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CLOSTRIDIAL DISEASE

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Many of the most serious disease problems of cattle, sheep, swine and sometimes poultry are caused by clostridial bacteria. This group of organisms have common characteristics that place them in a class by themselves.

These characteristics include the ability to produce potent toxins (poisons) that cause the signs of disease. Clostridial bacteria form highly resistant spores and can exist for extended periods in soils, vegetation, dead or decaying animal tissue, and in areas where livestock are kept. Another important characteristic of this group is their ability to grow readily in the absence of oxygen; hence, most of them are frequently associated with deep wounds that are contaminated with soil or manure. They are considered natural inhabitants of the soil and intestinal tracts of animals and man. These disease organisms are not generally considered contagious. Vaccines made from these organisms provide excellent protection from infection.

BOTULISM (FOOD POISONING)

The Clostridium botulinum organism produces a potent toxin in foodstuffs that might be consumed by humans, birds and sometimes animals. The form of food poisoning is not an infection, but due to toxins produced before it is consumed.

Though primarily a disease of man, it can also affect poultry (limber neck) and livestock. Wild ducks may be affected by consuming decaying vegetation in alkaline water.

The signs are primarily those associated with the nervous system including a progressive paralysis.

TETANUS (LOCKJAW)

The signs of tetanus result from the absorption of a neurotoxin produced by the Clostridium tetani organism. This organism is most often found in manure-laden soil, particularly where horses have been kept. Nearly all animals are susceptible to this disease, but horses are most susceptible. It gains entrance to the animal body through wounds, particularly if they are deep puncture wounds contaminated with dirt and containing dead tissue. Infection may result from castration, particularly with rubber bands, and also from navel infection and eartag wounds.

Symptoms may be observed one to three weeks following infection, but in some instances may not be obvious for as long as four months. A general stiffness is noted approximately one day later. The symptoms include muscle spasms, particularly upon touching the animal or following a sharp noise. Inability to chew (lockjaw) and to walk are other signs. The head may be pulled back over the back. The involved horse will have erect ears, extended stiff tail, dilated nostrils and dropping of the third eyelid. The animal may sweat profusely.

Tetanus can be prevented by vaccination. In that horses are particularly susceptible, they should be vaccinated before or at the time of any surgery. Cleaning fresh wounds and providing good wound drainage will aid in preventing this problem.

Tetanus toxoid will provide active immunity by 14 days after vaccination. If the animal should be subjected to wound following vaccination, a booster administration of toxoid may be given. If the animal has not been vaccinated previous to the injury, teta-

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nus antitoxin can be administered to provide immediate protection.

MALIGNANT EDEMA AND BLACKLEG

Malignant edema may be observed in cattle, sheep, horses, swine and man (gas gangrene). The primary signs are lameness, and swelling at the sight of injury. Decreased appetites and elevated temperatures are other signs. It is caused by Cl. septicum.

Blackleg is primarily a problem in cattle and sheep and is caused by Cl. chauvoei. The organism is found in the soil and the intestinal tract of ruminant animals. Most infections result from wounds caused by husbandry procedures such as castrating, dehorning, shearing, and dipping or by mouth. Blackleg is usually a problem of younger animals and the signs include lameness, depression, fever, and sudden death. Painful swellings at the hip, shoulder and neck are other signs associated with this infection.

Blackleg and malignant edema may appear very similar and are difficult to diagnose other than by detailed laboratory bacteriological investigations. Formalized whole culture vaccine is most effective in prevention and should be administered to calves at approximately three months of age. Re-vaccination may be beneficial when the calves are older.

ENTEROTOXEMIA (OVEREATING DISEASE)

Type B

Type B enterotoxemia intoxication results in death of young lambs and foals. This problem is rarely observed in the United States.

Type C

Type C enterotoxemia (struck, hemorrhagic enterotoxemia) is a highly fatal intoxication of suckling calves, lambs or pigs. The most vigorous animals under two weeks of age that are nursing high-producing dams are usually involved.

The involved animal will become listless and cease to nurse and exhibit signs of colic, including kicking at the abdomen and straining. Some animals may die with no signs of illness. Bloody diarrhea may be observed.

The C type enterotoxemia can best be controlled by vaccination of the dams with C type toxoid. Initially two administrations should be given with the last administration approximately one month before parturition. Booster vaccination should be given annually to maintain immunity. Antitoxin is effective for prevention of enterotoxemia in the new born of unvaccinated dams.

Type D

The type D form of enterotoxemia is often referred to as overeating disease or pulpy kidney disease. It frequently occurs in sheep and sometimes in cattle of any age. It is most frequently a problem in the nursing animal, feed lot animals on high concentrate rations, or animals placed in luxuriant pastures.

Like other forms of enterotoxemia, this type is caused by the type D toxin produced by the Cl. perfringens organism. This organism is frequently present in the digestive tract, but the toxic condition is stimulated by predisposing factors, the most common of which is consumption of excessive quantities of feed or milk. In young lambs it is usually a problem in a single lamb nursing a ewe that has an excellent milk supply.

Sudden death with no previous signs is the primary sign associated with the type D enterotoxemia. In some instances other signs may be observed previous to death including incoordination, circling, excitement, pushing against objects, head pulled over back and convulsions.

Control must be based on age and type of environment. Prevention of type D enterotoxemia in nursing animals can best be achieved by vaccinating the dams twice initially with the D type toxoid. There should be a waiting period of at least three weeks between vaccinations, and the last vaccination should be made no less than three weeks before parturition. Prevention in feeder animals can best be achieved by vaccination with D type toxoid at least two weeks before they are placed on a full ration of concentrates.

Outbreaks can best be controlled in nursing animals by the administration of type D antitoxin. Outbreaks in feeder animals can be controlled by reducing concentrate consumption by at least 75 per cent, increasing roughage and vaccinating with a D type toxoid. Increased concentrate consumption should

not begin for at least seven days following vaccination. Wide-spectrum antibiotics in the feed will also aid in preventing enterotoxemia.

Vaccines should be administered in areas on the body that is not a choice cut of meat. Cold abscesses may form following vaccination, and their presence will lower the value of the animal.

Most livestockmen administer both the C and D type toxoid simultaneously. This provides a greater spectrum of protection at a minimum of cost and effort.

BACILLARY HEMOGLOBINURIA

This disease is caused by the Cl. hemolyticum organism and is also known as redwater disease or infectious hemoglobinuria. It is primarily a disease of cattle but has been described in sheep and is often associated with liver fluke infection.

This clostridium organism is primarily found in the animal body and may survive for extended periods in contaminated soil or bones of carcasses of infested animals. Regardless of the source of infection, it ultimately becomes located in the liver as latent spores.

The signs observed include loss of appetite, decreased milk production, decreased rumen and bowel activity and labored breathing. Diarrhea, with blood stained feces, may develop. The urine is dark red and foamy. There is rapid dehydration and anemia develops soon after the first signs are observed. The body temperature is elevated but will become subnormal just before death. If recovery occurs the animal usually becomes a carrier.

Prevention can be achieved for approximately six months by vaccinating with a Cl. hemolyticum bacterin. Treatment involves the use of penicillin, wide-spectrum antibiotics or fluid therapy.

INFECTIOUS NECROTIC HEPATITIS

This disease is caused by the Cl. novyi and is also referred to as black disease. It is primarily a disease of sheep but has also been observed in cattle. This organism multiplies in areas of necrosis and is frequently associated with liver damage caused by the liver fluke.

The source of infection is carrier animals and contaminated soil, water or food.

The signs of this clostridial disease is usually a sudden death with no preliminary signs. Other signs include lagging behind the rest of the flock and going down before death.

Control consists of vaccination with a bacterin-toxoid and liver fluke control.

CLOSTRIDIUM SORDELLI INFECTION

This Clostridium species like other clostridia is found in soil and animal digestive tracts. It may enter the animal body by intestinal wall penetration or through skin breaks.

The signs of this disease usually are very similar to those observed for Cl. septicum or Cl. novyi. Death is usually sudden.

Prevention has been achieved by the use of a bacterin-toxoid vaccine.

GENERAL PROPHYLAXIS

Good management and sanitation will provide protection from the clostridial diseases. The wide use of the many available clostridial bacterins, toxoids or antitoxins has provided excellent protection against these diseases. Recently a new combination of bacterin-toxoid has been made available. This vaccine provides protection to the Cl. chauvoei (Blackleg), Cl. septicum (Malignant edema), Cl. novyi (Black disease), Cl. sordelli and Cl. perfringens types B, C, and D.

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