyperactivity disorder or attention-deficit disorder, phenylpropanolamine, decongestants (e.g., pseudoephedrine), and illicit drugs (e.g., cocaine, crystal methamphetamine, and 3, 4-methylenedioxymethamphetamine [MDMA, ecstasy]).

Clinical signs: Sympathomimetics can cause agitation, aggression, ataxia, tachycardia or bradycardia, hypertension, hyperthermia, vocalization, mydriasis, tremors, and seizures.

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MUSHROOM POISONING
Mushrooms most likely to cause hypoglycemia are those that contain amatoxins. Pictured here is *Amanita phalloides* [death cap].

Margin of safety: Variable, depending on the substance.

Mechanism for hypoglycemia: Direct and indirect. Hypoglycemia may result from increased use of glucose[14] and possibly increased release of insulin.[15]

Treatment Tip Treatment with acepromazine is very effective because of its α-adrenergic and dopaminergic-blocking effects.[14] Cyproheptadine may be used in conjunction with acepromazine if signs of serotonin syndrome (e.g., mydriasis, vocalization, hyperthermia, hyperesthesia, agitation, tachycardia, or fasciculation) are seen.

4. NSAIDs

Description: Therapeutic doses of veterinary use approved NSAIDs (e.g., carprofen or deracoxib) or low doses of human use–labeled NSAIDs (e.g., ibuprofen or celecoxib) are not expected to cause hypoglycemia in dogs. However, it is possible to see hypoglycemia with large overdoses of NSAIDs. Ibuprofen accounts for almost half of the documented cases of NSAID-associated hypoglycemia at the Animal Poison Control Center.[2] These cases may be overrepresented because of the popularity of ibuprofen.

Clinical signs: Overdoses of NSAIDs can lead to ulceration of the gastrointestinal tract and renal damage. Clinical signs may include vomiting, diarrhea, depression, melena, hematemesis, polydipsia, and polyuria. Ingestion of more than 350 mg/kg of ibuprofen can lead to neurologic signs, such as ataxia, tremors, seizures, and coma.

Margin of safety: Variable. Most dogs that demonstrate signs of ibuprofen toxicosis with hypoglycemia had ingested more than 350 mg/kg.[2]

Mechanism for hypoglycemia: Direct and indirect. NSAIDs are thought to cause hypoglycemia because of their effect on pancreatic β cells, causing increased insulin secretion.[16] Increased metabolic use of glucose (seizure activity) can lead to hypoglycemia.

Treatment Tip Naloxone may help reverse ibuprofen-induced central nervous system depression or coma.[17]

3. MUSHROOMS WITH AMATOXINS

Description: Most mushrooms ingested by dogs are never identified; the suspected diagnosis is often made from the clinical picture and history. Mushrooms most likely to cause hypoglycemia are those that contain amatoxins (e.g., *Amanita phalloides* [death cap], *Amanita verna* [spring destroying angel], *Galerina autumnalis* [deadly galerina], and *Lepiota josserandii* [deadly parasol]). The toxic components of these mushrooms consist of amatoxins, phallotoxins, and virotoxins.[18]

Clinical signs: Death cap mushrooms are among the most widely studied. Exposure to a death cap mushroom can cause clinical signs that occur in 3 phases:

1. **Phase 1** includes moderate to severe gastrointestinal signs, tachycardia, fever, and hyperglycemia lasting 24 hours.
2. **Phase 2** is a latent phase and may last as long as 24 hours.
3. **Phase 3** includes clinical signs such as hypoglycemia, liver and renal failure, coagulopathy, cerebral edema, acidosis, encephalopathy, coma, sepsis, and death.[18]

Margin of safety: Very narrow. Any exposure is a concern.

Mechanism for hypoglycemia: Direct and indirect. Severe hypoglycemia can occur, presumably resulting from the breakdown of glycogen in the liver.[19] In addition, insulin release may result from a cytotoxic effect on β cells.[20] Hypoglycemia can also result from liver failure.

Treatment Tip Early and aggressive decontamination is imperative (see Treatment Tip for Sago palm).

NexGard®
(afoxolaner) Chewables

CAUTION: Federal (USA) law restricts this drug to use by or on the order of a licensed veterinarian.

Description:
NexGard® (afoxolaner) is available in four sizes of beef-flavored, soft chewables for oral administration to dogs and puppies according to their weight. Each chewable is formulated to provide a minimum afoxolaner dosage of 1.14 mg/lb (2.5 mg/kg). Afoxolaner has the chemical composition 1-Naphthalenecarboxamide, 4-[5- [3-chloro-5-(trifluoromethyl)-phenyl]-4, 5-dihydro-5-(trifluoromethyl)-3-isoxazolyl]-N-[2-oxo-2-[(2,2,2-trifluoroethyl)amino]ethyl.

Indications:
NexGard kills adult fleas and is indicated for the treatment and prevention of flea infestations *(Ctenocephalides felis),* and the treatment and control of Black-legged tick *(Ixodes scapularis),* American Dog tick *(Dermacentor variabilis),* Lone Star tick *(Amblyomma americanum),* and Brown dog tick (*Rhipicephalus sanguineus*) infestations in dogs and puppies 8 weeks of age and older, weighing 4 pounds of body weight or greater, for one month. NexGard is indicated for the prevention of *Borrelia burgdorferi* infections as a direct result of killing *Ixodes scapularis* vector ticks.

Dosage and Administration:
NexGard is given orally once a month, at the minimum dosage of 1.14 mg/lb (2.5 mg/kg).

Dosing Schedule:

Body Weight	Afoxolaner Per Chewable (mg)	Chewables Administered
4.0 to 10.0 lbs.	11.3	One
10.1 to 24.0 lbs.	28.3	One
24.1 to 60.0 lbs.	68	One
60.1 to 121.0 lbs.	136	One
Over 121.0 lbs.	Administer the appropriate combination of chewables	

NexGard can be administered with or without food. Care should be taken that the dog consumes the complete dose, and treated animals should be observed for a few minutes to ensure that part of the dose is not lost or refused. If it is suspected that any of the dose has been lost or if vomiting occurs within two hours of administration, redose with another full dose. If a dose is missed, administer NexGard and resume a monthly dosing schedule.

Flea Treatment and Prevention:
Treatment with NexGard may begin at any time of the year. In areas where fleas are common year-round, monthly treatment with NexGard should continue the entire year without interruption.

To minimize the likelihood of flea reinfestation, it is important to treat all animals within a household with an approved flea control product.

Tick Treatment and Control:
Treatment with NexGard may begin at any time of the year (see **Effectiveness**).

Contraindications:
There are no known contraindications for the use of NexGard.

Warnings:
Not for use in humans. Keep this and all drugs out of the reach of children. In case of accidental ingestion, contact a physician immediately.

Precautions:
Afoxolaner is a member of the isoxazoline class. This class has been associated with neurologic adverse reactions including tremors, ataxia, and seizures. Seizures have been reported in dogs receiving isoxazoline class drugs, even in dogs without a history of seizures. Use with caution in dogs with a history of seizures or neurologic disorders (see **Adverse Reactions** and **Post-Approval Experience**).

The safe use of NexGard in breeding, pregnant or lactating dogs has not been evaluated.

Adverse Reactions:
In a well-controlled US field study, which included a total of 333 households and 615 treated dogs (415 administered afoxolaner; 200 administered active control), no serious adverse reactions were observed with NexGard.

Over the 90-day study period, all observations of potential adverse reactions were recorded. The most frequent reactions reported at an incidence of > 1% within any of the three months of observations are presented in the following table. The most frequently reported adverse reaction was vomiting. The occurrence of vomiting was generally self-limiting and of short duration and tended to decrease with subsequent doses in both groups. Five treated dogs experienced anorexia during the study, and two of those dogs experienced anorexia with the first dose but not subsequent doses.

Table 1: Dogs With Adverse Reactions.

	Treatment Group			
	Afoxolaner		Oral active control	
	N[1]	% (n=415)	N[2]	% (n=200)
Vomiting (with and without blood)	17	4.1	25	12.5
Dry/Flaky Skin	13	3.1	2	1.0
Diarrhea (with and without blood)	13	3.1	7	3.5
Lethargy	7	1.7	4	2.0
Anorexia	5	1.2	9	4.5

[1]Number of dogs in the afoxolaner treatment group with the identified abnormality.

[2]Number of dogs in the control group with the identified abnormality.

In the US field study, one dog with a history of seizures experienced a seizure on the same day after receiving the first dose and on the same day after receiving the second dose of NexGard. This dog experienced a third seizure one week after receiving the third dose. The dog remained enrolled and completed the study. Another dog with a history of seizures had a seizure 19 days after the third dose of NexGard. The dog remained enrolled and completed the study. A third dog with a history of seizures received NexGard and experienced no seizures throughout the study.

Post-Approval Experience (July 2018):
The following adverse events are based on post-approval adverse drug experience reporting. Not all adverse events are reported to FDA/CVM. It is not always possible to reliably estimate the adverse event frequency or establish a causal relationship to product exposure using these data.

The following adverse events reported for dogs are listed in decreasing order of reporting frequency for NexGard:

Vomiting, pruritus, lethargy, diarrhea (with and without blood), anorexia, seizure, hyperactivity/restlessness, panting, erythema, ataxia, dermatitis (including rash, papules), allergic reactions (including hives, swelling), and tremors.

Contact Information:
For a copy of the Safety Data Sheet (SDS) or to report suspected adverse drug events, contact Merial at 1-888-637-4251 or www.nexgardfordogs.com.

For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or online at http://www.fda.gov/AnimalVeterinary/SafetyHealth.

Mode of Action:
Afoxolaner is a member of the isoxazoline family, shown to bind at a binding site to inhibit insect and acarine ligand-gated chloride channels, in particular those gated by the neurotransmitter gamma-aminobutyric acid (GABA), thereby blocking pre- and post-synaptic transfer of chloride ions across cell membranes. Prolonged afoxolaner-induced hyperexcitation results in uncontrolled activity of the central nervous system and death of insects and acarines. The selective toxicity of afoxolaner between insects and acarines and mammals may be inferred by the differential sensitivity of the insects and acarines' GABA receptors versus mammalian GABA receptors.

Effectiveness:
In a well-controlled laboratory study, NexGard began to kill fleas four hours after initial administration and demonstrated >99% effectiveness at eight hours. In a separate well-controlled laboratory study, NexGard demonstrated 100% effectiveness against adult fleas 24 hours post-infestation for 35 days, and was ≥93% effective at 12 hours post-infestation throug h Day 21, and on Day 35. On Day 28, NexGard was 81.1% effective 12 hours post-infestation. Dogs in both the treated and control groups that were infested with fleas on Day -1 generated flea eggs at 12- and 24-hours post-treatment (0-11 eggs and 1-17 eggs in the NexGard treated dogs, and 4-90 eggs and 0-118 eggs in the control dogs, at 12- and 24-hours, respectively). At subsequent evaluations post-infestation, fleas from dogs in the treated group were essentially unable to produce any eggs (0-1 eggs) while fleas from dogs in the control group continued to produce eggs (1-141 eggs).

In a 90-day US field study conducted in households with existing flea infestations of varying severity, the effectiveness of NexGard against fleas on the Day 30, 60 and 90 visits compared with baseline was 98.0%, 99.7%, and 99.9%, respectively.

Collectively, the data from the three studies (two laboratory and one field) demonstrate that NexGard kills fleas before they can lay eggs, thus preventing subsequent flea infestations after the start of treatment of existing flea infestations.

In well-controlled laboratory studies, NexGard demonstrated >97% effectiveness against *Dermacentor variabilis,* >94% effectiveness against *Ixodes scapularis,* and >93% effectiveness against *Rhipicephalus sanguineus,* 48 hours post-infestation for 30 days. At 72 hours post-infestation, NexGard demonstrated >97% effectiveness against *Amblyomma americanum* for 30 days. In two separate, well-controlled laboratory studies, NexGard was effective at preventing *Borrelia burgdorferi* infections after dogs were infested with *Ixodes scapularis* vector ticks 28 days post-treatment.

Animal Safety:
In a margin of safety study, NexGard was administered orally to 8 to 9-week-old Beagle puppies at 1, 3, and 5 times the maximum exposure dose (6.3 mg/kg) for three treatments every 28 days, followed by three treatments every 14 days, for a total of six treatments. Dogs in the control group were sham-dosed. There were no clinically-relevant effects related to treatment on physical examination, body weight, food consumption, clinical pathology (hematology, clinical chemistries, or coagulation tests), gross pathology, histopathology or organ weights. Vomiting occurred throughout the study, with a similar incidence in the treated and control groups, including one dog in the 5x group that vomited four hours after treatment.

In a well-controlled field study, NexGard was used concomitantly with other medications, such as vaccines, anthelmintics, antibiotics (including topicals), steroids, NSAIDS, anesthetics, and antihistamines. No adverse reactions were observed from the concomitant use of NexGard with other medications.

Storage Information:
Store at or below 30°C (86°F) with excursions permitted up to 40°C (104°F).

How Supplied:
NexGard is available in four sizes of beef-flavored soft chewables: 11.3, 28.3, 68 or 136 mg afoxolaner. Each chewable size is available in color-coded packages of 1, 3 or 6 beef-flavored chewables.

NADA 141-406, Approved by FDA

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1050-4493-07
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FRONTLINE VET LABS
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Ingestion of xylitol can cause vomiting, depression, diarrhea, hypoglycemia, increased liver enzymes, ataxia, tremors, and seizures.

2. ANTIHYPERGLYCEMICS

Description: This heading encompasses all oral antihyperglycemic medications and insulin injections. The most common oral antihyperglycemic medications for which the Animal Poison Control Center receives calls are the sulfonylureas (e.g., glipizide and glyburide).[2] The hypoglycemia caused by insulin injections is dose-dependent.

Clinical signs: Aside from hypoglycemia, the clinical signs vary according to the agent.

Margin of safety: Variable. The margin of safety for sulfonylureas and insulin injections is narrow. For others, such as metformin or acarbose, the margin is much wider.

Mechanism for hypoglycemia: The mechanisms vary according to the drug. Sulfonylureas, for example, affect potassium channels on pancreatic β cells, thereby causing release of insulin.[13]

Treatment Tips

- Hypoglycemia with sulfonylurea exposures can be profound, and any exposure is a concern. The hypoglycemic effects can persist for well over 24 hours[2] and after large overdoses may last 72 hours.[13]
- Oral exposure to insulin does not cause hypoglycemia. The insulin is digested in the stomach and is inactivated.

1. XYLITOL

Description: This sugar alcohol is used as a sweetening agent in many foods, candies, mints, chewing gums, and supplements. It is also used as a cooling agent in nasal sprays, diapers, baby wipes, sunscreen, toothpaste, and mouthwashes.[21] Although there are many other sugar alcohols, xylitol is the only sugar alcohol that poses a concern for animal safety.

Clinical signs: Ingestion of xylitol can cause vomiting, depression, diarrhea, hypoglycemia, increased liver enzymes, ataxia, tremors, and seizures. Other signs include liver failure, hepatic encephalopathy, and coagulopathy. Liver failure has been seen within 12 hours[22] but can be delayed up to 72 hours.[23]

Margin of safety: Narrow. Ingestion of 100 mg/kg or more can cause hypoglycemia.

Mechanism for hypoglycemia: Direct and indirect. Xylitol-induced hypoglycemia can be profound and results from insulin release.[22] However, in some animals, liver injury can occur without the hypoglycemic phase. Hypoglycemia can result from liver failure rather than insulin release.[23]

Treatment Tip Xylitol is poorly absorbed by activated charcoal,[24] and charcoal use for xylitol toxicity is not indicated.

CONCLUSION

This list of toxicologic causes of hypoglycemia in the dog can be a useful tool for the small animal veterinarian. Reviewing these substances with the client may prove to be a practical way to determine the cause of hypoglycemia. Although this list of hypoglycemic substances is not comprehensive, it covers the 10 most common toxicologic causes of hypoglycemia in the dog as reported to the ASPCA Animal Poison Control Center. More information can be found by calling the ASPCA Animal Poison Control Center at 1-888-426-4435 and visiting the ASPCApro website (aspcapro.org) for helpful printouts, articles, and newsletters. TVP

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