

The physiology of belching and the place of rumen bacteria in bloat are studied at Cornell University and the University of Maryland.

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## Chemical Poisoning

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ARSENIC poisoning in livestock may be caused by arsenic trioxide (white arsenic), paris green, sodium arsenite, and sodium arsenate. Arsenic has been a common cause of accidental and criminal poisoning of animals because it often is used to kill insects, parasites, weeds, and rodents and as a tonic for animals.

Arsenic compounds usually are palatable to animals, especially when they are hungry for salt. Many cattle and sheep have died from eating arsenic-treated plants, grasshopper bait, or discarded arsenic residues. The toxic dose of arsenic depends on the species of animals, the type of arsenical compound, its physical state, and the method of administration.

ARSENIC POISONING may be acute or chronic, depending on the amount consumed.

In acute poisoning, a form frequently encountered, the symptoms may include intense abdominal pain, salivation, a diffuse diarrhea, depression, weakness, incoordination, and posterior paralysis, and a subnormal temperature. These symptoms terminate in convulsions and death within a few hours, or the animals may linger for 3 or 4 days. Some eventually recover.

The chronic form of arsenic poisoning in animals is hard to diagnose because the symptoms may be obscure

and much like the symptoms of other diseases. Some of the common symptoms are loss of flesh; a bright-red coloration of the mucous membranes; digestive disturbance, with slight to marked diarrhea, irregular pulse, and depression.

Animals poisoned with arsenic usually have taken such large amounts that treatment is of no value.

FLUORINE is an active chemical element. It is widely distributed in the soil, rocks, water, and plants. It combines with other elements to form fluorides.

The main naturally occurring fluorine compounds, which are used for commercial purposes, are fluorspar, cryolite, and apatite (rock phosphate).

Chronic fluorosis results from the ingestion of small amounts of fluoride for a long time. It occurs in livestock from the use of mineral mixtures containing rock phosphate, water high in fluorine, and from contaminated forage grown near industrial plants that emit fluorides into the atmosphere. It is possible that surface contamination of growing plants with dust from soil high in fluorides may cause chronic fluorosis in animals subsisting entirely on such forage.

All classes of livestock may be affected by excess amounts of fluorides. Considerable variation exists among

the different species in the levels of intake required to produce symptoms of chronic fluorosis. Cattle are the most susceptible, followed by sheep, swine, horses, and poultry.

The severity of the lesions caused by fluorine poisoning depends on the form of fluoride consumed, the nutritional quality of the feed, age and species of the animals, the level and the period of fluorine intake, and such other factors as reproduction and lactation.

Feeding experiments show that cattle may ingest about 1 to 2 milligrams per kilogram (2.2 pounds) of body weight or approximately 30 to 60 parts per million (p. p. m.) of fluorides in the total ration without harmful effects if they are in good state of nutrition. An excessive amount of fluorine interferes with the normal calcification of the bones and teeth.

The symptoms of chronic dental fluorosis appear during the periods of formation and eruption of the permanent teeth. The incisor teeth lose their ivorylike appearance. They may show definite crossmarkings and become dull and chalklike. Excessive wearing of the teeth, staining, and slight pitting or excessive erosion of the enamel, with exposure of the dentine (which becomes brown or black) occur. In severe cases, the joints may become enlarged and the shaft of the long bones may thicken and show slight roughness or prominent areas of outward enlargement, with intermittent lameness.

If excessive amounts of fluorides are not ingested until after all permanent teeth have erupted (in cattle, at the age of 4 to 5 years), the teeth will not show the characteristic lesions of dental fluorosis, but eventually some animals may show slight thickening of long bones, lameness, and loss of flesh.

Such changes as diarrhea, long toes, and reproductive disturbance are not specific symptoms of chronic fluorosis in cattle and have not been observed to be related to the level of fluorine intake.

Diarrhea, sudden loss of appetite, rapid loss of flesh, slight to marked inflammation of stomach and intestines, and, in some animals, a lowering of the blood calcium are often characteristic of acute fluoride poisoning.

The fluorine content of normal bones varies greatly and may reach a level of 1,200 p. p. m. or more on fat-free, dry basis. A fluorine content of more than 4,500 p. p. m. may occur in bones that show lesions of fluorosis.

I know of no substance that can be given to animals on a high fluorine diet to overcome completely the poisoning effects. Liberal amounts of calcium and phosphorus or aluminum compounds in the feed, however, reduce somewhat the toxic effects. Those compounds combine with the fluorine in the digestive tract and prevent to some extent its accumulation in the bones and teeth.

Prevention of chronic fluorosis depends entirely on eliminating any excessive fluoride intake by selecting feeds and mineral supplements that are low in fluorine.

**LEAD POISONING** occurs oftenest in cattle and calves, but all animals are susceptible. The lead compounds usually involved in livestock poisoning are lead oxide, red oxide of lead, lead acetate, white lead, and lead arsenate.

Animals may get lead by chewing or licking lead-painted objects, painted feeding buckets or troughs, discarded paint containers, lead storage batteries, and discarded painting materials, such as old brushes and drop cloths. Allowing stock to graze in orchards that were sprayed shortly before with lead compounds may be dangerous.

Many lead compounds are described as insoluble or slightly soluble in water, but they may be readily soluble in the digestive juices in the stomach and intestinal tract of animals.

Lead is a cumulative poison in the body, and toxic effects may result when the total amount of small daily doses equals that of a single toxic dose.

Symptoms of acute lead poisoning arc in two groups.

First, inflammation of the lining of the stomach and intestines resulting from the corrosive action of the lead on the mucous membranes, complete loss of appetite, diarrhea with a strong fetid odor, grinding of teeth, and salivation.

Second, symptoms arising from the toxic effect of the lead on the nervous system. Affected animals may walk in circles and run into objects because of blindness. Cattle often bellow, as if terrified, with a marked change in the voice, or they may stand with heads pressed against the wall or manger.

In chronic lead poisoning there may be a loss of weight, depression, staggering gait, and constipation alternating with diarrhea. Horses may show swelling of the knees, blue line on the gums, gradual paralysis of the hind legs, and a peculiar roaring when they inhale.

Epsom salts may be effective as an antidote to change the soluble lead in the stomach and intestines into a less soluble form so as to reduce further absorption of the poison.

**MOLYBDENUM POISONING** may occur in cattle and sheep that graze in areas where molybdenum is present in the soil in excessive amounts and is taken up by forage plants.

Small amounts of the element appear to be beneficial in the growth of nitrogen-fixing bacteria found in the roots of leguminous plants. When excessive amounts are present in the soil, however, alfalfa and the clovers generally take up much greater quantities of molybdenum than the grasses in the same area and arc responsible for most of the poisoning that occurs.

Poisoning has been produced experimentally by feeding calves 2 to 3.5 milligrams per pound of body weight of sodium molybdate. The presence of excess molybdenum in the animal's body apparently interferes with copper metabolism, and the condition produced is sometimes referred to as complicated copper deficiency.

Animals vary considerably in susceptibility to molybdenum poisoning. Young cattle are more susceptible than older animals, and dairy cattle seem to be more susceptible than beef cattle and sheep. Horses and swine appear to be resistant.

When the molybdenum content of forage is approximately 10 p. p. m., it may take 1 to 7 months of continuous feeding to produce the disease, while symptoms of molybdenum poisoning may appear 24 hours to a week after cattle are put on spring pastures, the forage of which contains 20 to 100 p. p. m. of molybdenum.

The clinical symptoms of chronic molybdenum poisoning are profuse diarrhea, with the feces containing gas bubbles, emaciation, swollen vulva, marked anemia, general weakness and stiffness, fading of the hair coat, and occasionally death from severe and prolonged diarrhea.

If the molybdenum content of the ration does not exceed 15 to 20 p. p. m., the diarrhea may be controlled to a considerable extent by using, during the grazing season, a salt mixture containing 0.5 pound to 1 pound of copper sulfate to 100 pounds of salt. The action of the copper sulfate on molybdenum poisoning is not yet definitely known, except that it may relieve the deficiency of copper created by the excess molybdenum.

**NITRATE POISONING** in animals may be produced by a number of plants, some of which are oat hay (mainly the leaves); redroot (*Amaranthus retroflexus*); Russian-thistle (*Salsola kaliternifolia*); white ragweed (*Franseria discolor*); and variegated thistle (*Silybum marianum*). Beet tops also have been found to be high in nitrate after the beets had been sprayed with a very low concentration of 2,4-D.

Nitrate fertilizer is quite palatable, especially to cattle, and may cause nitrate poisoning when animals can get at it while it is in storage.

The experimental feeding of potassium nitrate has shown that the

minimum lethal dose is approximately 25 grams (0.8 ounce) per hundred pounds of animal weight.

The nitrate level in plants depends largely on the amount of nitrate in the soil, although other unidentified factors are present. When the nitrate content exceeds 2-percent dry weight of the plants, such forage is dangerous to feed and may cause poisoning.

While nitrate poisoning results from the ingestion of excess amounts of nitrate, the active toxicant appears to be the highly toxic nitrite that has been formed by the reduction of the nitrate to nitrite probably by bacterial or enzymatic action within the digestive tract of the animal.

When the nitrites are absorbed into the blood stream, they change the oxyhemoglobin to methemoglobin; the red blood cells cannot then carry oxygen to the tissues.

Cattle, sheep, and horses are susceptible, but poisoning occurs most frequently in cattle.

The symptoms of nitrate poisoning include staggering gait, muscular tremors, blue coloration of mucous membranes, rapid and labored breathing, marked dilation of pupils, and coma. Death results from asphyxiation.

The object in treatment is to convert the methemoglobin in the blood back to oxyhemoglobin so the red blood cells can release oxygen to the tissues.

The injection intravenously of 4 milligrams of methylene blue per pound of body weight, given in a 4-percent solution with distilled water, is effective if given early and the methemoglobinemia is not greater than 70 percent. Repeated injections of this solution may be necessary until the material containing the nitrate has been eliminated from the digestive tract. The administration of a large volume of mineral oil through a stomach tube may help reduce the absorption of the nitrites and aid in elimination.

SALT POISONING may occur in livestock from the excessive consumption

of sodium chloride, or common salt, which is an essential food substance and harmless when properly used.

The normal consumption of salt by cattle on range and pasture is 1 to 2.5 pounds a month. For sheep the amount generally is estimated on the basis of 0.5 pound a month per ewe. Lambs in dry lots may consume about 0.6 pound of salt a month. Salt always should be available to livestock.

Cattle may consume up to 1 pound of salt daily without harmful effects if fresh water is available at all times. Natural waters that contain up to 1 percent of salt generally are considered satisfactory as a water supply for cattle.

Range cattlemen have been using salt in a mixture with feed concentrates in an effort to control the intake of protein or grain supplements that are fed from a self-feeder. This type of feeding usually has been satisfactory, although the amount of salt consumed by the animals greatly exceeds their requirements. Losses may occur with this system of feeding on ranges where the water supply is limited or when the salt and protein supplement mixtures are fed at too great a distance from the water supply.

Salt poisoning has occurred when animals were deprived of salt for a long time and then allowed free access to an abundant supply.

Hogs have been poisoned by drinking brine or by eating garbage that contained brine pickle.

Symptoms first noticed in salt poisoning are hypersensitivity to touch, loss of appetite, marked redness and dryness of mucous membranes of the mouth, and loss of coordination. Paralysis and death may follow.

The autopsy usually shows a distention of the rumen, slight congestion of the mucous membrane of the stomach, and marked edema along the lining of the first portion of the small intestines.

To prevent salt poisoning, it is important to give animals enough salt and a liberal supply of water if salt-protein supplements are used.

Treatment of salt poisoning usually

is ineffective, but sometimes in the early stages it helps to give a large volume of water through a stomach tube, and after that a large dose of mineral oil.

SELENIUM POISONING in animals is caused by the ingestion of plants that have absorbed selenium from the soils. Many areas of seleniferous soils that were derived from cretaceous formations have been found in the Western States, principally in the region from the Rocky Mountains to South Dakota and Kansas.

Chronic selenium poisoning results when an animal ingests feed containing low levels of selenium (10 to 40 p. p. m.) for a few weeks to several months.

Alkali disease was the name usually applied to chronic selenium poisoning before the cause was recognized. Symptoms of selenium poisoning are loss of hair from the mane and tail of horses, from the switch of cattle, and from the body of hogs. Rough horns, long and deformed hoofs, and later sloughing of the hoofs occur in all affected animals.

Affected animals may be sterile or may be slow breeders.

The hatchability of chicken eggs is greatly reduced. The chicks that are hatched have a wiry, greasy down; they are weak and live only a short time. Unhatched eggs usually contain deformed embryos.

Acute selenium poisoning may cause death within a few hours to 2 or 3 days if the animals have ingested forage containing 200 p. p. m. or more of selenium.

The only practical means of preventing selenium poisoning in livestock is to avoid the use of seleniferous forage and grain. The feeding of a ration high in protein is reported to give some protection to sheep and laboratory animals against the toxic effects of selenium.

WAYNE BINNS in 1954 joined the Department of Agriculture as a veterinarian in charge of studies of livestock poisoning by plants.

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