It is important that the animals be handled as little and as quietly as possible for a day or two before shipment, during shipping, and for at least 2 weeks after shipment. Above all, no operations, such as dehorning or castrating, should be done then.

After shipment, range cattle should be allowed to rest quietly where they have access to a good windbreak and a dry place to lie. Well-bedded, open sheds are preferred. Serious losses have occurred after cattle have been crowded into closed buildings overnight or during a storm. If any sick animals are present, the others have little chance of avoiding the infection.

For the first few days, the feed should be similar to what the animals have been eating. A nutritious ration to sustain the resistance of the animals is essential, but any changes, particularly to richer feed, should be made gradually. A little grain, preferably oats, may be fed but no animal should get more than its share. The amount then can be increased gradually. Large feeding establishments control grain feeding effectively by having it thoroughly mixed with chopped roughage.

Recently shipped cattle should be kept apart from other cattle, particularly from common watering tanks. The tanks should be cleaned often to reduce the possible spread of infections. At the first signs of illness the herd should be inspected 2 or 3 times daily. Sick animals should be isolated in dry, draftless quarters, where they will be undisturbed except when being treated. They should be reexamined and possibly re-treated at least once daily. When they recover, their feed should be increased gradually to avoid a digestive upset when they are returned to the feed lot.

Animals that die should be removed promptly and before they are opened for postmortem examination.

The stables, feed bunks, and tanks that may have been contaminated should be thoroughly cleaned and disinfected as soon as the animals recover or are removed, and should then be exposed to the air and sun as much as possible until they are used again.

To avoid losses from shipping fever in cattle, the chief reliance should be centered on buying healthy animals, avoiding infected yards and vehicles, preventing stress factors, and providing prompt treatment for the herd at the first sign of infection.

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Grass Tetany and Wheat-Pasture Poisoning

R. E. Davis and H. R. Crookshank

IN THE WINTER wheat grazing area, particularly the Texas and Oklahoma Panhandles, a condition known locally as wheat-pasture poisoning sometimes develops when cattle graze on the growing wheat. The condition is worse when growth is lush and moisture is plentiful.

The first symptoms are unusual excitement, incoordination, and loss of
Grass Tetany and Wheat-Pasture Poisoning

appetite. Viciousness, staggering, and falling come later. Nervousness becomes more apparent with muscular twitching, particularly in the extremities. The animal may grind its teeth and salivate profusely. The third eyelid will protrude or flicker, as in tetanus.

General tetanic contractions of the muscles follow until the animal nears a state of prostration. A sudden noise—or merely touching the animal—causes a reflex response.

**The Next Symptoms** are a labored breathing and pounding of the heart. A comatose condition follows. Convulsions, with periods of relaxation, occur before the animal dies. The animal usually becomes comatose 6 to 10 hours or so after the onset of the initial symptoms.

The chance of recovery is slight if treatment is not started before coma.

The symptoms are almost identical with those of highly fatal grass tetany, which occurs in a number of States and usually occurs when young, fast-growing pasture grass is grazed.

Because grass tetany and wheat-pasture poisoning appear to be the same except for the type of pasture, we consider them together.

The most commonly used treatment is the intravenous or intraperitoneal injections of a solution of calcium gluconate. Several solutions have been used experimentally, but calcium gluconate, with or without fortification, seems to be best. The solution should contain not less than 17 percent of calcium gluconate. It may be fortified with magnesium or phosphorus or both. Calcium gluconate solutions with added magnesium and phosphorus seem to speed recovery and lessen the need for a second injection. Some preparations of calcium gluconate also contain glucose (dextrose), but the glucose does not appear to have any appreciable effect on treatment or recovery.

The dosage, whether given intravenously or intraperitoneally, should be 500 milliliters of calcium gluconate. Intravenous injection should be given slowly. At least 20 to 30 minutes should be allowed for treatment. Intraperitoneal injection may be given more rapidly.

If treatment is given during the first few hours after the development of the symptoms, recovery is usually fairly rapid and uneventful. The chance of recovery is slight if treatment is delayed 8 to 12 hours. No type of treatment has been found that will be successful in cases when treatment is delayed. The removal of the cow from wheat pasture for a few days appears to speed complete recovery. It is unusual for an animal to have a second attack after recovery from the initial attack.

Sixty cases of wheat-pasture poisoning were studied at the Pan Tech Field Laboratory near Amarillo, Tex. None of the cases was complicated by other conditions, and it was possible to obtain a complete history of each case.

All were cows at least 2 years old and pregnant or with a calf at the side. The time of the attacks varied from the fifth month of pregnancy to 6 or 7 months after calving. The length of time on wheat varied from 1 week to 6 months. Most cases occurred between 60 and 150 days on winter-wheat pasture. Seventy-nine percent of the cases occurred in cows with a calf under 60 days of age. In 17 percent of the cases, the disease occurred within 1 week after calving; in 24 percent, between 1 and 2 weeks; in 11 percent, between 15 and 29 days; and in 27 percent, between 30 and 60 days. Seven percent of the cases occurred during the last 3 months of gestation. The remaining 14 percent had calves more than 60 days old.

Many reported cases of wheat-pasture poisoning in calves, steers, and dry cows were investigated, but the only cases observed occurred under the conditions we outlined. Beef, dairy, and crossbred cows have had the disease.

The use of salt, minerals, cottonseed meal, dry feeds, silage, or various combinations of these supplements has been
suggested as a preventive measure. An analysis of 41 cases of poisoning in the Panhandle region during the 1953-1954 grazing season, however, showed that supplements or combinations of them had little effect on the incidence of the disease.

Only 3 of the 41 cows had not received supplemental feed. All the others had received salt, mineral mixtures, cottonseed meal, dry feed, or silage, or a combination of them. Included among the dry feeds were sorghum bundles, grain, hay, and stalks. The diluting effect of some of the supplements apparently delayed the onset of the disease but did not prevent it.

The analysis disclosed also that a number of other maladies have been confused with wheat-pasture poisoning. Many investigations were made of what were thought to be cases in calves, steers, and dry cows. The most common condition confused with wheat-pasture poisoning is spinose ear tick infestation, in which there was incoordination much like that in wheat-pasture poisoning.

Black disease, or Clostridium novyi infection, supposedly confined to sheep, has accounted for many deaths of both yearling and mature cattle that grazed wheat. The diagnosis was confirmed by bacterial cultures and inoculation.

Other conditions that have been mistaken for wheat-pasture poisoning are: Acetonemia in mature cows, prussic acid poisoning from feeding bundles or grazing nearby stalk fields, pneumonia and shipping fever, milk fever in dairy breeds of cattle, "water belly" (urinary calculi) in young steers, and contagious abortion in cows—all of which were grazing wheat. These conditions and others often are confused with wheat-pasture poisoning when sickness and losses occur during the grazing season.

The chemical composition of the serums from afflicted cows was compared with that of cows that were grazing wheat but did not show any symptoms of wheat-pasture poisoning. The total calcium level was depressed well below the normal level, and the diffusible calcium dropped to near the tetany level, which is about 2.5 milligrams per 100 milliliters of serum. The magnesium level was depressed markedly. The level of potassium was increased slightly. The total serum protein level was raised. This increase was due to an increase in the globulin fraction. Hence, albumin globulin ratio was decreased. The values found in this study were in general agreement with those of other workers.

Research workers in the Netherlands have suggested that the cause of grass tetany in dairy cows is intestinal auto-intoxication. There is a similarity of the symptoms of grass tetany to the condition produced by administration of histamine; that is, intestinal disorder, acetonemia and hypoglycemia, dehydration, disturbances in the mineral and acid-base balance, hypomagnesemia, hypocalcemia, and frequently hypophosphatemia.

It is estimated that grass tetany affects 1 out of every 100 dairy cows in the Netherlands and that 1 of every 7 affected cows succumbs. In most cases, the dietary intake of magnesium is sufficient. Most cases occur within 1 or 2 weeks after the animals are put out to pasture. It requires more than 2 weeks on magnesium-deficient rations to reduce the absolute values of blood magnesium in dairy cows.

There appears to be a definite difference in time required for the development of symptoms of grass tetany and wheat-pasture poisoning.

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