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Nutritional Disorders in Beef Cattle

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Introduction

Nutritional disorders associated with both forage and feed consumption can have a large impact on the profitability of beef cattle operations. Forages are an important component of beef cattle production systems in Arkansas. Most cow-calf and stocker cattle enterprises in Arkansas rely heavily on forage-based nutritional programs. Forages are used for both livestock grazing and hay production. Arkansas has over 4.4 million acres of pastureland and harvests over 1.3 million acres of hay each year. Grains and by-product feeds are also included in the nutritional programs of many Arkansas cattle herds. These concentrate feeds are often fed as nutritional supplements to cattle during periods of low forage quality, when forage supply is limited, as part of performance testing programs or when feeding animals with elevated nutritional requirements.

Mineral imbalances and sudden shifts from high roughage to high concentrate diets are some of the factors associated with nutritional disorders in beef cattle. Simple management practices can be implemented to reduce the risk of experiencing a nutritional disorder in a cattle herd. Identifying potential problems, using proper treatments, and preventing future occurrences of nutritional disorders can help protect both cattle health and profitability.

Grass Tetany

Cause: Grass tetany is associated with low levels of magnesium or calcium in cattle grazing ryegrass, small grains (e.g., oats, rye, wheat) and cool-season perennial grasses (e.g., tall fescue, orchardgrass) in late winter and early spring. In Arkansas, the grass tetany season runs from February through April (Figure 1). During this time of the year, there is often a flush of new forage growth. Forages grown on soils deficient in magnesium, wet soils or soils low in phosphorus but high in potassium and nitrogen may contain very low levels of magnesium and calcium. This is also the time of the year when many spring calves are born and nursing. Grass tetany most commonly affects lactating cattle, particularly the highest-producing animals in the herd. Magnesium and calcium requirements of lactating cattle are far greater than those of dry cattle. This predisposes cattle to grass tetany during lactation. Grass tetany results when magnesium and calcium levels

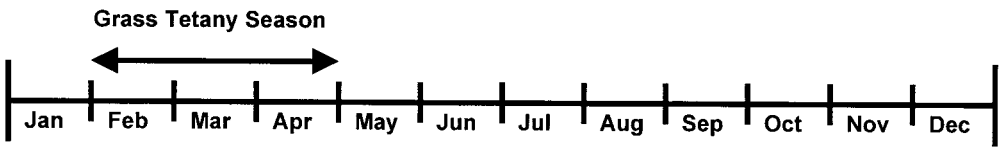

Figure 1. Grass tetany season in Arkansas

in forages are too low to meet the requirements of cattle and cattle do not receive adequate magnesium and calcium supplementation. Clinical signs of grass tetany include nervousness, muscle twitching and staggering during walking. An affected animal may go down on its side, experience muscle spasms and convulsions and die if not treated.

Prevention: Magnesium-deficient pastures should be limed with dolomitic lime, which contains magnesium. This may not be effective in preventing grass tetany on water-logged soils, since plants may not be able to take up sufficient magnesium under wet conditions. Phosphorus fertilization may also be useful for improving forage magnesium levels. However, environmental concerns associated with excessive soil phosphorus levels should be considered. Legumes (e.g., clovers, alfalfa, lespedezas) are often high in magnesium and may help reduce the risk of grass tetany when included in the forage program. **The most reliable method of grass tetany prevention is supplemental feeding of magnesium and calcium during the grass tetany season.** Both can be included in a mineral mix as part of a mineral supplementation program. Start feeding a high magnesium mineral one month prior to grass tetany season.

Treatment: Early treatment of grass tetany is important. Collapsed cattle that have been down more than 12 to 24 hours will seldom recover. Blood magnesium levels can be increased within 15 minutes by intravenously administering 500 ml of calcium borogluconate solution with 5 percent magnesium hypophosphate. The solution must be administered slowly, and heart and respiratory rates should be monitored closely during administration. After treating with the intravenous solution, orally administer one tube of CMPK gel (a source of calcium, phosphorus, magnesium and potassium) or intraperitoneally administer another 500 ml bottle of calcium borogluconate solution with 5 percent magnesium hypophosphate for slow absorption to decrease the possibility of relapse. If the animal is treated using subcutaneous (under the skin) administration, the desired effect may not occur for three to four hours. A 20 percent magnesium sulfate (epsom salt) solution is recommended for subcutaneous administration, because tissue sloughing may occur with a higher dosage.

Bloat

Cause: Bloat results from the formation of a stable foam in the rumen that prevents eructation (belching) and release of gases produced normally from microbial fermentation. Gas production may then exceed gas elimination. Rumen expansion from gases compresses the lungs and reduces or cuts off the animal's oxygen supply resulting in suffocation. Cattle will swell rapidly on the left side and may die within an hour in some cases. Cattle may exhibit signs of discomfort by kicking at their bellies or stomping their feet. Susceptibility to bloat varies with individual animals. There are two types of bloat: legume/pasture bloat or frothy/feedlot bloat. Several different forage species can cause legume bloat including **alfalfa, ladino or white clover and persian clover**. Other legumes contain leaf tannins that help break up the stable 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. Similarly, tropical legumes such as kudzu, cowpea, perennial peanut and alyceclover rarely cause bloat. Bloat can also occur on **lush ryegrass or small grain pastures, particularly in spring**. Feedlot bloat occurs in cattle fed **high grain diets**. Feedlot bloat is not a major concern for many cattle producers in Arkansas. However, "feedlot" bloat is a concern with cattle on high grain diets, e.g., bulls on feed-based on-farm bull performance tests.

Prevention: Do not turn shrunk or hungry cattle out onto lush legume or small grain pastures without first filling them up on hay. Poloxalene can be provided in a salt-molasses block (30 grams of poloxalene per pound of block) or as a topdressing to feed at a rate of one to two grams per 100 pounds of body weight per day. If a poloxalene block is provided, make sure cattle consume the blocks at least three days before placing them 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 Rumensin® in grain-based rations can reduce the risk of feedlot bloat. Cattle should be slowly adapted from forage-based diets to grain-based diets over a period of at least three weeks.

Treatment: Poloxalene may be administered through a stomach tube to help break up the stable foam and allow the animal to eructate (belch). **Do not drench a bloated animal** because of the danger of inhalation and subsequent pneumonia or death. Feed coarsely chopped roughage as 10 to 15 percent of the ration in a feedlot diet. A bloat needle (six to seven 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. This treatment option should be considered a last resort as severe infections may result. Although there is no label claim, research indicates that Rumensin® reduces the incidence and severity of frothy bloat.

Acidosis, Rumenitis, Liver Abscess Complex

Cause: Acidosis is a disorder associated with a **shift from a forage-based diet to a high concentrate (starch) diet**. This is a problem that is most often discussed as a feedlot problem, but acidosis may also occur in other cattle on **aggressive grain feeding programs** such as 4-H projects and on-farm bull tests. Acidosis is a potential problem for backgrounders using self-feeders and high starch feeds such as corn and bakery by-products.

As the name implies, acidosis results from **low rumen pH** (Figure 2). The rumen contains many different species of bacteria and other microorganisms. Some of the bacteria prefer forage (slowly fermented structural sugars) while others prefer starch (rapidly fermented sugars). During the change from a forage-based diet to a concentrate diet, the microbial population shifts from predominately forage fermenters to predominately starch fermenters. All bacteria in the rumen produce acids as a fermentation waste product. These acids are an extremely important source of energy for the ruminant animal. The dominating forage fermenters produce acetic acid (more commonly known as vinegar), which is a mild acid. 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 corresponding shift in the bacterial population results in an increase in propionic acid production. Propionic acid is a stronger acid than acetic acid and, therefore, it reduces rumen pH. The pH of the rumen now will be between 5 and 6 depending on the forage to concentrate ratio of the diet. Low pH (<5) may support the growth of lactic acid producing bacteria. Lactic acid is a very strong acid and reduces rumen pH even further. It is this low pH from lactic acid production that is associated with acidosis. **Acidosis is likely to occur when calves with developed rumens are exposed too quickly to a high concentrate diet.** This will result in fluctuations in eating behavior. The calf fills up on the high concentrate diet, and the rumen becomes acidic. The calf feels ill and goes off feed. The calf recovers, fills back up on the high concentrate diet, and the cyclical eating behavior starts all over again. Acute lactic acidosis can result in death.

Liver abscesses are often a secondary result of acidosis. The low pH from acidosis results in necrotic lesions of the rumen wall. Necrotic lesions of the rumen wall provide an escape route for the bacteria from the rumen into the blood supply connecting the rumen to the liver. The bacteria are transported to the liver where they take up residence. Damage to the ruminal wall from acidosis can be further aggravated by damage from foreign objects (i.e., wire, nails) and predispose the animal to abscess formation. The National Beef Quality Audit–2000 revealed that the incidence of liver condemnations in beef carcasses was 30.3 percent, with the leading cause being liver abscesses. Too frequent liver condemnations ranked in the top ten quality challenges for the fed beef industry according to survey participants in the Strategy Workshop of the National Beef Quality Audit—2000. Severe liver abscesses may reduce feed intake, weight gain, feed efficiency and carcass yield.

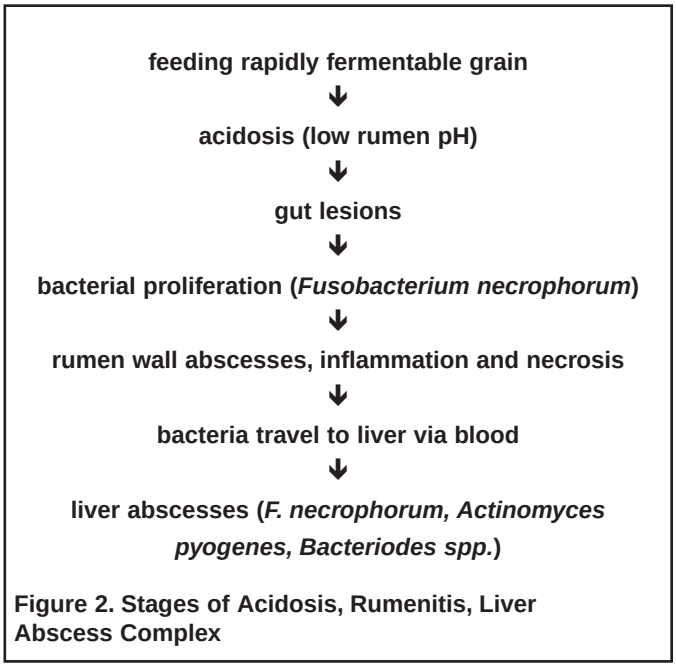

Figure 2. Stages of Acidosis, Rumenitis, Liver Abscess Complex

Prevention: To reduce the incidence of acidosis, use a warm-up feeding period and ensure at least 10 percent roughage in the final diet. A warm-up period should consist of starting the calves with a diet that contains 40 to 60 percent roughage and over a three- to four-week period gradually reduce the roughage content of the diet while increasing the concentrate level. Keeping at least 10 percent roughage in the diet will help moderate rumen pH. The fiber should be long enough to serve as a "scratch factor" and stimulate rumination. Cud chewing stimulates saliva production, and saliva is a good source of buffers. Forages and cottonseed hulls are both good sources of effective fiber. Ionophores can help reduce incidence of acidosis as well. Research has shown that monensin (Rumensin®) may reduce intake and thus can help moderate concentrate intake when calves are started on higher concentrate diets. Always follow labeled instructions and withdrawals when using medicated feed additives.

Treatment: Treatment for acidosis is similar to prevention efforts.

Urinary Calculi or "Water Belly"

Cause: Urinary calculi (kidney stones) are hard mineral deposits in the urinary tracts of cattle. Affected cattle may experience chronic bladder infection from mechanical irritation produced by the calculi. In more serious cases, calculi may block the flow of urine, particularly in male animals. The urinary bladder or urethra may rupture from prolonged urinary tract blockage, resulting in release of urine into the surrounding tissues. The collection of urine under the skin or in the abdominal cavity is referred to as "water belly." Death from toxemia may result within 48 hours of bladder rupture. Signs of urinary calculi include straining to urinate, dribbling urine, blood-tinged urine and indications of extreme discomfort, e.g., tail wringing, foot stamping and kicking at the abdomen. Phosphate urinary calculi form in cattle on high grain diets, while silicate urinary calculi typically develop in cattle on rangeland.

Prevention: Strategies to prevent problems with urinary calculi in cattle include lowering urinary phosphorus levels, acidifying the urine and increasing urine volume. To lower urinary phosphorus levels, rations high in phosphorus should be avoided. **Maintain a dietary calcium to phosphorus ratio of 2:1.** Acid-forming salts such as ammonium chloride may be fed to acidify the urine. Ammonium chloride may be fed at a rate of 1.0 to 1.5 ounces per head per day. Urine volume may be increased by feeding salt at 1 to 4 percent of the diet while providing an adequate water supply.

Treatment: Limited success with treatments designed to facilitate passing or dissolving urinary calculi leaves few other treatment options. Surgery may be the most effective treatment. However, the cost of surgery should be considered and weighed against the value of the animal.

Hardware Disease

Cause: Hardware disease may occur when **sharp, heavy objects such as nails or wire are consumed by cattle**. These objects fall to the rumen floor and are swept into the reticulum (another stomach compartment) by muscle contractions. A sharp object may puncture the reticulum wall and cause severe damage to and infection of the abdominal cavity, heart sac or lungs. Signs of hardware disease vary depending on where the puncture occurs. Loss of appetite and indications of pain are common signs. Fatal infection can occur if the object penetrates close to the heart.

Prevention: Cattle should be managed so that they do not have opportunity to ingest heavy, sharp objects. Keep pastures and paddocks free of wire, nails and other sharp objects (even heavy plastic items) that could be swallowed. Magnets can be placed on feeding equipment to catch some of the metal objects in feed. An intraruminal magnet can be inserted 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 will eventually "fill up" if enough metal is ingested, so a second magnet may be administered if signs of hardware disease persist. Magnets are relatively inexpensive particularly when compared to the cost of surgery.

Treatment: It is often difficult to diagnose hardware disease, yet it is prudent to administer an intraruminal magnet when hardware cannot be ruled out. Confinement and feed intake limitation may allow puncture sites to heal in less serious cases. If infection is suspected, a broad-spectrum antibiotic should be administered. Cattle with extensive infection in the heart or abdomen have a very poor prognosis and will often die of starvation despite attempts to encourage feed intake. In some instances, cattle suffering from hardware disease will respond only to surgery and physical removal of the object. These cattle may recover if infection is controlled after the object is removed. It is important to note that surgery may not be a cost-effective option, particularly for less valuable cattle.

Polioencephalomalacia

Cause: Polioencephalomalacia is caused by a disturbance in thiamine metabolism. Thiamine is required for a number of important nervous system functions. This disease most commonly affects **young, fast growing cattle on a high concentrate ration** and may result from a **thiamine-deficient diet**, an increase in thiaminase (an enzyme that breaks down thiamine) in the rumen or an increase in dietary sulfates.

A thiamine-deficient diet is usually associated with an increase in the dietary concentrate:roughage ratio. When concentrates (feed grains such as corn) are increased and roughage (forage, cottonseed hulls, etc.) is decreased in the diet, rumen pH drops. This increases the numbers of thiaminase-producing bacteria in the rumen and decreases the amount of total useable thiamine. Thiaminase breaks down the form of thiamine that the animal could normally use. Some species of plants produce thiaminase and can cause a decrease in the useable amount of thiamine when consumed. Examples of these types of plants include **kochia, bracken fern and equisetum**.

A **high sulfate diet** can also inhibit an animal's ability to properly utilize thiamine. Feeds such as molasses and corn gluten are often high in dietary sulfates. Some water sources can also contain a high amount of sulfur (i.e., "gyp" water). When these are consumed in excessive amounts, clinical signs of polioencephalomalacia can occur.

Cattle that are affected with this disease usually exhibit several signs of generalized neurological disease. These signs can include but are not limited to blindness, inconsistent and uncoordinated movements, head pressing, "goose" stepping, lateral recumbency (lying with full lateral contact of the body trunk, head, neck and legs with the ground with the head, neck and legs usually extended), tetany (muscle spasms), convulsions with paddling motions and death. These signs usually exhibit sudden onset, with the animals typically having normal temperatures and rumen function.

Prevention: Preventative strategies should focus on the diet. Risk factors such as high concentrate rations or high sulfate diets should be avoided if possible. Thiamine can also be added to a feed ration or a free-choice mineral supplement at 3-10 ppm. However, this may not be cost effective in some instances.

Treatment: For successful recovery, **early treatment is essential**. Thiamine should be administered at 5-7 mg/lb (10-15 mg/kg) intravenously. The initial treatment should be followed with intramuscular injections twice daily with the same dosage for the next 2-3 days. If calves are not treated early in the disease process, they may show residual neurological effects indefinitely.

White Muscle Disease

Cause: "White muscle disease" (enzootic nutritional muscular dystrophy) most commonly affects cardiac or skeletal muscle of **rapidly growing calves**. This disease causes muscle **degeneration due to vitamin E and/or selenium deficiency**. This metabolic imbalance can be due to dietary deficiency or to calves being born to dams that consumed selenium-deficient diets during gestation. There are two distinct syndromes of this disease: a cardiac form and a skeletal form. The cardiac form of the disease usually has rapid onset with the most common clinical sign being sudden death. Initially, animals may exhibit an increased heart rate and respiratory distress, but death usually occurs within 24 hours. The skeletal form of the disease generally has a slower onset. Calves affected by the skeletal form exhibit stiffness and muscle weakness. Although these animals usually have normal appetites, they may have an inability to stand for extended time periods and exhibit respiratory distress if their diaphragm or intercostal muscles are involved. Some animals may show signs of dysphagia (difficulty swallowing and possible pain while swallowing) if the muscles of the tongue are also affected.

Necropsy of an affected animal often reveals pale discoloration of the affected muscle. The texture of the muscle is dry with white, chalky, streaked sections representing the fibrosis and calcification of the diseased tissue, hence the name "white muscle disease."

Prevention: Control of this disease is achieved through supplementation of vitamin E and selenium. Salt/mineral mixtures can be used to supplement the deficiencies. A free-choice mineral supplement with an expected intake of 4 ounces/head/day should contain 27 ppm of selenium. If you are in a known selenium-deficient area, it is recommended to administer 25 mg of selenium and 340 IU of vitamin E intramuscularly approximately four weeks before calving.

Treatment: Treatment for the cardiac form of the disease is rarely successful. The skeletal form may be treated with an injection of vitamin E and selenium. The appropriate dose is 1 mg of selenium and 68 IU of vitamin E per 40 pounds of body weight intramuscularly. This dose may be repeated in two weeks if needed. There is a 30-day slaughter withdrawal period once calves have been treated. Supplements may be given simultaneously to help with recovery.

Milk Fever

Cause: Milk fever (parturient paresis or hypocalcemia) is generally associated with older, high-producing dairy cattle. However, incidences of milk fever may also occur with beef cattle. Milk fever occurs **shortly after calving and the onset of milk production**. Milk fever occurs when the lactating cow is not capable of absorbing enough calcium from the diet or has not started mobilizing bone calcium to meet the increased calcium demand of lactation. Calcium losses from lactation coupled with inadequate supply results in a drop in blood calcium level. Since calcium is needed for muscle contraction, cows suffering from milk fever often lose their ability to stand.

Prevention: Numerous steps can be taken to prevent milk fever. The first is to evaluate the calcium and phosphorus levels of the diet. Excessive dietary calcium during late pregnancy could leave the cow unprepared to absorb or mobilize (resorb from bone) enough calcium to meet elevated requirements

when lactation starts. This situation sometimes occurs with feeding poultry litter. Feeding low calcium diets a month or two prior to calving was once thought to be the best method of prevention because the body would be geared to mobilizing bone calcium. However, this approach has had limited success with high-producing dairy cattle and is difficult with high forage diets. If milk fever is a common problem in the herd, feeding an anionic pre-partum diet (a negative dietary cation – anion difference, DCAD) will help prevent milk fever. Adequate vitamin D is also important in preventing milk fever, but is not a problem with beef cattle on pasture.

Treatment: The most common treatment is slowly applying an intravenous injection of a calcium gluconate solution. Single-dose bottles are typical and are available at local veterinary clinics and supply stores. Calcium may also be provided orally as calcium propionate in a gel form. Re-treatment is necessary in some cases.

Summary

Nutritional disorders (Table 1) may not be a concern for cattle producers until animals in the herd are affected. That can be too late. It is important to be alert for "red flags" in animal behavior and appearance to catch a problem early and minimize losses. Your local veterinarian should be familiar with nutritional disorders that are common in your area and can assist you with prevention and treatment programs. A good defense (prevention) is a good offense when it comes to nutritional disorders. Understanding what causes nutritional disorders in beef cattle and implementing proper forage, feeding and animal management practices may spare the experience of production, animal or economic losses associated with nutritional disorders. It is much less costly to prevent a problem than to try to treat one. Keeping Arkansas cattle herds healthy is not only beneficial to the pocketbook, it is good, sound animal husbandry.

Table 1. Summary of nutritional disorders in beef cattle

Disorder	Signs	Cause	Prevention	Treatment
Grass Tetany	Nervousness, muscle twitching, staggers, collapse, muscle spasms, convulsions, coma, death	Low blood magnesium or calcium levels	Provide magnesium and calcium mineral supplement during grass tetany season to lactating cattle Use dolomitic lime on pastures Use legumes in forage program	500 ml of calcium boroglu-conate solution with 5% magnesium hypophosphate administered slowly intra-venously while monitoring heart and respiratory rate followed by one tube of oral CMPK gel to minimize the chance of relapse
Bloat	Distended left side, kicking at belly, stomping feet, absence of belching, frequent urination and defe-cation, labored breathing, suffocation, death	Stable foam in rumen prevents eructation (belching), animal eventually suffocates	Put out poloxalene (Bloat Guard™) salt-molasses blocks or feed additive to cattle at risk Fill cattle up on hay before turning out onto lush pasture and provide hay during initial grazing days Do not place animals on lush "problem" forage just after a frost Keep legume levels in pastures at ≤50% of the available dry matter Check cattle on legume pasture frequently and remove if signs of bloat develop Keep water and salt avail-able at all times on legume pastures Feed Rumensin®	Insert 3/4" rubber hose into rumen via esophagus Administer defoaming agent (poloxalene or mineral oil) via stomach tube (do not drench) Use bloat needle or trocar to puncture hole in rumen wall to relieve pressure as a last resort

Table 1. Summary of nutritional disorders in beef cattle (cont.)

Disorder	Signs	Cause	Prevention	Treatment
Acidosis, Rumenitis, Liver Abscess Complex	Reduced feed intake, animal goes off feed, reduced weight gains, decreased feed efficiency, lameness in some cases	Overloading on concentrate after a period of reduced feed consumption, increasing concentrate level in diet too rapidly	Reduce concentrate to roughage ratio Gradually increase the concentrate level in the diet when switching from a roughage-based diet Feed Rumensin®	Similar to prevention
Urinary Calculi "Water Belly"	Straining to urinate, dribbling of urine, blood-tinged urine, signs of extreme pain (tail wringing, feet stamping, kicking at abdomen), death following urethral or urinary rupture	Sudden change in ration, calcium to phosphorus imbalance that promotes alkaline urine high in phosphorus	Maintain a 2:1 dietary calcium to phosphorus ratio; higher phosphorus levels can contribute to the problem Feed ammonium chloride at a rate of 1.0 to 1.5 ounces per head per day to acidify urine Feed salt (NaCl) at 1 to 4% of diet and provide adequate water to increase urine volume	Surgery (may not be cost-effective)
Hardware Disease	Loss of appetite, reduced milk production, abdominal pain, labored breathing	Ingestion of sharp objects that puncture the reticulum wall and may cause severe damage to the abdominal cavity, heart sac or lungs	Keep cattle in an area where they cannot find and eat wire, nails and other sharp objects Magnets can be placed on feeding equipment to catch metal objects in feed Intraruminal magnets can be used to trap metal fragments	May heal in mild cases if feed intake is reduced and animal is immobilized Antibiotics Surgery in severe cases (may not be cost-effective)
Polioencephalomalacia	Generalized neurological disease: blindness, uncoordinated movements, head pressing, "goose" stepping, lying with extended head and legs, muscle spasms, convulsions with paddling motions, death	Thiamine deficiency, particularly in young, fast-growing cattle on a high concentrate ration	Evaluate dietary thiamine levels and avoid risk factors such as high sulfate diets Add thiamine to feed ration or free-choice mineral supplement at 3-10 ppm (may not be cost effective)	Thiamine administered intravenously at 5-7 mg/lb (10-15 mg/kg) followed by intramuscular injections twice daily with the same dosage for the next 2-3 days (early treatment essential)
White Muscle Disease	Cardiac form: rapid onset commonly resulting in sudden death Skeletal form: exhibit stiffness and muscle weakness, inability to stand for extended time periods, respiratory distress, difficulty swallowing, normal appetite, necropsy often reveals pale discoloration and dryness of the affected muscle with white chalky streaks	Muscle degeneration due to vitamin E and/or selenium deficiency	Mineral supplementation of 27 ppm selenium for free-choice feeding at 4 ounces per day or 54 ppm selenium for free-choice feeding at 2 ounces per day In known selenium-deficient areas administer 25 mg selenium and 340 IU vitamin E intramuscularly approximately four weeks before calving	Treatment for cardiac form rarely successful For skeletal form administer intramuscular injection of 1 mg selenium and 68 IU vitamin E per 40 pounds of body weight repeated in two weeks if needed (30-day slaughter withdrawal after treatment)

Table 1. Summary of nutritional disorders in beef cattle (cont.)

Disorder	Signs	Cause	Prevention	Treatment
Milk Fever	Muscle stiffness, tremors, extreme weakness, incoordination, inability to stand, loss of consciousness	Drop in blood calcium levels, often occurs shortly after calving and the onset of milk production	Evaluate dietary calcium and phosphorus levels and avoid excessive dietary calcium Feed an anionic prepartum diet if milk fever is a common problem in the herd Removal of poultry litter from the diet of gestating cows 30 days before calving	Intravenous injection of a calcium gluconate solution Calcium provided orally as calcium propionate in a gel form (re-treatment may be necessary)

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