



# Texas Agricultural Extension Service

The Texas A&M University System

## Forage-related Cattle Disorders

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Cattlemen often are relieved when it is time to move cattle from a winter feeding program to spring pasture because cattle health problems usually are minimized by adequate quantities of high quality forage. Forage supplies the major portions of protein, energy, minerals and vitamins in most beef cattle diets.

However, infrequent observation of cattle on pasture and the difficulty of treating grazing animals often result in deaths or severe production loss when pasture-related disorders occur. Because of such problems as bloat or prussic acid poisoning, cattlemen may avoid some agronomic practices that could greatly increase productivity.

This information is not a guide for the diagnosis and treatment of cattle disorders, but addresses pasture situations that may result in major health problems and management steps to minimize their probability of occurrence. General descriptions of symptoms and treatments are presented.

Some disorders are closely related. Prussic acid poisoning and nitrate poisoning are different disorders but often are confused because both can occur in cattle consuming certain plants of the sorghum family. Similarly, nitrate poisoning and grass tetany may be confused and lumped together as "pasture poisoning" when two different situations actually exist—poisoning from high nitrate levels and, separately, a deficiency of magnesium. Bloat can be a symptom of several of the disorders discussed in this publication. Professional assistance may be needed for proper diagnosis of disorders encountered.

### Forage Bloat

Bloat describes a digestive disorder of ruminants characterized by over-distention of the rumen with gas. It is a serious, practical livestock problem resulting in significant economic losses each year. Even greater

economic losses result from the limited use of high-yielding legumes in pastures because of the *fear* of bloat.

Bloat may occur in a variety of feeding situations. Grain rations rich in protein, carbohydrates or fat predispose feedlot cattle to bloat. The majority of bloat on pasture, however, occurs with animals grazing succulent legumes. Lush pastures, particularly those dominated by rapidly growing legume plants, most commonly are associated with serious bloat. Such pastures do not always induce bloat and other types of pastures are not always safe. Legumes most commonly identified with bloat are immature ladino clover, persian clover and alfalfa.

**Causes.** Forage-induced bloat results from the formation of a stable foam in the rumen, a retention of gas with increasing pressure and an inhibition of the belching mechanism. Exact causes of bloat are not fully understood, but production of stable foam in the rumen is caused by a complex interaction of animal, plant and microbial factors.

Marked differences exist among animals in their susceptibility to bloat. Some animals in a herd are bloaters and some are not. Knowing there are animal differences in susceptibility and plant differences in predisposition, researchers have attempted to isolate and identify chemical causative agents of bloat. Much information has been gained from these studies, but no compound has been accepted as the singular cause of bloat. Theories have been proposed, but none has provided a completely satisfactory explanation.

**Symptoms.** In exceptional cases, bloating and death of susceptible animals have occurred within an hour after entry to the pasture. Usually, however, several hours of grazing pass before severe symptoms develop. The animal's inability to expel gas as it forms allows pressure to increase within the rumen. As pressure increases, the rumen becomes distended on the animal's upper left side between the last rib and the point of the hip. As pressure continues to increase, the upper right

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flank also becomes distended. This sign usually is accompanied by labored breathing, excessive salivation, staggering, and occasionally, vomiting. After the animal is no longer able to stand, death follows within a few minutes.

**Prevention.** Although it may be impossible to prevent all bloat, some precautions can reduce the incidence of this problem:

- Plant grass-legume pasture mixtures instead of pure legumes.
- Provide a full feed of dry roughage before turning animals into a lush legume stand and observe animals frequently during grazing.
- Avoid grazing very immature legumes.
- Where bloat has occurred, remove cattle to hay at night or alternately graze legume and grass pasture.
- Cull chronic bloaters as susceptibility to bloat is heritable.
- Feed hay on young legume pasture if a history of bloat exists.
- Put animals on legumes only when plants are free of surface moisture (dew or rain).
- Restrict the area grazed and the length of the grazing period.

A combination of the above management practices has reduced the incidence of bloat in many herds. Recently developed additives also have successfully prevented bloat. Poloxalene, in either a dry or liquid feed supplement, is quite effective if all animals consume the recommended amounts daily.

**Treatment.** Give a defoaming agent immediately. A pint of vegetable oil, such as peanut oil or corn oil, or about one-fourth cup of mild detergent given as a drench is sometimes effective. Place a gag 1 to 1-1/2

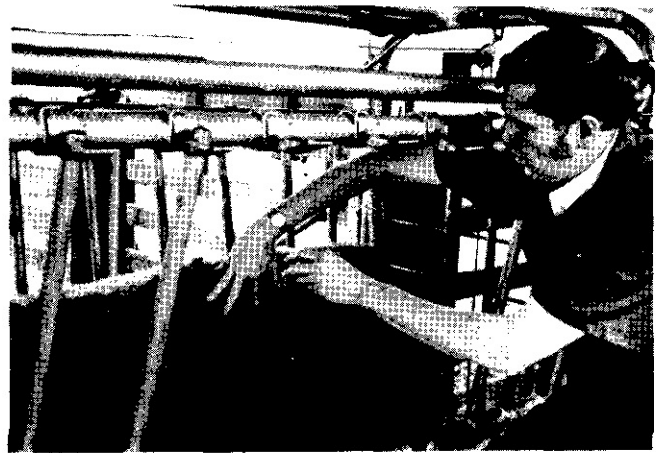
**Figure 1. A yearling with a gag in its mouth to induce belching.**



inches in diameter in the animal's mouth in the position of a bit in a horse's mouth. The animal's chewing on the gag often induces belching. If relief is not soon evident, summon a veterinarian. Sustained pressure may cause ruminal gases to enter the bloodstream, resulting in a toxemia.

A large diameter trocar and cannula (a sharp, pointed instrument fitted in a tube) may be used to puncture the rumen at the highest distention on the left side. If death appears imminent, relief may be obtained by making an incision 3 to 4 inches long in the highest distention on the left side. This incision must then be surgically repaired and antibiotics administered to minimize infection.

**Figure 2. Correct positioning of a trocar and cannula is at the high point of distention midway between the last rib and the point of the hip.**



### Red Clover Slobbers

Excessive salivation (slobbering) occasionally has been observed in livestock consuming second or later cuttings of red clover hay. This condition is associated with a disease of red clover called "black patch." The name black patch is due to microscopic dark brown to black filaments on the leaves and stems of red clover, produced by the fungus (*Rhizoctonia leguminicola*). It should not be inferred that all second-cutting red clover is unpalatable or that it will cause slobbering when eaten. Black patch is transmitted through infected red clover seed and from plant to plant. Growth of the fungus is favored by a high relative humidity, a temperature between 75° F and 85° F, and a soil pH of 5.9 to 6.5. Delaying hay harvest beyond the early bloom stage results in lower palatability and a build-up of the fungus. The fungus or the toxin it produces dissipates gradually from stored hay.

**Cause.** Slaframine, an alkaloid produced by the black patch fungus, is converted to an active compound by enzymes produced in the liver. This "slobber factor" has a stimulating effect, causing excessive activity of

involuntary muscles in certain systems of the body. The effects of the toxin are long-lasting, and will persist for several days after feeding of the affected hay is discontinued.

**Symptoms.** The most prominent sign is excessive salivation. Infected hay is dusty and unpalatable, resulting in reduced consumption. Other symptoms are tear shedding, diarrhea, frequent urination, reduced gains and lowered milk production. Subnormal temperature and bloat are signs of severe poisoning, requiring immediate treatment.

**Prevention.** The fungus is present in most red clover fields and no resistant varieties are available. Do not harvest seed from an infected field. To minimize the severity of the problem when a field is infected:

- Harvest each cutting of red clover during early bloom.
- Mix later cuttings of red clover hay with other hay for feeding if slobbering problems exist.
- Store second-cutting red clover hay until the next year's feeding season, or feed it last during the winter following harvest.

**Treatment.** Remove all infected hay from the diet of affected animals. Administer phenothiazine derivative antihistamines and/or atropine to counteract the effect of the toxin present in the body.

### Fescue Foot

Fescue foot is a noninfectious disease of cattle characterized by rear leg lameness that progresses to dry gangrene of the rear feet, occasionally the tail and, rarely, the ears. The condition has been observed in cattle grazing tall fescue in New Zealand, Australia and the United States. In the United States, the condition has been reported from most areas of the southeast with the majority of cases occurring in a belt extending from Kansas through Missouri, southern Illinois and southern Indiana to Kentucky.

Fescue foot results in severe economic losses within affected herds; but, when the total number of cattle grazing fescue is considered, the percentage affected by this disease is very low. Other conditions often confused with fescue foot are foot rot, injury, frostbite and ergot poisoning. Fescue foot may occur any time during the year, but most cases are found in animals grazing unclipped pure stands of fescue during late fall and winter.

**Cause.** The specific cause of fescue foot is unknown. Evidence suggests it may be caused by a toxin produced by fungus that grows on or in conjunction with the fescue plants.

**Symptoms.** The first symptoms usually develop within 5 to 15 days after cattle begin grazing tall fescue. Affected cattle often isolate themselves from the rest of the herd and have a depressed appetite. Early signs are

**Figure 3. An inflamed band at the top of this rear foot indicates an early case of fescue foot.**



slightly arched back, a rough hair coat, soreness and lameness in one or both hind feet. While standing, the animal may shift from one rear foot to the other. As the condition progresses, a reddened indented line or band usually encircles the rear limb just above the hoof. With increased severity, that portion of the foot below the line sloughs. The tip of the tail and outer one-third of the ears also may fall off. Emaciation is common as feed intake is reduced. There is a higher incidence of fescue foot among malnourished cattle than among well-fed cattle.

**Prevention.** Pasture mixtures of fescue and legumes appear to cause less trouble than fescue in pure stands. Pasture rotation, clipping and a sound soil fertility program may reduce the incidence of fescue foot.

**Treatment.** Experimentally, thiabendazole has been effective when administered early in the course of the disease. Treatment with this compound is expensive and recommended levels have not been developed. If affected cattle are removed from fescue pasture, protected from extreme cold and supplied with another feed source before dry gangrene has commenced, they usually will recover.

### Grass Tetany

Grass tetany is sometimes referred to as magnesium tetany, hypomagnesemic tetany, transitional tetany, grass staggers, wheat pasture poisoning, lactation tetany and winter tetany. It is a noninfectious disorder of cattle and sheep characterized by abnormally low levels of magnesium and, usually, calcium in the blood. Although this condition can occur in cattle of any age or condition, it most often affects adult, lactating cows grazing grass pastures in winter and early spring. Heifers, cows that

are dry or cows with calves older than 2 months and stocker cattle are less susceptible to grass tetany.

The incidence of grass tetany is influenced by forage species, weather and soil fertility. Forage species differ in their seasonal growth patterns and magnesium contents. Legumes usually have a higher magnesium content than do grasses. The level of magnesium increases in both grasses and legumes during late spring and summer.

Temperature appears to be a factor in that grass tetany usually occurs either during cool weather or following a sharp weather change resulting in a rapid growth of grass. Grass tetany is a threat any time the mean daily temperature is between 40°F and 60°F. After the soil temperature has reached 60°F, the occurrence of grass tetany is low. Although the problem is more common in early spring, it also occurs in fall and winter. The addition of high levels of nitrogen and potassium fertilizers to the soil has increased the incidence of grass tetany.

**Cause.** Tetany may result any time the cattle diet is sufficiently low in magnesium or when, for some reason, magnesium in the ration is insufficiently absorbed or utilized in the body. Contributing factors are age of the animal, lactation status, type of forage, other feed available, weather and content of other elements such as nitrogen and potassium in the diet.

**Symptoms.** Initially, the affected animal usually is nervous with pricked ears, head held high and staring eyes. The gait is stiff and the animal may stagger. Muscle tremors, especially of the face and ears, develop. A blood serum magnesium level below 1.5 percent indicates a high probability of tetany. As the condition progresses, the animal becomes dull in appearance and separates from the herd. Within a few hours or up to several days following onset of symptoms, extreme excitement and convulsions may develop. Following a convulsion, the animal may lie flat on its side with only occasional movement of the forelegs and jaw. Normally, when treatment is delayed or ineffective, death will occur during one of the convulsions or the animal may pass into a coma and die. A chronic form of tetany, characterized by a stiff gait and gradual loss of condition, can persist for a few weeks without apparent depression of appetite or milk yield. This chronic form is followed either by recovery or by the acute form described above.

**Prevention.** Preventive measures primarily are aimed at increasing the magnesium and energy intake of susceptible animals during danger periods. Consider the following management practices to prevent or decrease the incidence of tetany:

- Supply supplemental magnesium to susceptible cattle. Necessary supplemental intake depends on magnesium level in the ration as well as other ration components and susceptibility of animals. Cows

with young calves have a higher magnesium requirement than dry cows and are more susceptible to tetany. The incidence of tetany in young, growing cattle usually is limited to nitrated, small grain or winter annual grass pastures. In herds where history and conditions indicate the high risk of tetany, 1-1/2 to 2 ounces of magnesium oxide should be supplied to each cow in a palatable form. In herds where there is only a mild risk of tetany, sufficient magnesium supplementation can be obtained by providing a magnesium-containing mineral mix that provides 0.33 to 0.5 oz of Mg per day. Mineral containers should be placed in several locations and checked often.

- Graze the more susceptible animals on lower-risk pastures.
- Feed small amounts of hay or grain to cattle on lush pasture during susceptible periods. On cereal grain pastures, it often is best to limit grazing to 2 to 3 hours per day while offering hay, dry pasture or other dry feed during the off-pasture period.
- For short periods of protection, magnesium oxide can be sprayed or dusted on standing forage at the rate of 25 pounds per acre. The duration of benefits is dependent upon the frequency and amount of rain following application. Bentonite slurry permits plants to retain the magnesium longer.
- Test the soil and adjust fertilizer programs to avoid high soil potassium levels. Apply fertilizers based on soil test and use no more potassium than recommended, because grasses are luxury consumers of the element.

**Treatment.** The animal's heart is severely affected by electrolyte changes caused by grass tetany and by effective medication. Treatment, to have a high degree of success, must be prompt and correctly administered. A proprietary solution containing both calcium and magnesium may be given intravenously if administered slowly. If the animal is severely affected, calcium may be given intravenously followed by magnesium subcutaneously. A sedative may be required to control severe tetany or convulsions. Consult a veterinarian familiar with the herd and its management about treatment procedures and the practicability of keeping emergency medication and equipment for administration on hand. To prevent relapse, remove the recovered animal from pasture, feed hay and concentrate, and supplement with 2 ounces of magnesium oxide daily for at least a week.

### **Prussic Acid Poisoning**

The primary cause of hydrocyanic (prussic) acid poisoning in domestic animals is the ingestion of plants containing this potent toxin. Cyanide-producing compounds (cyanogenic glucosides) occurring in living plant



cells are converted to prussic acid when cells are crushed or otherwise ruptured.

The prussic acid potential of plants is affected by species and variety, weather, soil fertility and stage of plant growth. Plants of the sorghum group and leaves of wild cherry trees have a potential for producing toxic levels of prussic acid. There are wide differences among varieties. Some of the sudangrasses, such as Piper, are low in prussic acid. Pearl millet apparently is free of prussic acid in toxic amounts.

**Cause.** Prussic acid is one of the most potent toxins in nature. As ruminants consume plant materials containing cyanide-producing compounds, prussic acid is liberated in the rumen, absorbed into the bloodstream and carried to body tissues where it interferes with oxygen utilization. If toxin is absorbed rapidly enough, the animal soon dies from respiratory paralysis.

**Symptoms.** When lethal amounts are consumed, dead animals may be found without visible symptoms of poisoning. Symptoms from smaller amounts include labored breathing, irregular pulse, frothing at the mouth and staggering.

**Prevention.** Forage species and varieties may be selected for low prussic acid potential. The risk from potentially dangerous forages may be reduced by following certain management practices.

- Graze sorghum or sorghum-cross plants only when they are at least 15 inches tall.
- Do not graze plants during and shortly after drought periods when growth is severely reduced.
- Do not graze wilted plants or plants with young tillers.
- Do not graze for 2 weeks after a non-killing frost.
- Do not graze after a killing frost until plant material is dry (the toxin is usually dissipated within 48 hours).
- Do not graze at night when frost is likely.
- Delay feeding silage 6 to 8 weeks following ensiling.
- Do not allow access to wild cherry leaves whether they are wilted or not. After storms always check pastures for fallen limbs.

**Treatment.** A proprietary sodium nitrite-sodium thiosulfate combination can be administered and repeated once only if necessary. It must be injected intravenously and very slowly; the dosage and method administration are critical. Most animals that live 2 hours after onset of symptoms will recover.

## Nitrate Poisoning

Nitrate and nitrite poisoning may be considered as one entity. When nitrates are ingested, they are reduced

to nitrites before being absorbed from the digestive tract. Nitrate poisoning in ruminants usually occurs as a result of nitrate fertilizer consumption or forage with a high nitrate or nitrite content. Cattle with access to nitrate fertilizers, especially if they are deprived of salt may consume toxic quantities. Cattle consuming plants containing normal amounts of nitrates are able to convert the nitrates to protein.

Few plants normally contain high nitrate levels. Under normal growing conditions, roots of forage plants absorb nitrate from the soil. Shoot tissues convert nitrate into protein about as fast as it is absorbed. Under certain conditions, such as high nitrate fertilization, drought or sudden weather changes, plant species used for pasture, hay or silage can develop potentially dangerous nitrate levels.

**Cause.** Nitrites or nitrates, when consumed more rapidly than they can be converted to protein, enter the bloodstream as nitrite. The absorbed nitrites combine with hemoglobin of red blood cells to produce methemoglobin, a form incapable of transporting oxygen.

**Symptoms.** Oxygen deprivation (asphyxiation) results from tying-up of hemoglobin. Symptoms include rapid, labored breathing; rapid, weak heart beat; staggering; muscle tremors; and pupil dilation. Affected animals may die within an hour after the appearance of symptoms. More often, signs of asphyxiation persist for 3 or 4 hours before death occurs. Animals that are severely affected but recover may develop emphysema and continue to experience difficult breathing for 10 to 14 days. Pregnant cows may abort following recovery from nitrate poisoning. A brown (chocolate)-colored blood is characteristic of nitrate poisoning.

**Prevention.** Store nitrate fertilizer where cattle cannot get to it. Clean up accidental spills if there is any possibility of cattle gaining access to them. Avoid grazing warm season grasses fertilized with high amounts of nitrogen when growth ceases because of drought or cold damage. Cool season grasses and small grain pastures that have been fertilized heavily with nitrogen should be grazed continuously or not at all during early spring when cool, overcast days retard growth. Test suspected forage for nitrate level. Consult your county Extension agent for information concerning sampling, sample preparation and a testing laboratory location. Forage with high nitrate levels can be mixed with forage or grain known to be low in nitrate to reduce the risk from feeding. Cattle have the ability to increase their tolerance to nitrates in their diet. To aid in increasing this tolerance, the diet should be sufficient in energy, vitamin A and the trace minerals normally contained in trace mineral salt.

**Treatment.** Remove animals showing symptoms of nitrate poisoning from the source of toxicity and feed a

high concentrate diet. Mineral oil or other emollients may be given to protect the lining of the digestive tract. A 2 percent solution of methylene blue injected intravenously aids in converting methemoglobin back to hemoglobin.

### **Acute Bovine Pulmonary Emphysema**

Acute bovine pulmonary emphysema (ABPE) is a respiratory disorder associated with sudden changes in the diet of cattle. The disorder most frequently occurs when cattle are changed suddenly from a less to a more palatable forage diet, such as a change from dry feed to lush pasture or from a grass pasture to a pasture containing a high proportion of legumes. While ABPE does not appear to be a major problem, the frequency of occurrences is not known and there is no method of predicting it. Cows that have calved recently appear to be most susceptible.

**Causes.** Causative agents have not been identified. Because ABPE is nearly always associated with feed change, the cause may be some toxic substances derived from the feed that is peculiar to the cattle digestive system.

**Symptoms.** ABPE is characterized by the sudden onset of acute respiratory distress, labored breathing, grunting while exhaling and frothing at the mouth. There may be some frothy nasal discharge and infrequent coughing. A slight elevation in body temperature may be present.

Because of forced expiration, there may be an accumulation of air beneath the skin over the rib and brisket areas. Death may occur as soon as 12 hours but many fatal cases survive until the second or third day. The average mortality rate is about 30 percent. Survivors often have chronic emphysema and are unthrifty. A chronic form may be more slow in onset and the animal may be affected 3 or 4 days before being sufficiently sick for the illness to be recognized. There is an increase in the rate and depth of respiration with frequent deep coughing, a fall in milk yield and a loss of weight.

Complete recovery rarely occurs. Death may not occur for weeks or months and there may be periods of partial recovery during this time. Most affected animals chronically involved are disposed of because of poor condition.

**Prevention and control.** With acute bovine pulmonary emphysema, like other animal diseases without clearly identified causes, the control measures are based on avoiding the circumstances under which the disease is known to occur. The following management and feeding practices may reduce the chance that ABPE will occur:

- Cattle placed on a more succulent pasture or during the first few days on the new pasture should be fed some palatable grass or legume hay.

- Introduce cattle gradually on pastures that contain young green forages. Particularly dangerous is regrowth following a frost or new growth hidden beneath mature plants. Limit access to such a pasture to less than 1 hour the first day and gradually increase the time, taking at least 1 week to reach unrestricted use. If this is impractical or cases of ABPE still occur, mow and windrow the field. If all these practices fail in a particular field, further limit its use for grazing.
- If an outbreak of ABPE has occurred, gently remove all the cattle from that field and place the visibly affected stock in drylot and the remainder of the herd in a field of grain stubble, corn stalks or dry mature grasses. Avoid exciting the cattle. If excited, the afflicted cattle may suddenly die and additional cattle may begin to show symptoms.
- Certain drugs are useful in treating afflicted cattle. Atropine appears to be the most effective. Consult your veterinarian.

Because several different diseases may cause sudden death, often before any animals are observed to be sick, it is advisable to have dead animals posted to establish a diagnosis.

**Treatment.** Initiate treatment immediately in the acute cases without exciting the animal, as any exertion may prove fatal. Remove affected animals from the herd to protect them from jostling or being pushed around by other cattle. Remove grazing cattle from the pasture immediately. If necessary, use a small portable corral to pen the animals separately.

Atropine sulfate administered intravenously may give quick temporary relief. Atropine causes a relaxation of the muscular coat of the bronchi and produces a dilating effect, allowing easier passage of air. The secretions of the nose and throat or bronchi are diminished, thus lessening the amount of fluid in the respiratory tract.

Repeat treatment at 8- to 12-hour intervals, depending upon the response. Antihistamines and cortisone have been used extensively with varying results.

### **Bermudagrass Tremors**

Bermudagrass tremors have been reported in Oklahoma, Arkansas and the Gulf Coast states. One of the biggest outbreaks occurred in 1971 in Louisiana, affecting more than 25,000 cattle in more than 500 herds and resulting in the death of more than 600 animals.

The problem occurs where cattle graze bermuda pastures that have been allowed to grow tall and rank. Tremors have been observed mostly during the fall and winter months following a period of cloudy, damp, humid weather that is thought to promote the growth

of toxin-producing fungi. Hay cut from problem pastures also has remained toxic even after a year of storage. The disease seems to affect only cattle.

**Cause.** The causative agent has not been specifically identified, but is suspected to be toxin-producing fungi whose growth on forage is favored by cool, cloudy, damp, humid conditions.

**Symptoms.** Symptoms are similar to ergot poisoning. Infected cattle twitch, tremble, become stiff legged in the hind quarters, weak in the front legs and poorly coordinated. When excited, cattle may fall to their knees. Down cattle frequently will not get up when prodded, but may later get up on their own. Animals remain alert and continue to eat if feed is available. Rapid loss of weight and condition are frequently observed.

Deaths generally have been associated with secondary causes. Down and weakened cattle are susceptible to pneumonia, and to dehydration from not being able to reach water. Affected cattle that make it to water frequently drown.

**Prevention.** Manage pastures to prevent tall rank growth that tends to fall over and matt, retaining moisture during wet weather. No problem has been observed on short-grass pastures beside problem pastures containing rank, old growth.

**Treatment.** Animals recover readily when removed from toxic pastures or hay and fed grain and nontoxic forage.

### **Ergot Poisoning**

Ergot is a parasitic fungus that grows in the seed heads of small grains and pasture grasses such as ryegrass, dallisgrass and bahiagrass. Ergot poisoning in cattle is most commonly observed on pastures containing a high percentage of dallisgrass. A toxin produced by ergot is extremely poisonous to livestock, especially cattle.

**Symptoms.** The toxin produced by ergot interferes with circulation, resulting in reduced blood flow to the extremities of the limbs and tail. Lameness is often an early symptom, with a sloughing of the tip of the tail and possibly the feet if cattle continue to consume the ergot. The central nervous system can be stimulated and then depressed. Elevated body temperature and increased respiratory or pulse rate may also be observed.

**Prevention.** Because ergot grows primarily in seedheads, pasture clipping and grazing management that retards or prevents seedhead development should be practiced.

**Treatment.** Change cattle immediately to an ergot-free diet.