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
and D types of Coordinative Perturbations and
may occur up to 2 weeks after the administration of C
immediate postexposure and will last for approx.
Nursing calves of dams can be provided with

What are the signs of enterotoxaemia?

The organism in young calves is most commonly detected by
vomiting followed by diarrhea and death.

The sympotms caused by enterotoxaemia may also be visible
at the level of enterotoxaemia.

Can enterotoxaemia be prevented?

In feedlot calves, the post-exposure form is often observed.
Although the organism is in a chronic form there is
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Although the organism is in a chronic form there is
in enterotoxaemia is a chronic form which will
have severe diarrhea, followed by
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. Ob-

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