Managing Clostridial Diseases in Cattle

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Introduction

The many diseases of cattle that are attributed to *Clostridial* bacteria are shown in the following table.

***Clostridial* type**	**Disease**
C. tetani	Lock jaw, spastic paralysis
***C. novyi* type B**	Black disease, malignant edema, gas gangrene
***C. perfringens* type A**	Jejunal hemorrhage syndrome, abomasal ulcers and tympany, gas gangrene, sudden death
***C. perfringens* type C**	Necrotic enteritis
***C. perfringens* type D**	Enterotoxemia
C. septicum	Malignant edema, gas gangrene, enterotoxemia
C. chauvoei	Blackleg, black quarter, malignant edema
C. sordellii	Enterotoxemia (sudden death syndrome), malignant edema
C. hemolyticum	Red water disease

Clostridial organisms are, for the most part, normal flora of cattle and only become problematic with dietary stress, injury, changes in management, parasitism or other unusual circumstances that set up a favorable growth environment and result in production of potent toxins. While some of the diseases rarely, if ever, occur in the US, most others occur sporadically in herds. In general, clostridial diseases carry a very poor prognosis and the first sign of illness may be death. Because treatment success is rare, emphasis is properly placed on preventive measures. Vaccines enjoy widespread use within the dairy industry and can be an efficient way to reduce losses due to these bacteria. A single vaccination with most Clostridial vaccines does not provide adequate levels of protection and must be followed within a period of 3 to 6 weeks by a booster dose. Young calf vaccination does not yield adequate protective immunity for at least 1 to 2 months so most vaccination strategies target the pregnant cow so that maximum immunity is imparted to the calf in colostrum. Inactivated commercial vaccines, which contain 2, 4, 7 or 8-way combinations of clostridial organisms, should be appropriately timed to provide maximum protection at the age of susceptibility.

The focus of this paper will be clostridial enteric disease as these are the most important disease syndromes in cattle. All types of *C. perfringens* have been incriminated in enteric

syndromes affecting both calves and cows. As a group, *C. perfringens* also cause disease in humans and other animals, including pigs, poultry and lambs. Although the different types of *C. perfringens* (types A through E) have different toxin profiles and can cause different disease syndromes, there is not always a clear-cut distinction amongst them and there can be considerable overlap amongst the clinical signs and the risk factors associated with the diseases that the different types of *C. perfringens* produce.

Clostridium perfringens occurs widely in the environment and in the gastrointestinal (GI) tract of most mammals. Type A is routinely isolated from soil and clinically normal animals. While it is unusual to find *C. perfringens* type A in corn silage and high moisture corn, the organism has been found in haylage under some storage conditions. Types C and D are only rarely isolated from soil but they can be isolated from asymptomatic animals, particularly those that demonstrate an immune response in their blood. Clostridial diseases are not spread from animal to animal. Susceptible animals are those that have the organism and have one or more risk factors that are identified below. Management is critical factor in control of clostridial diseases. These bacteria proliferate after death, often to the exclusion of other normal flora, and can invade tissues beyond the gut. Thus, isolation of the bacteria in a post mortem sample is not sufficient basis for a diagnosis.

Clostridium perfringens **in adult cattle**

In the last few years, producers and their veterinarians have recognized with increasing frequency, a syndrome in adult cattle that is referred to as Hemorrhagic Bowel, Bloody Gut or Jejunal Hemorrhage Syndrome (JHS). While no specific cause has been elucidated, *Clostridium perfringens* type A is believed to play some role in the syndrome based on recovery of large numbers of this organism from most (but not all) cases of JHS. The disease syndrome is characterized by a sudden onset, when affected cows are unexpectedly off-feed, produce little or no milk, have a painful or distended abdomen, hemorrhage into their intestine, make very little manure and can die acutely despite medical and/or surgical intervention. Luckily this disease is usually sporadic and involves individual animals on individual farms, but multiple deaths in a short period of time have been common in some herds. High producing cows appear to be at a greater risk than late lactation or lower producing cattle. The risk factors that have been identified with JHS of cattle are summarized below:

- High production
- < 100 days in milk
- Aggressive eaters
- Second lactation or greater cows
- Recent feed change
- Feeding TMR and selection for smaller particles or decreased long stem fiber
- Rumen acidosis

- Excessive rumen fill with spillover into the intestine of contents with high soluble protein and carbohydrate levels
- Feeding corn silage ensiled < 1 week
- Decreased intestinal motility

The prognosis for affected cows with JHS is grave, even with aggressive medical and surgical therapy. Post mortem examination of cows with JHS showed severe hemorrhage into the intestine, necrosis of the bowel wall and blood clots within the lumen of the involved segments. In many cases, the blood clot caused obstruction of the bowel. *Clostridium perfringens* type A has been isolated from feces in the majority (but not all of) the cases.

***Clostridium perfringens* in calves**

In calves, a sudden onset of abdominal distension with pain, depression, feed refusal and sudden death have associated with abomasal ulcers, inflammation and gas in the wall of the stomach. While there can be more than one cause for this syndrome, researchers have isolated *Clostridium perfringens* type A from affected calves and reproduced the disease by placing the organism in the rumen of susceptible calves. Post mortem examination shows inflammation of the lining of the rumen and abomasums, with ulceration and hemorrhage.

Clostridium perfringens type C causes necrotic enteritis in newborn calves. Affected calves may die before they develop diarrhea. Calves are suddenly depressed, weak, may be distended or show abdominal pain. If diarrhea develops, it may have blood and tissue streaks. Intensive care with antitoxin, fluids, antibiotics and anti-inflammatory drugs is necessary but frequently unsuccessful. As described, these signs are not specific for *Clostridium* and other causes such as salmonellosis, coccidiosis, and unusual forms of *E. coli* should be considered. Post mortem examination shows small intestine necrosis and hemorrhage.

Clostridium perfringens type D produces the classic overeating disease, a syndrome more important in lambs than in calves. The disease is characterized by sudden death in thrifty, well-fed calves. Other affected calves may be neurologic, uncoordinated, trembling, recumbent with head back or convulsing. Other diseases such as septicemia, polioencephalomalacia, lasalocid overdose, salt poisoning and *E. coli* can produce similar signs. The post mortem examination will reveal pulpy kidneys and brain edema and glucose in the urine.

Possible risk factors for calves

- Ingestion of *C. perfringens* in the first few days of colostrum feeding
- Ingestion of protein-rich diet in a protease-deficient intestinal tract allows rapid growth of *C. perfringens* organisms.

- Protein-rich milk replacers and/or high grain consumption may be risk factors
- Inconsistent feeding practices – feed changes, temperature, mixing, frequency, volume
- Limited access to water after feed consumption
- Abnormal intestinal flora from abundant oral medications
- Stressful interventions that result in erratic intakes

Sample submission from cows or calves with suspected Clostridial disease

- Intestinal contents
- Blood clots
- Intestinal lining placed in formalin
- Liver
- Blood or serum
- Feces

Samples for culture (intestinal contents, blood clots, liver, and feces) should be placed in clean zip-lock bags and refrigerated or frozen until they can be shipped. Note well: *C. perfringens* is a common occurrence in the intestine of normal animals, there is a tendency for rapid bowel overgrowth and systemic invasion of tissues after death so rapid harvest and appropriate preservation of tissues prior to submission is essential. Combined findings of organism, toxin and compatible history and lesions are necessary to confirm a diagnosis.

Vaccination

Currently, no vaccine approved for use in cattle in the US contains *C. perfringens* type A or *C. perfringens* type A toxin thought to be important in the disease syndrome. Vaccines containing *C. perfringens* type C and D are not likely to protect cattle against JHS but producers routinely vaccinating with *C. perfringens* type C and D toxoid 2 to 3 times a year claim some benefit. *C. perfringens* type A autogenous bacterins are being used in many herds with JHS but it is unlikely that a bacterin without toxoid will prevent the disease. *C. perfringens* type C and D antiserum products probably do not contain the appropriate toxin antibodies to aid in treatment of JHS affected animals but are considered to be the treatment of choice for calves with signs suggestive of clostridial disease.

Vaccination strategies should take into consideration the necessity for a booster vaccination approximately 4 weeks after the first vaccination. Dry cow vaccination with good colostrum feeding is the best way to prevent clostridial diseases in calves. Vaccination of 4 to 6 month old heifers with *Clostridium perfringens*-containing bacterin/toxoids can produce a sustained immune response that can be boostered annually or semi-annually.

Control of clostridial enteritis syndromes

The hallmarks of intestinal disease caused by *Clostridium perfringens* are:

1. The presence of the organism in the normal gut or recent acquisition from feed or the environment
2. Rapid multiplication in response to specific opportunities or conditions

    - Carbohydrate overload
    - Acidic environment
    - Nutrient spillover (high concentrations of protein or sugar) from the rumen into the small intestine
    - Motility disturbance

3. Production of toxin with local and/or systemic effects

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Prevention
Maintain healthy flora
Limit oral medications
Properly fermented feeds
Limit bacteria in colostrum
Feed one colostrum meal

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Prevention
Consistent feeding practices
Adaptation before change
Appropriate fiber length, storage and fermentation of TMR
Avoid rumen acidosis
Manage intensive MR feeding carefully
Water availability for calves
Vaccination
Control other GI infections

Most of the difference in disease levels amongst herds are due **not** to microbiological differences among the herds but rather to differences in the host and environmental factors under the control of management.