

# Taking the Bait: METALDEHYDE TOXICOSIS

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**A** golden retriever, Ginger, is brought into your veterinary clinic on the Pacific Coast. Ginger had been playing in the backyard by herself for over an hour. When the owner brought her inside, the dog appeared anxious and was frothing at the mouth. On presentation, the dog is vomiting, has muscle tremors, and is severely hyperthermic. The owner says she does not know what Ginger could have gotten into because she and her husband had spent a lot of time in the yard the previous day. On further questioning, the owner reveals that they had been gardening and had placed down slug bait. Metaldehyde toxicosis is suspected.

Metaldehyde is a snail, slug, and rat poison used primarily in the southern and Pacific coastal areas of the continental United States and in the Hawaiian Islands. Formulated as a liquid, powder, or pelleted bait, metaldehyde is used around vegetable and ornamental gardens. It is a very palatable product that is usually formulated as baits in concentrations of 4% or less.<sup>1</sup> Metaldehyde acts as an attractant as well as a toxicant. Manufacturers try to make the baits more attractive to slugs and snails by adding various food processing by-products (e.g., bran, molasses).<sup>2,3</sup> Some preparations also contain small amounts of arsenic and carbamate (i.e., insecticidal com-

pounds), but these ingredients are generally of less importance than the metaldehyde present.<sup>2,4</sup>

Careless placement of the baits by homeowners can lead to toxicosis because many dogs find the meal and pellets attractive and may readily consume them. Metaldehyde has also been reported as the causative agent in poisoning cases involving children, livestock, and horses in addition to dogs.<sup>5</sup> Although cats are quite sensitive to metaldehyde, it has not been reported to be a major problem in this species.<sup>5</sup>

The LD<sub>50</sub> of metaldehyde is 100 mg/kg in dogs, 60 mg/kg in horses, and 207 mg/kg in cats. The minimum oral lethal dose has been reported to



Patient appears anxious and is panting on physical exam.

be 100 mg/kg for dogs, 200 mg/kg for cattle, and 60 to 300 mg/kg for horses; however, severe clinical effects can be seen at much lower doses.<sup>6</sup>

## MECHANISM OF ACTION

The exact mechanism of toxicity is unknown. It was thought for many years that the signs of metaldehyde toxicosis were related to its degradation to acetaldehyde. However, recent studies suggest that this is unlikely and that metaldehyde itself may be the actual toxicant.<sup>3</sup> Metaldehyde readily crosses the blood-brain barrier where interference with neurotransmitters in the brain results in central nervous system stimulation. Hyperthermia, possibly secondary to muscle tremors, often plays a major role in the pathophysiology of metaldehyde toxicosis. Body temperatures exceeding 107°F (41.7°C) may result in cellular necrosis of all the organ systems within minutes. Metaldehyde is also reported to cause severe metabolic acidosis, which is often associated with hyperpnea and central nervous system depression.<sup>3</sup>

## CLINICAL SIGNS

Dogs frequently tend to eat all of the bait available. The onset of signs may

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be within a few minutes to 3 hours after ingestion of the bait. Initial signs include increased heart rate, anxiety, nystagmus, mydriasis, hyperpnea, panting, hypersalivation (which may be frothy), stiff legs, and ataxia. These signs may be rapidly followed by muscle tremors, vomiting,<sup>4</sup> and hyperthermia (temperatures up to 108°F [42.3°C] have been reported).<sup>1,4</sup> Later stages may develop as depression followed by narcosis. If death occurs, it is usually from respiratory failure and takes place from 4 to 24 hours after exposure. Animals that survive the first 24 hours may develop liver failure within 2 to 3 days.<sup>6</sup> Temporary blindness may occur and may take days to weeks to resolve. Signs in cats are similar to those in dogs, except that nystagmus may be more prevalent.<sup>4</sup>

## DIAGNOSIS

Accurate diagnosis of metaldehyde toxicity is based on a careful correlation of data collected from the history, clinical signs, clinical pathology, and toxicologic analysis.<sup>3</sup> Geographic region and compatible clinical signs

should be considered. Severe hyperthermia, a crucial clinical sign of metaldehyde poisoning, requires special attention because this high degree of hyperthermia is not as commonly observed in other neurotoxicoses. Bait found in the patient's vomitus or lavage material and/or acetaldehyde odor (similar to formaldehyde) on the breath or from stomach contents may be noted during diagnosis. Laboratory analysis of stomach contents or lavage fluid should also be considered, but because of turnaround time, analysis is of little benefit in making a rapid diagnosis. Residues are difficult to detect in tissues and, therefore, may not be reliable for diagnosis.<sup>1</sup>

## TREATMENT

There is no antidote for metaldehyde toxicosis. The main goals in treating animals exposed to metaldehyde are prevention of absorption, controlling clinical signs, monitoring and correcting acidosis and dehydration, and providing supportive care.<sup>5</sup> Emesis may be induced if the patient is asymptomatic. Emesis should be performed within 30 minutes of the ingestion and if the patient is free of any medical conditions that would preclude emesis (e.g., heart disease, epilepsy, recent abdominal surgery). Hydrogen peroxide or apomorphine hydrochloride may be used to induce emesis in dogs and cats. In cases in which large amounts of metaldehyde have been ingested, anesthesia and gastric lavage may be indicated. A cuffed endotracheal tube should always be used when performing gastric lavage to prevent aspiration.

Activated charcoal should be given to dogs and cats at a recommended dose of 1 to 4 g/kg.<sup>2,7</sup> It may be beneficial to repeat the activated charcoal every 6 to 8 hours at half the original dose. Activated charcoal that contains a cathartic, such as sorbitol, can be used as long as the patient is not dehydrated or having diarrhea. Cathartics should be limited to every third dose of activated charcoal. Warm water enemas may also be beneficial

## Glossary

**Ataxia** — Incoordination

**Hyperpnea** — Increased deep and rapid respirations

**Mydriasis** — Excessive dilation of the pupil

**Nystagmus** — Rapid, involuntary movement of both eyeballs

to help remove metaldehyde from the gastrointestinal tract.

To control tremors, methocarbamol may be used. The recommended dose in dogs and cats is 55 to 220 mg/kg given slow IV. Once the volume dose is calculated, the first half of the calculated dose may be given rapidly at a rate not to exceed 2 ml/min. Once the animal has relaxed, the remainder is administered to effect.<sup>7</sup> Methocarbamol may be repeated as needed, but the maximum daily dosage of 330 mg/kg/day should not be exceeded. Diazepam may also be used to control tremors and seizures at a rate of 1 to 5 mg/kg IV.<sup>7</sup> Other anti-convulsants, such as barbiturates, gas anesthesia, or propofol, may be required in severe or refractory cases. Barbiturates should be used carefully because they can compete with enzymes that degrade acetaldehyde.<sup>1,2</sup>

It is also important for veterinary technicians to monitor and correct electrolyte imbalances, blood gases, body temperature, anion gap, and urine pH. Controlling tremors and seizures often corrects hyperthermia. Aggressive cooling measures, such as ice baths, are contraindicated because hypothermia may result from cooling methods. Dehydration, body temperature, electrolyte imbalances, and acidosis may be corrected with IV fluids, such as lactated Ringer's solution or Normosol-R. Urinary excretion of metaldehyde in dogs is less than 1%; therefore, diuresis is not indicated because it does not enhance metaldehyde excretion.<sup>2,3</sup> Myoglobinuria and secondary renal dysfunction may be caused by prolonged and excessive

## Substances to Rule Out<sup>2</sup>

Bromethalin  
Chlorinated hydrocarbons  
Illicit drugs (e.g., amphetamines)  
Lead  
Methylxanthines  
Organophosphates  
Sodium monofluoroacetate  
Strychnine  
Tremorgenic mycotoxins  
Zinc phosphide

tremors or seizures. When this occurs, diuresis is recommended to prevent damage to the kidneys. If blood gases can be monitored, sodium bicarbonate may be used to correct acidosis after other clinical signs are controlled. Technicians should also monitor liver enzymes at baseline, 72 hours after exposure, and then as needed. Other treatment includes symptomatic and supportive care, such as maintaining hydration and nutrition intake.<sup>2</sup>

## CONCLUSION

Metaldehyde toxicosis occurs primarily in specific regions of the country and is characterized by acidosis and severe hyperthermia. When treating emergency patients that meet these criteria, the patient should be tested for metaldehyde toxicosis. Although metaldehyde ingestion can be life threatening, rapid decontamination and treatment can lead to a successful outcome.

## REFERENCES

1. Carson TL, Osweiler GD: Insecticides and molluscicides, in Morgan RV (ed): *Handbook of Small Animal Practice*, ed 3. Philadelphia, WB Saunders, 1997, pp 1256–1258.
2. Dolder LK: Metaldehyde toxicosis. *Vet Med* 98(3):213–215, 2003.
3. Puschner B: Metaldehyde, in Peterson ME, Talcott PA (eds): *Small Animal Toxicology*. Philadelphia, WB Saunders, 2001, pp 553–562.
4. Beasley VR: Toxicants associated with CNS stimulation or seizures, in *A Systems Affected Approach to Veterinary Toxicology*. Urbana, University of Illinois, 1999, pp 94–97.
5. Mull RL: Metaldehyde poisoning, in Kirk RW (ed): *Kirk's Current Veterinary Therapy VIII: Small Animal Practice*. Philadelphia, WB Saunders, 1983, pp 106–107.
6. ASPCA Animal Poison Control Center Case Database: Unpublished data, Urbana, IL, 2003.
7. Plumb DC: *Veterinary Drug Handbook*, ed 4. St. Paul, MN, Pharma Vet Publishing, 2002, pp 530–532. VI