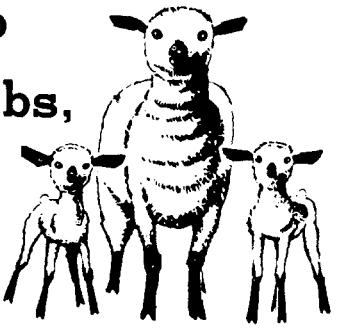
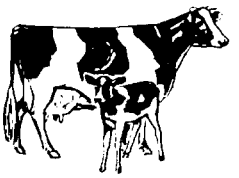
CIRCULAR V-378 Rev. DECEMBER 1975

Enterotoxemia

of sheep and lambs,





cattle and calves

I. A. SCHIPPER
Professor of Veterinary Science
Agricultural Experiment Station

Cooperative Extension Service
North Dakota State University
Fargo, North Dakota / 58102

ENTEROTOXEMIA is a frequent killer of feeder lambs and is often a chronic or acute problem in feeder calves. The cause is a Clostridium bacteria that is a close relative to the organisms causing lockjaw, blackleg and malignant edema. It is often observed in feeder calves as a diarrhea, bloat or lameness. Enterotoxemia could be mistakenly diagnosed as Vitamin A deficiency, listeriosis, coccidiosis, nitrate or chemical poisoning or simple scours on the basis of clinical symptoms.

Enterotoxemia is often referred to by other names, such as over-eating disease, pulpy kidney, struck, milk colic and apoplexy. The disease is caused by a toxin (poison) produced by the Clostridium perfringens organism which is a normal inhabitant of the lower intestinal tract of most animals and present in some soil and forages. The group of organisms produces 12 different toxins, and at least half of these are known to cause disease in animals. One of these organisms produces a toxin designated as the D type while another produces the C type toxin. The disease is a result of the action of the toxin. The C type is usually a cause of enterotoxemia in cattle while the D type is usually a cause of the disease in sheep. However, either type of toxin may cause enterotoxemia in either lambs or calves.

Though the organisms causing enterotoxemia are normal intestinal inhabitants of most livestock or frequent residents of the animal's surroundings, they only become a problem when some change in management or other predisposing factors bring about an overproduction of the organisms. These predisposing factors can in general be classified as over consumption of food, hence the term over-eating disease. Some of the predisposing factors or initiating causes include over feeding of concentrates, placing the animal on concentrates too rapidly, drastic changes in feeding procedures, lush pastures, over supply of milk by the dam or drastic changes in weather. It is usually a problem of the most rapidly growing animal. Inclement weather may be the cause of less exercise while the animal continues to nurse or consume a high concentrate diet. These organisms have also been associated with the scour problem in newborn calves.

It has been theorized that when there is an overproduction of Cl. perfringens in the digestive tract there is an increase in fermentation of grain to lactic acid and a change of the normal pH of the rumen to an acidic condition. Some of the partially fermented rumen contents may pass into the small intestine causing an increase in multiplication of the Cl. perfringens organism, decreased motility of the rumen and small intestine and an increased toxin absorption.

What animals may be affected?

Calves nursing high-producing dams or feeder calves receiving a high concentrate ration may often be affected. A good quality single lamb nursing a good milk-producing ewe is very susceptible to enterotoxemia. Feedlot lambs, like feeder calves, are often involved. Range lambs suddenly placed on luxuriant pasture or unlimited succulent forage will often exhibit enterotoxemia.

The enterotoxemia organisms may also affect the young swine. The symptoms observed may include any one or all of the following: vomiting, bloody diarrhea, paralysis and death. The condition in swine is most often caused by the organism producing the C type toxin.

What are the signs of enterotoxemia?

Frequently the only symptom in lambs or calves is sudden death. Less acute cases may have severe diarrhea, followed by convulsion and death. Often sick animals will hold the head back over their neck, walk in circles and drool previous to the signs of convulsions. enterotoxemia is in a chronic form there will be intermittent bloat, lameness, diarrhea staggering. The chronic form is often observe in feedlot calves.

Can enterotoxemia be prevented?

The scour problem in calves associated with Cl. perfringens can be prevented by vaccinating cows twice initially at intervals of one month with the last vaccination made not later than one month before calving. The vaccine employed should be a C and D type Cl. perfringen toxoid. Immunity can be maintained in the mother by annual vaccination one month before calving.

Prevention of enterotoxemia death in calves and lambs can also be achieved by this vaccination procedure. Protection will be provided to the offspring for approximately four weeks

Prevention for enterotoxemia in feedlot animals is best accomplished by vaccination with the C and D type toxoid at least two weeks before the animals are started on high concentrate feeding. The feeding of wide-spectrum antibiotics such as Terramycin or Aureomycin may also be helpful in prevention or treatment

All animals regardless of age should be vaccinated with both the C and D types of vaccine to provide maximum protection.

Treatment

Nursing calves or lambs can be provided with immediate protection that will last for approximately two weeks by the administration of C and D types of Clostridium perfringens anti-

toxins (antiserum). The antitoxin may be given orally and intramuscularly as a treatment.

If enterotoxemia occurs in lambs or calves on full feed, all concentrates should be withheld immediately upon diagnosis and the animals maintained on forage and mineral supplement only. The most important aspect of treatment is to drastically restrict the consumption of concentrates or luxuriant green forage. The animals should then be vaccinated with toxoid or bacterin (both C and D types) and feeding of concentrates or luxuriant green forage should not be started for 10 to 14 days or until immunity has had time to develop.

It is far better to prevent enterotoxemia by vaccination of the dam or of the feedlot animal before placing them on concentrates than to resort to treatment.

How can enterotoxemia be diagnosed?

The diagnosis of enterotoxemia cannot be made on the evidence of the symptoms alone. Finding the typical organism in the intestinal contents is also insufficient proof that the animal has died from enterotoxemia. To establish a diagnosis, the contents of the small intestine must be collected within six hours after death. Freeze this specimen immediately and get it to the laboratory as soon as possible, as the toxins are inactivated if not frozen within six hours. If the specimen cannot be frozen, add one drop of chloroform to 10 ml of small intestine ingesta to prevent loss of toxins.

The fluid of this specimen is filtered out and injected into two groups of laboratory animals. One group is protected with the antitoxin of the suspected toxin and the other is not. The protected animals live, while the unprotected die within four hours if the toxin is present. Both groups live if no toxin is present. Obviously, if the samples have been collected improperly and the toxin has been destroyed, the laboratory test will be of no value.

Cooperative Extension Service, North Dakota State University of Agriculture and Applied Science, and U. S. Department of Agriculture cooperating. Myron D. Johnsrud, Director, Fargo, North Dakota. Distributed in furtherance of the Acts of Congress of May 8 and June 30, 1914. We offer our programs and facilities to all people without regard to race, creed, color, sex, or national origin.

5M-12/75