MISSISSIPPI STATE UNIVERSITY EXTENSION

Beef Cattle Nutritional Disorders

Nutritional disorders in beef cattle can result in poor animal health, lowered production, and animal loss. The National Animal Health Monitoring System reports that 7 percent of beef cattle death losses in the southeast United States in 2005 were caused by digestive problems such as bloat and acidosis. Cattle deaths from digestive problems increased as herd size decreased.

Mineral imbalances, sudden shifts from high-roughage to high-concentrate diets, and consumption of foreign objects are some of the factors associated with nutritional disorders in beef cattle. Conditions associated with mineral imbalances include grass tetany, water belly, polioencephalomalacia, white muscle disease, and milk fever. Mississippi State University Extension Service Publication 2484 *Mineral and Vitamin Nutrition for Beef Cattle* discusses these conditions in detail. Nutritional disorders outlined in this publication include bloat, acidosis, and hardware disease.

Bloat

Cause

Bloat is a common digestive disorder in beef cattle. It occurs most often in feedlot cattle but affects cattle in all production phases. It results when cattle cannot belch (eructate) or release gases produced from microbial fermentation in the rumen. The animal may produce more gas than it can eliminate. Rumen expansion from gases puts pressure on the diaphragm and lungs. This compression reduces or cuts off the animal's oxygen supply and can eventually suffocate cattle.

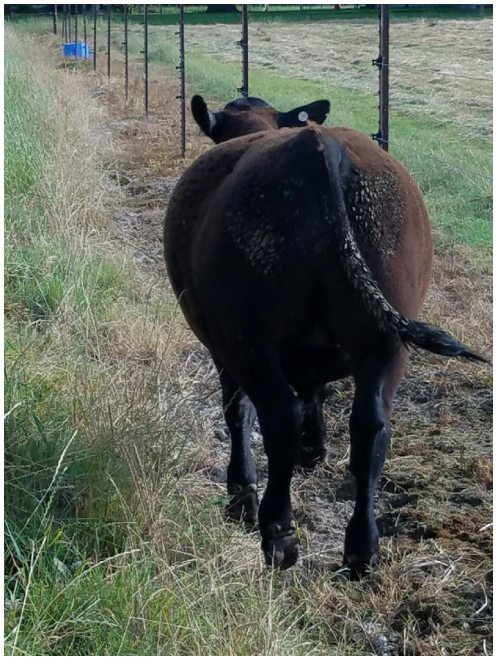
Frothy bloat (feedlot bloat) is the most common type of bloat. It happens when foam in the rumen stops the animal from expelling rumen gases. The foam can cover the cardia (esophageal entrance from the reticulorumen) and prevent the animal from belching. Frothy bloat occurs in cattle fed high-grain diets but is not a major concern for many Mississippi cattle producers. Feedlot bloat is a concern, though, with cattle on high-grain diets, such as bulls on feed-based bull development programs.

Consuming forages with high levels of soluble protein (such as alfalfa, winter wheat, and white clover) contributes to stable foam production. This is called frothy pasture bloat or legume bloat. Legumes that contain leaf tannins help break up the foam in the rumen and are rarely associated with bloat. These tannin-containing legumes include arrowleaf clover, berseem clover, birdsfoot trefoil, sericea lespedeza, annual lespedeza, and crownvetch. Tropical legumes such as kudzu, cowpea, perennial peanut, and alyceclover rarely cause bloat. Bloat can also occur on lush annual ryegrass or small grain pastures, particularly in spring.

Free-gas bloat is another type of bloat that happens when the cardia or esophagus is obstructed or damaged or when rumen movement is depressed.

Clinical Signs

Cattle suffering from bloat swell rapidly on the left side and may die within an hour. Sudden death from bloat is frequently cited in feedlots as a cause of cattle losses. Cattle may show signs of discomfort by kicking at their sides or stomping their feet. Susceptibility to bloat varies with individual animals. Some animals tend to bloat when others do not.



Steer displaying a distended upper left side, indicating bloat.

Management Guidelines

Do not turn shrunken or hungry cattle out onto lush legume or small grain pastures without first filling them up on hay. Bloat is still possible on these forages, even after a frost. Bloat risk is lower later in plant growth when legumes begin to flower. Use of forages containing condensed tannins can help prevent bloat. Slowly adapt cattle from forage-based diets to grain-based diets over a period of at least three weeks. Manage the nutritional programs of chronic bloaters carefully.

Provide poloxalene in a salt-molasses block or as a top dressing according to label recommendations. If providing poloxalene blocks, make sure cattle consume the blocks at least three days before placing the cattle on a pasture with a significant bloat risk. Remove other sources of salt, and place poloxalene blocks (30 pounds per four to five animals), where they will be easily accessible to the cattle. Feeding monensin can reduce the risk of both feedlot and pasture bloat. Monensin is reported to be more effective than lasalocid in controlling bloat, while poloxalene is more effective than monensin for bloat prevention.

Discuss bloat treatment options with a veterinarian. A veterinarian or experienced person can administer poloxalene through a stomach tube to help break up the stable foam and let the animal belch. Do not drench a bloated animal, because of the danger of inhalation and the resulting possibility of pneumonia or death. Feed coarsely chopped roughage as 10 to 15 percent of the ration in a finishing diet. A bloat needle (6 to 7 inches long) or a trocar can be used in extreme cases to puncture the rumen wall on the left side of the animal to relieve pressure inside the rumen. Consider this a last resort, because severe infections may result. Although there is no label claim, research indicates that monensin reduces the incidence and severity of frothy bloat.

Acidosis, Rumenitis, and Liver Abscesses

Cause

Acidosis is often associated with a shift from a forage-based diet to a high-concentrate diet or excessive consumption of fermentable carbohydrates. Acidosis may occur in cattle on high-grain diets common with youth livestock projects, bull development programs, and cattle finishing programs. It can also occur in stocker calves when self-feeders and high-starch feeds such as corn are used.

Acidosis is the result of low rumen pH. The typical pH of the rumen on a forage-based diet is 6 to 7. As the amount of forage or roughage in the diet decreases and the amount of concentrate increases, the pH of the rumen falls between 5 and 6, depending on the forage-to-concentrate ratio of the diet. Low pH supports growth of lactic acid-producing bacteria. Lactic acid is highly acidic and reduces rumen pH even more. Acute (severe) acidosis occurs when ruminal pH drops below 5.2, and subacute (less severe) acidosis occurs at a ruminal pH of less than 5.6. Other conditions such as laminitis, liver abscesses, and polioencephalomalacia often accompany acidosis.

Clinical Signs

Effects of acidosis on cattle may include slowing or stopping gut movement (rumen stasis), diarrhea, and dehydration. Cattle often appear weak, anorexic, and uncoordinated. Manure is often soft, gray, and foamy. Nutrient absorption may be impeded after animals recover from a bout of acidosis. Cattle with subacute acidosis may exhibit reduced but variable feed intake and decreased performance. Susceptibility to subacute acidosis may greatly vary among animals. Acute acidosis can result in heart and lung failure and death.

Both subacute and acute acidosis can lead to rumenitis, which is an infection of the rumen wall. The low pH from acidosis creates lesions in the rumen wall. Damage to the rumen wall from sharp objects (such as wire or nails) predisposes the animal to abscess formation. When rumenitis develops, liver abscesses often follow. Bacteria (*F. necrophorum, Actinomyces pyogenes, Bacteriodes* spp.) from the rumen that cause liver abscesses enter the blood supply through ulcerative lesions, hairs, or foreign objects embedded in the rumen wall. These bacteria then travel via the blood to the liver.

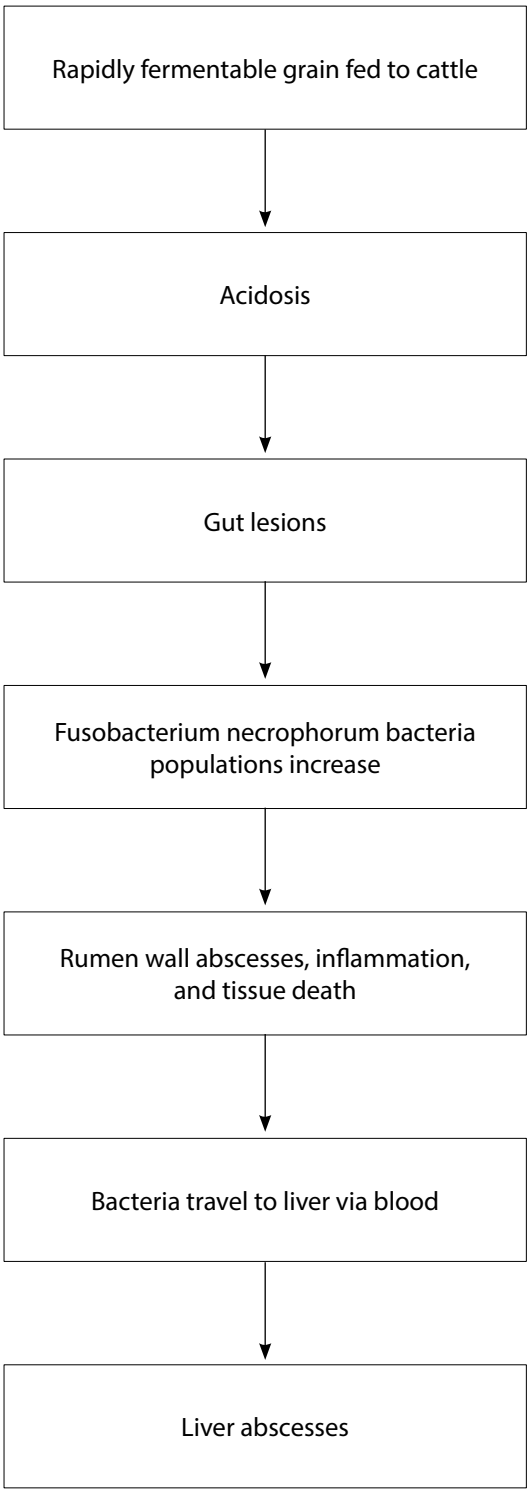
Liver abscesses are most often seen in feedlot cattle. Severe liver abscesses may reduce feed intake, weight gain, feed efficiency, and carcass yield. Abscessed livers are condemned at harvest, regardless of abscess severity. Liver condemnations from abscesses were observed in 13.5 percent of fed cattle in the 2005 National Beef Quality Audit, resulting in about a 2 percent reduction in carcass weight per head. Abscesses were the primary cause of liver condemnations.

Management Guidelines

When cattle are exposed to a high-concentrate diet too quickly, acidosis may result. Fluctuations in eating behavior are often observed. To reduce the incidence of acidosis, use a warm-up feeding period by introducing high-concentrate feeds gradually over three to four weeks. Keep at least 10-percent roughage in the final diet to help moderate rumen pH. Forages and cottonseed hulls are both good roughage sources high in effective fiber.

While acidosis most often occurs during adaptation to concentrate-rich diets, chronic acidosis may continue

Stages of Acidosis, Rumenitis, and Liver Abscess Development



throughout the feeding period. Feeding a combination of grains or feeding a dry grain with a high-moisture grain can reduce the risk of acidosis. Potential for acidosis is higher when feeding wheat compared to corn.

Processing grains less thoroughly and limiting the quantity of feed offered should reduce acidosis incidence but may also lower animal performance. Good bunk management where all feed is consumed before the next feeding may lessen daily fluctuations in feed intake and reduce acidosis risk.

Feeding ionophores (monensin or lasalocid) can help reduce the incidence of acidosis. Ionophores may reduce intake and help moderate concentrate intake when calves start on higher-concentrate diets. Adding bicarbonate, fat (up to 8 percent of the diet), probiotics, virginiamycin, or thiamin to the diet or increasing protein in the diet may decrease acidosis risk.

Treatment for acidosis is similar to prevention efforts. Tylosin is effective in decreasing liver abscess incidence. Virginiamycin and chlorotetracycline are also used in addressing liver abscess problems.

Hardware Disease

Cause

Hardware disease is the common name for traumatic gastritis and traumatic reticulitis. It may occur when cattle consume sharp, heavy objects, such as nails or wire. These objects fall to the rumen floor and then are swept into the reticulum by muscle contractions. Cattle may ingest these objects and never have hardware disease, or muscle contractions may cause these sharp objects to puncture the reticulum wall, diaphragm, and heart sac. Forceful abdominal movement during calving may force a sharp object through the reticulum wall. This leads to severe damage and infection in the abdominal cavity, heart sac, or lungs.

Clinical Signs

Signs of hardware disease vary, depending on where the puncture occurs. Loss of appetite, depression, a reluctance to move, and arched back, and indications of pain are common signs. The animal may grunt when forced to walk. Recurring bloat may be observed on the upper left side, with fluid accumulation on the lower right. If the heart sac has been punctured, fluid may accumulate around the heart and in the brisket. Fatal infection can occur if the object penetrates close to a site near the heart.

Management Guidelines

Take measures to prevent cattle from ingesting heavy, sharp objects. Keep pastures, paddocks, and feed bunks free of wire, nails, fencing staples, and other sharp objects (even heavy plastic items) that could be swallowed. Debris from structures and equipment may appear in areas where cattle graze, after high winds. Deteriorating steel-belted tires also pose a risk.

Place magnets on feeding equipment to catch some of the metal objects in feed. An intraruminal magnet can be inserted directly into the rumen to trap metal fragments. Ingested metal is drawn to the magnet instead of working its way through the stomach wall. The magnet eventually becomes full if enough metal is ingested. Administer a second magnet if signs of hardware disease persist. Magnets are relatively inexpensive, especially when compared to the cost of surgery.

It is often difficult to diagnose hardware disease. Seek veterinary advice for suspect cases. Administer an intraruminal magnet when hardware disease cannot be ruled out. Confining cattle and limiting feed intake may allow puncture sites to heal in less serious cases. If infection is suspected, administer a broad-spectrum antibiotic.

The severity of the infection and the duration of the condition affect the animal's treatment outlook. Cattle with extensive infection in the heart or abdomen have a very poor prognosis. Even with attempts to encourage feed consumption, these animals ultimately often die of starvation. Sometimes only surgical removal of the object works. Controlling infection is important after the object is removed for successful recovery. Surgery is often not a cost-effective option unless the cattle affected are very valuable.

Conclusions

Knowledgeable beef cattle producers can reduce or eliminate risk factors for bloat, acidosis, and hardware disease. Watch cattle for signs of nutritional disorders to facilitate early intervention and treatment. Seek veterinary assistance when developing and implementing treatment programs for suspected cases of nutritional disorders. For more information on beef cattle nutritional disorders, contact your local MSU Extension office.

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