

Bloat

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Most cases of bloat are caused by a stable foam which covers the cardia of the stomach. Dougherty and Habel demonstrated in 1955 that the cranial esophageal sphincter would not permit foam to escape into the pharynx, thus blocking eructation when foam fills the esophagus (1). The observation that some bloated ruminants have a cap of free gas in the rumen and can be "Let down" with a stomach tube or trocar does not preclude the fact that a stable foam covers the cardia of the stomach (2).

Not all bloat is caused by a stable foam. Other causes are vagal nerve damage, esophageal obstruction, pressure on the esophagus as from a tumor, factors that interfere with eructation such as pneumonia or diphtheria, alkalosis as from urea (ammonia) poisoning, founder, cyanide and hydrogen sulfide poisoning.

Components of Foam

The chief components of a stable foam are protein, pectins, mucopolysaccharides and saponins.

Soluble protein having a high molecular weight of about 550,000 derived from either feed or microorganisms produces a foam when gases bubble through it (3). Once the soluble protein is whipped into a froth, the protein at the gas-water interphase becomes denatured and stiffens similar to the beaten egg white on a meringue pie. The smaller and more numerous the bubbles, the more surface is presented, the more protein is denatured and the more stable the foam becomes. The denatured protein is more resistant to bacterial degradation than the soluble protein, thus slowing bacterial destruction of the foam (4).

The legumes which cause bloat are rich in high molecular weight, soluble protein (3). Some of the legumes that rarely cause bloat contain tannins which precipitate the protein. The tannins in alfalfa do not precipitate protein (5).

Protozoa and bacteria produce considerable protein. Slime produced by encapsulated bacteria may contain 33% protein on a dry weight basis (6).

Protein will not be very soluble and it will not become denatured at the gas-water interphase unless the pH is near the isoelectric point. This is different for each protein, but is about 5.5 to 5.8 (7,8). This degree of acidity destroys the Holotrich protozoa thus releasing their protein and polysaccharides. As a rule bloaters will have more acid rumen contents than non-bloaters (9).

Immature, rapidly growing vegetation and molasses are rich in organic acids. Rumen microorganisms contribute to the acidity. High concentrate rations may need an added base such as dolomite or limestone to prevent excess rumen acidity and bloat (10). One of the methods by which saliva prevents bloat is by the buffering action of bicarbonate and phosphates (11).

Immature, green legumes are rich in pectin and pectin methyl esterase. Both increase markedly on high incidence days (12). Pectins are degraded to pectinic and polygalacturonic acids which increase viscosity of the rumen contents and have gelling properties.

The encapsulated Streptococci produce slime which contains 18% polysaccharides. The percentage of encapsulated rumen microorganisms increases with time as the cattle are on full feed (13). These organisms also increase rapidly in number on a bloat producing high concentrate ration. In one experiment by Gutierrez and Davis full fed steers with no bloat had no slime in the rumen contents, animals with slight bloat had 72 mg of slime per 20 mls of rumen fluid and severe bloaters had 90 mg of slime per 20 mls of rumen fluid (6).

Saponins are natural foam producers. At high concentrations they poison the rumen causing bloat by rumen atony (14). They probably never reach a high concentration as they are readily decomposed by rumen bacteria with protein and polysaccharides produced as by-products (15). The latter role is the most likely method by which saponins contribute to bloat.

Viscosity

Formation of most types of froth is dependent upon a high consistency of the ruminal ingesta. Most animals that bloat have no liquid phase in the rumen (16). If the ruminal contents are watery, gas bubbles readily coalesce, rise to the surface rapidly, not much protein is denatured on the surface of gas bubbles and a stable foam is not apt to develop (17).

Consistency of the ruminal ingesta is increased when the rate of salivation is decreased (18). Balch showed that the cow secretes 43 to 57 pounds of saliva while eating 10 lbs of dry hay, but only 12 to 15 pounds of saliva is secreted while eating 10 pounds of concentrates (19). Coarse roughage in the rumen is another important stimulus for salivation. One reason that the gag or wooden bit placed in the mouth

of a bloated cow may give relief is because of the increased rate of salivation.

Eating succulent legumes which contain much water does not increase the amount of free fluid in the rumen. Instead, the viscosity or consistency index is considerably increased. Weiss measured the consistency index by pulling a bulb through a column of rumen ingesta. The consistency index after feeding mature plants or stalks was five to ten. It was 15 to 66 after feeding succulent plants. Bloat occurred when the index went over 50 (18). Cattle that eat rapidly growing legumes drink very little water (20).

The viscosity of the rumen contents is high in cattle bloated on feedlot rations. Little or no free rumen water is present. The high viscosity is associated with a low pH plus slime produced by rumen microorganisms (8).

One method of treating or controlling feedlot bloat until the ration can be corrected is to add 4% salt to the ration (21). The increased water intake probably reduces the consistency of the rumen contents to prevent the formation of a stable foam.

Gas Formation

It is difficult to measure precisely the amount of gas produced in the rumen because carbon dioxide is readily absorbed through the rumen wall and expired via the lungs. Some gas may pass into the intestine. Both methane and carbon dioxide are trapped in air bubbles, all of which preclude accurate measurement. It is estimated that two liters of gas per minute may be produced in the rumen. The cow's capacity to eructate gas is at least three times this amount, providing the vagus is intact and there is no obstruction or stable foam covering the esophageal orifice. Eructation is stimulated by fiber. However, simple rumen distention provides plenty of stimulus for eructation with or without fiber. It is difficult to overload the rumen or to elevate the hind quarters sufficiently to prevent eructation of gas (22). Thus the basic problem is a stable foam and not excess gas formation per se. On the other hand, there will be neither bloat nor foam without reasonably rapid gas formation.

The site of gas production in the rumen may be important. Legumes and concentrates sink to the floor of the rumen so gas bubbles are forced to rise through the rumen fluid contributing to the foaming (23).

Legumes are rich in citric, malic (malonic) and succinic acids. These acids react quickly with the bicarbonate initially present in the rumen to release large quantities of carbon dioxide. These acids are further readily reduced by bacterial action to contribute 40 to 60 percent of the total gas production (24).

Genetics

It is common knowledge that some cattle are more susceptible to bloat than others, but the factors responsible for resistance have not been clearly defined. Bloaters have more volatile fatty acids after graz-

ing ladino than non-bloaters (25). The amount of free fluid maintained in the rumen and the pH of the rumen are important factors in bloat susceptibility. Both of these are regulated to a degree by the amount and rate of saliva production.

Prevention

Foamy bloat can be prevented by feeding one to two grams of Poloxalene per 100 lbs of body weight per day. The less mature the legume the more apt it is to produce bloat and the more difficult it is to get cattle to eat a supplement containing Poloxalene. It may not be possible to safely graze some cattle on alfalfa before it reaches the 1/10th bloom stage. If Poloxalene is administered in a grain supplement, the animals should be fed the supplement daily before being turned to pasture or if they are given the grain free choice they should be accustomed to the supplement before they are turned to pasture. If Poloxalene is placed in molasses and administered by a lick-wheel there should be one lick-wheel for every 25 cattle. The lick-wheels should be placed near waterers and the cattle should be confined so they are never more than 400 yards from the lick trough. This will probably require daily fencing. If the Poloxalene is administered in blocks, five blocks should be available for every 25 cows (26,27).

Laby's capsules are being tested in Australia and New Zealand. A plastic capsule containing a detergent in an ethyl cellulose gel is administered by tube into the rumen. The detergent slowly diffuses into the rumen fluid and is effective for several weeks (28).

Cheng and Hironaka experimentally fed the same high concentrate feed to two groups of cattle. One group received the feed finely ground and the other coarsely ground. The viscosity of the rumen contents was twice as high in the cattle getting the fine feed. The pH of the rumen contents of the cattle getting the fine feed dropped to 5.6, but did not go below six on the cattle getting the coarse grind. A severe frothy condition developed in the rumens of the cattle on the fine grind (8).

Rations composed of finely ground feed improves digestability for feedlot cattle, but the fine grind is a major cause of bloat. Hironaka and Cheng indicate that feedlot bloat can be controlled and good digestability maintained by putting a three-quarter inch garden hose to a grain auger to add 8% water. Allow the grain to soak a few hours and then roll it. The same thing can be accomplished by using high moisture or acid preserved corn. The larger particle size of the feed reduces slime production, prevents excess acidity in the rumen and thus precludes the formation of a stable foam (29).

Bloat which occurs in young bucket fed calves can often be corrected by feeding in such a manner that less milk spills into the rumen. This can be done by using nipples that do not deliver milk so fast, feeding calves more often and supplying them with drinking water (30).

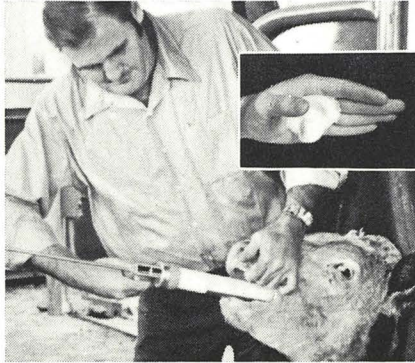
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For a satisfactory diagnosis, a microscopic fecal examination should be performed prior to worming.

DOSAGE OMNIZOLE Wormer Paste is given orally to cattle. The dosage is proportional to body weight and also depends on the severity of infection. The routine dose of thiabendazole for *Trichostrongylus* sp, *Haemonchus* sp, *Nematodirus* sp, *Ostertagia* sp, and *Oesophagostomum radiatum* is 3 g per 100 lb body weight. For *Cooperia* sp, or severe infections with the other species, give 5 g per 100 lb. For most effective results, severely parasitized animals or those constantly exposed to helminth infection should be re-treated every two to three weeks.

Each full depression (3 "clicks") of the dosing-gun trigger delivers one dose containing 7.5 g thiabendazole. Administer OMNIZOLE Wormer Paste as follows:

For routine worming, give one dose for each 250 lb body weight.
For *Cooperia* sp, or severe infections with the other species, give one dose for each 150 lb.

WARNING Milk taken from treated animals within 96 hours (8 milkings) after the latest treatment must not be used for food. Do not treat cattle within 3 days of slaughter.

*Genera *Trichostrongylus* sp, *Haemonchus* sp, *Nematodirus* sp, *Ostertagia* sp, *Oesophagostomum radiatum*, and *Cooperia* species

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Lesions and Signs

Depending upon the degree of abdominal muscling and the length of the "coupling" some animals will show evidence of bloat with as little as 13.6 mm of Hg pressure. Some die at 55 to 60 mm of Hg pressure (2). Others will maintain this degree of pressure for an hour and recover spontaneously. Few survive pressures of 70 mm of Hg for long. However, equal pressures produced artificially with air or inert gas do not cause death, suggesting that absorbed rumen contents are a factor in the cause of death. Very high blood pressure, increased cerebral spinal fluid pressure and respiratory embarrassment are additional contributors to the cause of death (31).

Animals that die of bloat have the following lesions which help differentiate bloat from other causes of sudden death, many of which result in post mortem rumen distention. The foam which was present at the time of death may or may not be present at the time of necropsy. There is usually little or no free fluid in the rumen. Portions of the intestine are distended with gas. The muscles of the hind limbs are pale and bloodless and are separated by edema. The prefemoral and popliteal lymph nodes are edematous and ischemic. In contrast, the prescapular, axillary and cervical lymph nodes are very congested. The congestion, hemorrhage and edema seen in the submucosa of the trachea, fascia and muscles of the cervical area seen in many sudden death cases will be more severe and occur more quickly after death in cattle dying of bloat. The mucosa of the cervical esophagus is dark red. The mucosa of the thoracic esophagus is often blanched with a relatively sharp line of demarkation being at the thoracic inlet (31).

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