

POISONING BY PESTICIDES

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**THE
MERCK
VETERINARY
MANUAL**

**A HANDBOOK OF DIAGNOSIS, THERAPY, AND
DISEASE PREVENTION AND CONTROL
FOR THE VETERINARIAN**

SIXTH EDITION

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POISONING BY PESTICIDES (SM AN)

Dogs and cats are exposed to many potentially toxic materials that can lead to poisoning. The ingestion of pesticides is a major hazard. Cats, although potentially exposed to the same poisons as dogs, are less frequently affected, possibly because of their selective eating habits. However, when poisonings do occur, signs observed are comparable to those in dogs.

Metalddehyde and N-methyl carbamates have supplanted arsenic, strychnine, and phosphorus as the major causes of poisonings in small animals. Anticoagulants, organophosphorus compounds, and chlorinated hydrocarbons are also significant problems. A variety of other pesticides are also involved to a much lesser extent. Due to the recent reformulation of snail baits so that the inert ingredients are no longer attractive to most dogs, there has been a dramatic decrease in the number of metalddehyde poisonings; however, a significant number of these cases still occur. Some of these are misdiagnosed as strychnine poisoning due to the similarity of signs.

In the rural and suburban areas, organophosphorus compounds are frequently a major cause of poisoning. Many crops are treated with them and dermal contact alone can lead to poisoning.

The N-methyl carbamates (esters of N-methyl carbamic acid) are widely used in home and garden types of insecticides as well as some snail baits. When ingested, the cholinergic signs can be dramatic but respond well to prompt treatment with sufficient amounts of atropine.

Although some of the older scientific literature suggests that oximes are contraindicated for carbamate treatment, recent experimental animal studies suggest that they are only contraindicated for treatment of poisoning by carbaryl (Sevin). If an animal is showing severe cholinergic signs and there is no information as to whether an organophosphorus compound or a carbamate was involved, oximes such as pralidoxime chloride (2-PAM) should be considered for supportive treatment. 2-PAM is given IV to all species at 11-23 mg/lb (25-50 mg/kg) body wt. If organophosphate poisoning is suspected, atropine is also indicated; it may be given IM or subcut., b.i.d. to q.i.d. as necessary. For dogs, the dose is 40-50 µg/lb (90-110 µg/kg); for cats, 20-40 µg/lb.

Cats and dogs are occasionally poisoned by feeding on animals or rodents that have died from pesticide exposure. Most flea collars contain organophosphorus compounds or carbamates and do not normally cause problems. However, young or debilitated dogs or cats may develop signs of cholinesterase inhibition or dermatitis. Solvents may contribute to pesticide poisonings or by themselves may cause the toxic effects. Whenever a pesticide exposure is suspected, the pesticide container should be obtained, since the toxic ingredients are listed on the label, and treatment information for man is required on the labels of the more toxic pesticides.

(See also TABLE OF CONTENTS, TOXICOLOGY, p 1328, and the INDEX for more general discussions of most of the poisons listed below, and for discussions of other toxins of dogs and cats.)

Some pesticides and solvents commonly involved in dog and cat poisoning are listed below with their signs and suggested treatments.

ANTICOAGULANTS

COUMARIN TYPES
(Warfarin, Coumafuryl)

Clinical Findings: Hemorrhage and sudden death with no previous warning. In subacute cases, anemia, weakness, pale mucous membranes, dyspnea, moist rales, bloody feces, and scleral, conjunctival, and intraocular hemorrhage. Staggering, ataxia, blood-tinged froth around mouth and nose, and CNS signs may appear if hemorrhage occurs in the brain or spinal cord.

Treatment: In severe cases, treatment should be initiated promptly, and include 1 or more of sedation or light anesthesia to prevent trauma, oxygen,

citrated whole blood (9 mL/lb [20 mL/kg]) IV, thoracocentesis to remove blood, if present, and vitamin K₁ (0.25-1 mg/lb [0.55-2.0 mg/kg]) IV in dextrose solution. The animal should be kept warm and free from physical trauma for at least 24 hours. Oral vitamin K for 2-6 days is indicated.

1,3 INDANDIONE TYPES (Diphacinone/diphenadione, Pindone)

These tend to be more toxic than the coumarins. The signs are similar to those of coumarin poisoning plus signs of cardiopulmonary and neurologic damage. Treatment is the same as for coumarins (above).

ARSENICALS

Clinical Findings: In general, organic arsenicals are less toxic than the inorganics. **Acute:** Intense abdominal pain, staggering, extreme weakness, trembling, salivation, vomiting, diarrhea, fast and feeble pulse, normal to subnormal temperature, collapse, and death. **Subacute:** Anorexia, depression, watery diarrhea, increased urination followed by anuria, partial paralysis of hind limbs, stupor, subnormal temperature, and eventual death.

Treatment—Acute: Treatment must be administered early: give emetics or gastric lavage only if signs are not yet present; if signs are present, give dimercaprol at the rate of 3 mg/lb (6 mg/kg) body wt IM every 4 hours for first 2 days, then twice a day for the next 10 days. **Subacute:** Supportive therapy is indicated for additional signs such as dehydration and uremia.

CHLORINATED HYDROCARBONS (Organochlorines)

Clinical Findings: Apprehension, hypersensitivity, and continuous to intermittent spasms of the eyelids and front quarters progressing to the hindquarters. Clonic-tonic seizures, loss of coordination, circling, and abnormal posturing also may be seen and the animal may become comatose.

Treatment: Convulsions should be controlled with anesthetics such as chloral hydrate or pentobarbital. If exposure was oral, gastric lavage and saline cathartic should be administered. Diazepam may be successful in controlling violent neuromuscular activity.

GLYPHOSATE ("Roundup", "Kleen-up")

Clinical Findings: This herbicide is of low toxicity, but a few dogs and cats show eye, skin, and upper respiratory tract signs when exposed during or subsequent to an application to weeds or grass. A few cases of nausea, vomiting, staggering, and rear-leg weakness have been seen in dogs and cats that were exposed to fresh chemical on treated foliage.

Treatment: The signs, although of considerable concern to some pet owners, usually disappear when exposure ceases, and minimal symptomatic supportive treatment is needed. Washing the chemical off the skin, evacuating the stomach, and tranquilization are usually all that is needed.

METALDEHYDE

Clinical Findings: Muscle weakness, frothing at the mouth, hypersalivation, incoordination, tachycardia, loss of consciousness, cyanosis, and often, convulsions are seen. Death usually is due to respiratory failure.

Treatment: Emetics, gastric lavage, ventilatory support for respiratory failure, and glucose or calcium gluconate for treatment of possible liver damage are indicated. It is often necessary to anesthetize the animal to control convulsions.

N-METHYL CARBAMATES

Clinical Findings: These include hypersalivation, gastrointestinal hypermotility, abdominal cramping, vomiting, diarrhea, sweating, dyspnea, cyanosis, miosis, muscle fasciculation (in extreme cases, tetany followed by weakness and paralysis), and convulsive seizures. Death is usually a result of hypoxia due to bronchoconstriction.

Treatment: Atropine sulfate, approximately 2 mg for an average dog. 2-PAM (11-23 mg/lb [25-50 mg/kg]) and other oximes are also useful in addition to atropine treatment; however, 2-PAM is contraindicated for treatment of carbaryl poisoning.

ORGANOPHOSPHORUS COMPOUNDS

Clinical Findings: Sialosis, gastrointestinal hypermotility, tenesmus, vomiting, diarrhea, sweating, dyspnea, cyanosis, miosis, muscle fasciculation (in extreme cases, tetany, followed by weakness and paralysis), and convulsive seizures may all be seen. Death is usually a result of hypoxia due to bronchoconstriction.

Treatment: Atropine sulfate (0.25 mg/lb [0.5 mg/kg]), every 4 hours. 2-PAM (11-23 mg/lb [25-50 mg/kg]) is useful to reactivate cholinesterase. Atropine therapy alone will not abolish the skeletal muscular manifestations of poisoning.

PHOSPHORUS

Clinical Findings: The chief signs are abdominal irritation and vomiting; the vomitus may be luminous in the dark and have a garlic-like odor. The animal may appear to recover for a few hours to a few days, only to relapse into vomiting and abdominal pain accompanied by icterus, nervous signs, convulsions, coma, and death from severe hepatic and renal degeneration.

Treatment: Gastric lavage should be followed by oral administration of 0.1-0.2% potassium permanganate, or 0.2% copper sulfate. Cardiac stimulants and 5% glucose IV may be indicated. Avoid contact with gastric washings containing phosphorus since it can burn the skin and eyes.

PYRETHRINS AND PYRETHROIDS

Clinical Findings: The pyrethrins tend to be of low toxicity and, at the most, upper respiratory signs and some wheezing is seen in dogs and cats that have walked through treated foliage or have been in a room during or subsequent to pesticide treatment. Some of the pyrethroids are much more toxic and in the case of significant exposure, besides upper respiratory and pulmonary problems, there can be signs of nervous irritability, tremors, and ataxia.

Treatment: The skin should be washed if it has been exposed, and symptomatic treatment given for allergic reactions and breathing difficulties such as aminophylline, adrenalin, and antihistamines. For significant ingestive exposure, the stomach should be evacuated; for nervous system signs, a tranquilizer is indicated.

PYRIMINIL ("Vacor")

Clinical Findings: Vomiting, gastric pain, chills, nervousness, and weakness are the chief signs. A few animals progress to a state of shock.

Treatment: Apomorphine (0.035 mg/lb [0.077 mg/kg], subcut.) should be used to empty the stomach if ingestion has been recent; gastric lavage is indicated if a few hours have elapsed since ingestion. Nicotinamide is a useful antidote if given within 1 hour after ingestion. Symptomatic treatment may be useful for other developments.

SOLVENTS

Acetone: Gastroenteric irritation, narcosis, and kidney and liver damage are the main signs. Treatment consists of gastric lavage, oxygen, and a low-fat diet. Additional supportive treatment may be given as the signs dictate.

Isopropyl Alcohol: The signs are gastroenteric pain, cramps, vomiting, diarrhea, and CNS depression (dizziness, stupor, coma, death from respiratory paralysis). The liver and kidneys are reversibly affected. Dehydration and pneumonia may occur. Treatment consists of administration of emetics, gastric lavage, milk, CNS stimulants, oxygen, and artificial respiration.

Methanol: Nausea, vomiting, gastric pain, reflex hyperexcitability, opisthotonos, convulsions, fixed pupils, and acute peripheral neuritis are the typical signs. Toxic effects are due in part to the alcohol itself and in part to formic acid produced by its oxidation. Treatment should include emetics (apomorphine) followed by gastric lavage with 4% sodium bicarbonate, administration of saline laxative, oxygen therapy, sodium bicarbonate solution IV, and analgesics, although the prognosis is poor. Intensive and prolonged alkalization is the mainstay of treatment. Ethyl alcohol retards the oxidation of methanol and may be given as an adjunct to alkali therapy.

Petroleum Derivatives: Light mineral oils, xylene, and kerosene causes erythema, drying, cracking, and blistering of the skin. Inappetence, depression, dyspnea, vomiting, diarrhea, salivation, dizziness, coma (sometimes convulsions), trembling, narcosis, and death may be seen. Effects of xylene are noticed within minutes and may last for hours. Signs of exposure to other solvents may appear after a few days or last for several weeks.

These solvents must be removed from the skin with copious quantities of water and soap or detergent. Thereafter, clinical signs are treated symptomatically. Treatment of systemic toxicity should be based on clinical signs. Gastric lavage and laxative may be beneficial for ingestion incidents. There is no effective treatment for aspiration pneumonia induced by lipids.

STRYCHNINE

Clinical Findings: Signs appear 10 minutes to 2 hours after ingestion; they include apprehension, nervousness, tenseness, and stiffness. Violent tetanic spasms may be spontaneous or the result of any external stimulus. Intermittent periods of relaxation may appear but become less and less frequent. Cyanosis, exhaustion, hypoxia, and death follow. If not treated, the entire syndrome may proceed from onset to death in <2 hours. When treated, the strychnine should be metabolized within 24 hours.

Treatment: Apomorphine (0.035 mg/lb [0.077 mg/kg], subcut.) is the emetic of choice for the dog if the animal is not convulsing. Xylazine (0.2 mg/lb [0.44 mg/kg], IM) is an effective, safe emetic for cats. Pentobarbital is indicated for short-term control of seizures; inhalation anesthetic or administration of methocarbamol for longer control; gastric lavage (using potassium permanganate solution, then activated charcoal), and forced diuresis with mannitol should follow. Positive pressure ventilation may be necessary, and the animal should be kept warm and in a quiet area.

ZINC PHOSPHIDE

Clinical Findings: The signs may be similar to those of strychnine poisoning (see above). The onset is rapid and repeated vomiting is an early sign; animals with signs rarely survive >24-48 hours. Signs include anorexia, lethargy, wheezing, vomiting, and abdominal pain; the vomitus often contains dark blood. Ataxia, weakness, recumbency, and terminal hypoxia follow. The odor of phosphine (similar to acetylene) is detectable on the breath and vomitus. If an animal survives for 3 days, the prognosis is favorable.

Treatment: Gastric lavage with sodium bicarbonate is followed by symptomatic and preventive treatment for acidosis and liver damage.

POISONOUS PLANTS OF TEMPERATE NORTH AMERICA

INTRODUCTION

Poisonous plants are among the important causes of economic loss to the livestock industry; therefore, consideration should be given them in evaluating livestock illness and decreased productivity. There are many ways that poisonous plants affect animals: death, chronic illness and debilitation, decreased weight gain, abortion, birth defects, and photosensitization. In addition to these more obvious losses, loss of forage, additional fencing, increased labor and management costs, and frequently, interference with proper harvesting of forage must be considered.

Most poisonous range plants fall into 2 general categories: 1) those that are indigenous to a range and increase with heavy grazing, and 2) those that invade after overgrazing or disturbance of the land. Among those not in these categories are certain locoweeds and larkspurs, both of which are climax plants and form part of the normal range plant community. Therefore, many poisonous plants have a certain niche in most plant communities.

Livestock poisoning by plants often can be traced to problems of management or range condition, or both, rather than simply to the presence of poisonous plants. Usually, animals are poisoned when enticed to eat poisonous plants because of hunger or other conditions that cause them to graze abnormally. Overgrazing, trucking, trailing, corraling, or introducing animals onto a new range tend to induce hunger or change behavior, and poisoning may occur.

Not all poisonous plants are unpalatable, nor are they restricted to overgrazed ranges and pastures. Furthermore, poisonous plants do not always kill or otherwise harm animals when consumed. Plants such as lupine and greasewood may form a beneficial part of the grazing animal's diet and the animal dies only when it consumes too much of the plant too fast.

The definitive diagnosis of suspect plant poisonings is difficult. Most individuals need not concern themselves with large numbers of poisonous plants; they need only be acquainted with those growing in the area where they are involved with livestock, but they should be acutely aware of those plants and the conditions under which livestock may be poisoned. If one knows of: 1) any local soil deficiencies or excesses (which may complicate plant toxicities or simply confuse as to cause of a syndrome), 2) the syndromes associated with each of the poisonous plants in the area, 3) the time of year during which each is most likely to cause problems, and 4) the detailed history of the animal(s) over the last 6-8 months, a tentative diagnosis is possible. Indeed, in some cases, eg, locoism, this may be all that is required in addition to identification of the plant in the area involved. Identification of the plant in question is important, whatever its stage of growth—and is especially useful if it can be identified in the stomach contents of the poisoned animal. Chemical analysis of toxicants often is not useful. Metabolic profiles are useful for some toxicities, and in some, the postmortem lesions are distinctive.