Preventing Forage Related Disorders

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Bloat

Bloat refers to excessive accumulation of gas in the rumen. Bloat results when an animal can not eructate or "belch up" gases produced in the process of rumen fermentation. The gas may be in the free form or may be mixed with rumen contents in the form of froth. It occurs both on pasture and in feedlots and can be a major cause of death in cattle wherever intensive farming is practiced. Additional losses can include decreased milk production and reduced rate of gain. Although legumes may increase the opportunity for bloat to occur, fear of bloat should not keep you from using high quality legumes, such as alfalfa and clover, in your pasture. Reducing the endophyte effects of fescue with legumes is important for improved animal performance.

Pasture bloat (Legume) Frothy bloat results from the production of a stable foam that does not allow gas bubbles to form free gas and be "belched" off. It usually occurs in cattle grazing lush legumes, such as alfalfa, ladino, or other white clovers. The danger of bloat is greatest when pasture plants are young, lush, and high in soluble protein. The disorder is due to the foaming properties of soluble leaf proteins, which are more prevalent in legumes.

The essential feature is that coalescence of the small gas bubbles is inhibited and intraruminal pressure increases because eructation (belching) cannot occur. This condition can arise from diets of lush legumes or winter wheat pasture; it may be seen with high-concentrate finishing ratios in the feedlot. Of the commonly grown legumes, alfalfa and ladino clover are classified as higher bloat risk, red clovers are moderate risk, and crimson and subterranean clovers as lower risk.

Current research supports both animal and plant characteristics as predisposing to legume bloat. Research has shown that some cows can be classified according to their susceptibility to pasture bloat into high or low susceptibility and the offspring show influences of their parentage. A number of inherited characteristics are related to bloat. Individual cattle have been classified as having either high or low susceptibility to legume bloat. High susceptible cattle have larger rumen volumes than non-bloating animals. There is a slower rate of passage of particles from the rumen in bloating cows. In a research study, cattle that bloated on a given day consumed 18 to 25% less alfalfa immediately before bloat than non-bloaters did in the same time period.

Adaptation of animals to a particular feed is an important factor. Animals may be at increased susceptibility for the short-term due to changes in rumen microflora. As animals become adjusted to a particular pasture or ration, they have less susceptibility to bloat. The claim that the risk of bloat may be reduced by waiting until the dew is off the alfalfa is true. A study demonstrated that cattle were 2 to 17 times more often likely to bloat when alfalfa grazing began between 7 and 8 AM compared to beginning 4 hours later.

There is a relationship between plant factors associated with bloat and the rapidity with which leaf structure is disrupted after ingestion. Bloat-inducing plants are more readily macerated, thus providing quicker bacterial access to the inner leaf cells. Spring (mid-March to mid-May) is the most dangerous season when the pastures are lush, young and the leaves of the plant contain high concentrations of soluble proteins. As the legume plant matures to early bloom stage, the fiber content increases and the risk for bloat decreases.

Legume bloat may resolve spontaneously if the animal stops consuming the bloat-producing feed and microbial digestion eliminates the froth-stabilizing factors.

Free-gas bloat It is usually necessary to pass a stomach tube to differentiate free gas from frothy bloat. A stomach tube will deflate the free gas bloat. Milk fever can cause free-gas bloat, but the most common cause is when an animal has become cast on its side and cannot belch.

Diagnosis A preliminary diagnosis is usually based on observation of the distended abdomen with extreme tightness of skin and distortion in the upper left side. It is usually necessary to pass a 3/4 inch to 1-inch rubber hose or stomach tube to differentiate free gas from frothy bloat. If it is pasture bloat, a stomach tube is usually not adequate.

The condition may be fatal if the distension is extreme enough to compromise ventilation by compressing on the lungs. As the rumen enlarges and compresses on the diaphragm, breathing becomes more labored. Open-mouth breathing and collapse leading to death may occur within a few minutes if the animal becomes frantic from the abdominal pain and problem breathing. Cattle that die of most causes will bloat with free gas after death. Therefore, an animal found dead with bloat is not diagnostic of bloat.

Treatment Bloated cattle should **not** be removed from legumes at the first sign of bloat. Cattle can adjust to the pasture. Only remove the ones in obvious distress who require treatment. The risk of bloat is substantially lower when cattle graze alfalfa continuously than when grazing is interrupted and cattle are allowed to graze for only 6 hours daily (Table 1).

Table 1. Effect of feeding on the incidence of bloat in cattle grazing alfalfa

	Grazing System	
	Continuous	Graze 6 hours per day
	----- Number of cases -----	
Week 1	1	25
Week 2	4	16
Total	5	41

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With frothy bloat, the stability of the foam must be reduced before the gas can be removed. Anti-foaming agents such as Bloat-Pac® or Bloat Treatment® (DSS, dioctyl sodium sulfosuccinate) can be used as a drench or via a 3/4 inch to 1-inch stomach tube passed into the rumen. If no product is available, one may drench with 1 ounce (30 ml) of a non-toxic household dish detergent liquid in 1 liter of water. Any vegetable oil is also useful in reducing the foam. Turpentine is an old treatment that is reasonably effective but is highly irritating to the tissues and also imparts undesirable flavors to meat and milk.

As a last resort in emergencies, pending the arrival of the veterinarian, relief can be obtained by making a hole in the rumen large enough to release the foam. The incision should be made on the left side, halfway between the last rib and the hook bone/hipbone. A trochar with cannula, or knife can be used. The incision will need to be sutured, and antibiotics must be administered.

Prevention A very effective method of bloat prevention is to provide molasses-mineral blocks or pre-mix containing Bloat Guard® (poloxalene), which is a detergent-type compound that reduces development of stable foam in the rumen (Table 2). Although blocks are relatively expensive, any of these poloxalene compounds will eliminate bloat and prevent animal deaths if consumed at the appropriate dose when grazing high risk pastures. Generally, bloat control is needed in early spring and not necessary once the grazing season progresses.

Feeding an ionophore can also reduce the potential for bloat. Monensin (Rumensin®) can greatly reduce incidence and severity of pasture bloat but it will not eliminate the problem (Table 2). Some animals do not respond as well as others. Lasolacid (Bovatec®) does not have a beneficial effect in pasture bloat reduction; Bovatec® does have a significant effect in grain bloat reduction. Monensin inhibits rumen protozoa that normally produce gas and protein foam-stabilizing substances, thus leading to a reduction in the bloat potential. To be effective, these materials must be consumed by the animal the same day that the bloat-causing pasture is grazed and started just prior to bloat season in order to acclimatize the microbial population.

Table 2. Effect of feeding Monensin, Lasalocid, or Poloxalene on Alfalfa Pasture Bloat

Treatment Product	Dose, mg/lb body weight	% Reduction in bloat
Monensin[a]	0.3 0.6	71 72
Lasolacid[a]	0.3 0.6	30 16
Monensin[b]	0.3 0.45	41 73
Lasolacid[b]	0.3 0.45	25 12
Polaxalene[a,b]	20	100

[a] J. Anim. Sci. 1983. 56:1400-1406; [b] J. Anim. Sci. 1986. 63:1246-1257

Other management practices that also can help prevent bloat:

- Avoid first grazing in early morning.
- Once cattle are turned onto pasture, do NOT remove them at the first signs of bloat. Mild subacute bloat cases occur frequently on alfalfa pasture, unless a bloat preventive is fed.
- Provide a grass-legume mixture for pasture.
- Feed grain or a grain-roughage mixture to reduce pasture intake.
- Concentrate on making hay from high risk pastures and utilize safer fields for grazing during the early spring.
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Acute Bovine Pulmonary Emphysema

Acute Bovine Pulmonary Emphysema (ABPE) is a disorder also known as Fog Fever and Atypical Interstitial Pneumonia. It is actually a nutritional disorder manifested by the sudden onset of acute respiratory distress. Symptoms may include very labored breathing, grunting while exhaling, and frothing at the mouth. A slight elevation in body temperature may occur. Death may occur very quickly with mortality rates averaging as much as 30%. Complete recovery is not likely because lung tissue damage may have occurred. Later stress such as handling the cattle for working may cause relapse and death at this time.

ABPE occurs most often when cattle are moved from a well grazed pasture to an area of lush, succulent growth. The higher quality the pasture moved into, the more often the disorder will be seen. The disorder is caused by the formation of 3-methylindole (3MI) in the rumen, absorption and metabolism in the lungs. Rumen bacteria produce 3MI from the natural catabolism of the amino acid tryptophan. Improved pastures with higher protein levels naturally provide greater sources of tryptophan than lower quality pastures.

Many rumen bacteria break down tryptophan. Only one, a species of Lactobacillus, can actually form 3-MI in the rumen. Research into the disorder has centered on methods of altering the ruminal bacteria population in order to prevent 3-MI from being formed. Research has shown that both Monensin and Lasalocid can prevent the production of 3-MI in the rumen, thus reducing the incidence of ABPE in cattle (tables 3 and 4).

Table 3. Effect of Monensin and Lasalocid on Prevention of Acute Bovine Pulmonary Emphysema

Group	Treatment/Dose	Clinical signs	Lung Lesions	Mortality
1	M 100 mg, 2X	0/4	0/4	0/4
2	M 200 mg, 1X	0/4	0/4	0/4
3	L 100 mg, 2X	2/4	4/4	1/4
4	Control	4/4	4/4	1/4

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Table 4. Effect of Lasalocid on Prevention of Acute Bovine Pulmonary Emphysema

Group	Dose, mg/hd/day	Clinical signs	Mortality
1	0	5/5	3/5
2	200	0/5	0/5
3	400	0/5	0/5
4	600	0/5	0/5

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Producers must think in terms of prevention and not treatment of this disorder. The only practical prevention is to continuously feed one of the ionophores (monensin, lasolacid) during the risk of ABPE. If an outbreak does occur, treatment must be immediate for satisfactory results. Affected cattle must be removed from the pasture at once. This must be done very gently and carefully or fatalities may occur from the stress of movement. Check with your local veterinarian for drugs to provide relief in breathing. Chronics should be culled.

Nitrate Toxicity

Nitrate toxicity may affect cattle consuming forages containing an excessive amount of nitrate. It may also occur if animals (especially those hungry for salt) have access to nitrate fertilizer. Consumption of high levels of nitrates will poison cattle. When cattle consume nitrate, nitrate is reduced to nitrite and eventually to ammonia by bacteria in the rumen. Nitrite rather than nitrate is actually the toxic entity. The conversion of nitrate to nitrite is faster than the conversion of nitrite to ammonia thus allowing for a buildup of nitrite when large amounts of nitrate are consumed. The nitrite can be absorbed into the blood stream and combine with hemoglobin in the red blood cell. This produces methemoglobin which is incapable of transporting oxygen.

Symptoms can vary but will generally include:

- rapid and labored breathing
- rapid and weak heart beat
- muscle tremors
- staggering
- cyanosis (darkening of mucous membranes due to oxygen deprivation; brown)

Death may occur from asphyxiation anytime within the first 3 to 4 hours from onset of symptoms. Animals showing symptoms should be removed from the feed or pasture and fed a high concentrate diet. Treatment of affected cattle by a veterinarian with intravenous injection of methylene blue solution can be effective. Pregnant animals that do recover may abort the fetus.

Forage crops most likely to collect nitrates are warm season annual grasses such as sorghum, sorghum-sudan hybrids, sudangrass, corn and johnsongrass. Avoid grazing these warm season grasses, especially those heavily fertilized with high amounts of nitrogen, when growth ceases due to drought or cold damage. Suspect forage should be tested for nitrate level. Consult your County Extension Agent for

Agriculture or veterinarian for information concerning sampling and how to send samples to a diagnostic lab.

Cattle can adjust to higher than normal nitrate levels without problems if they are slowly introduced to higher levels. High nitrate levels can be diluted with carbohydrates (grain) and this will reduce the toxic potential of the forage. Harvesting high nitrate forages as hay will not reduce the toxic potential over time. Harvesting as silage will reduce the nitrate level through fermentation by 40-60%.

Proper management will avoid most situations that could lead to nitrate toxicity, including to prohibit cattle access to nitrate fertilizer. The following weeds can accumulate toxic levels of nitrate: Johnson grass, pigweed, common lambs quarter, wild sunflower, Canada thistle, black nightshade, jimson weed and barnyard grass. Nitrate concentrations peak in these weeds at prebud to bud and decrease as they mature. Do not let cattle have access to fields with a lot of these weeds.

Table 5 provides interpretation for laboratory results. Results are generally reported on a percentage or parts per million (ppm) of nitrate on a dry basis. Be sure results are reported as nitrate levels.

Table 5. Nitrate Levels in Forages (dry matter basis)

% Nitrate in Dry Matter	PPM Nitrate in Dry Matter	Comments
0 to 0.44	0 to 4,400	Safe to feed. Be cautious with pregnant and young animals at upper level of range
0.44 to 0.88	4,400 to 8,800	Generally safe when fed with a balanced ration. For pregnant animals limit to one-half of total dry ration. Be sure water is low in nitrates. Prolonged feeding may result in a Vitamin A deficiency. Do not feed with non-protein (NPN) supplements. Be cautious with pregnant and young animals
0.88 to 1.50	8,800 to 15,000	Limit to one-fourth of ration. Fortify well with energy, minerals, and Vitamin A. May experience decreased milk production in 405 days, possible occurrence of reproductive problems.
Over 1.50	Over 15,000	TOXIC. Do not feed. Sudden death, abortion, severe depression, and difficult breathing may occur.

Grass Tetany

Grass tetany (hypomagnesemia, grass staggers, winter tetany, barley poisoning) is a disorder caused by an abnormally low amount of magnesium in the animal's blood. Beef cattle producers in Kentucky have generally been successful in reducing the incidence of tetany. However, the potential still exists for this disorder to be a problem in most herds.

Grass tetany occurs most often in cows grazing lush spring forages, especially small grains and cool season perennials such as fescue. It is most common in spring-calving cows, especially if they are high producers in their third to fifth lactation. Several factors contribute to the increased incidence of tetany at this time. The magnesium requirement of cows doubles from late gestation to early lactation (from 9 grams to 21-22 grams). When this rapid change in magnesium needed by the cow is coupled with lowered magnesium in the plant, along with certain components which lower the availability of magnesium (such as high applications of nitrogen and potassium fertilizers), tetany may develop. Weather may also have an effect; the greatest threat is when temperatures are between 40 º and 60 º F. Temperatures in excess of 60 º F for a week markedly decrease the incidence of tetany. When all of these factors are combined, the risk can be very high.

Cattle affected with grass tetany may isolate themselves from the herd and stagger. As the disease progresses, they may exhibit extreme nervousness, rapid breathing, and muscle trembling. They may become aggressive and charge anyone in the pasture. In the most severe stage, the animal collapses to the ground with muscular spasms. Treatment must be given rapidly as death can occur within an hour after the onset of convulsions.

For the cow down with tetany, treatment is the only option. Treatment consists of an intravenous injection of solutions containing magnesium, calcium, and glucose. This must be done correctly! If this IV solution is administered too rapidly it may result in death. Consult a veterinarian familiar with the herd and its management about treatment procedures and whether you should keep emergency medication and equipment on hand. To prevent relapse, recovered animals should be removed from the pasture and fed a hay/concentrate mixture supplemented with magnesium oxide for at least a week.

As a producer, you should be concerned with preventing tetany. About 2 ounces of magnesium oxide (22 grams of magnesium) is recommended to meet the magnesium needs of lactating beef cows. Since legumes are higher in magnesium than grasses, feeding cows legume hay during the early spring may supply some magnesium. Cows grazing spring grass pasture should have magnesium in the mineral mixture; in "high risk" situations, it may be supplied in a supplement.

Many commercial mixtures are available in various forms to prevent tetany. Before you make a purchase, you must decide if the product will give adequate magnesium intake. This depends on the magnesium content and the expected consumption of the product. Both should be listed on the tag. If it appears that magnesium intake will not be adequate, then a product with more magnesium or greater intake should be used. In "high risk" situations where tetany is a frequent problem, it may be necessary to force feed the daily magnesium needs. Magnesium oxide can be included in grain or protein supplement.

Cyanide Toxicity

Cyanide or prussic acid poisoning occurs when animals ingest plants containing large amounts of cyanogenic glycosides that yield hydrocyanic (prussic) acid when digested in the rumen. Plants common to grazing situations that contain these compounds are sudan, sorghum-sudan crosses and sorghums such as Johnson grass. Many other plants also contain the compounds (e.g. wild cherry) but are not common

for grazing. Conditions that enhance the toxic possibilities are high nitrogen fertilization, wilting, frost, and plant disease. Very young, rapidly growing plants contain higher levels of toxic compounds than more mature plants. Some sudan grasses are low in prussic acid; Pearl millet is free of toxic amounts of prussic acid.

Hydrocyanic acid stops cellular respiration. Death may occur very quickly from respiratory paralysis. If the animal survives for 2 hours after onset of symptoms, recovery generally occurs. Treatment can be effective if started in time; however, animals are often found dead before symptoms are observed.

Prevention must be the primary course of action. Select varieties of the affecting forages that are low in cyanogenic glycosides or use other forages that are free of these compounds. You can lower the risk of prussic acid poisoning by following these management practices:

- Don't graze sorghum or sorghum-cross plants until they are at least 15 inches tall.
- Don't graze wilted plants.
- Don't graze these plants during or shortly after drought periods when growth is retarded.
- Don't graze for two weeks after a non-killing frost.
- Don't graze until about 48 hours after a killing frost (until plant material is dry).
- Don't graze at night when a frost is forecast.
- Don't allow cattle access to wild cherry leaves.
- Do check pastures after storms for fallen wild cherry trees or limbs.

Poisonous Plants

Taxus (Japanese Yew)

Taxus is an ornamental shrub generally around foundations. Livestock will really consume trimmings or prunings, especially when hungry. Cardiac failure is produced quickly after consumption which leads to death. There is no treatment. Most common poisonous plant diagnosed at the Livestock Disease Diagnostic Center to cause death.

Oak/ Acorns

In the autumn after a dry summer, acorns can be plentiful. Cattle, especially calves, grazing near oak trees begin supplementing their diet with acorns and poison themselves. Signs include constipation, followed by diarrhea that sometimes includes blood. The affected animal may appear dull and depressed. In mild cases, cattle can recover. In severe cases, the kidneys are irreversibly damaged and the animal dies within several days. Treatment is not usually rewarding in severe cases. Oak poisoning has been prevented experimentally by mixing 15% calcium hydroxide in the pelleted ration.

Buckeye

Cattle are affected by consuming young shoots and leaves in the early springtime or mature seed in the fall. Generally, symptoms of drunkenness are observed. Trembling, muscular weakness and incoordination are commonly seen. Paralysis or coma may follow. There is no specific treatment.

Osage orange (hedgeapple)

The large hedgeapple causes asphyxiation due to mechanical blockage of the throat or trachea.