Bloat

What is bloat?

Bloat is simply the buildup of gas in the rumen. This gas is produced as part of the normal process of digestion, and is normally lost by belching (eructation). Bloat occurs when this loss of gas is prevented. There are two sorts of bloat.

1. Gassy bloat
which occurs when the gullet is obstructed (often by foreign objects such) or when the animal can't.
2. Frothy bloat
which happens as the result of a stable foam developing on top of the rumen liquid, which blocks the release of the gas. This is by far the most common form of bloat, and unlike gassy bloat, it is highly seasonal with peaks in the spring and autumn. This is because the foam is formed by breakdown products from rapidly growing forages (particularly legumes such as clover and alfalfa). These increase the viscosity (stickiness) of the rumen fluid and prevent the small bubbles of gas formed by rumen fermentation from coming together to form free gas that can be belched off

Clinical Signs

1) Distended left abdomen is the most obvious sign
2) Usually associated with pain, discomfort, and bellowing.
3) Death can occur within 15 minutes after the development of bloat
4) Gaseous bloat is usually seen in one or two animals. Frothy bloat can affect up to 25% of cases
5) In some cases, sudden death may be the first sign seen by the stockman, although in such cases it is likely that there will be other cattle with bloat that are still alive.

Treatment

Passing a stomach tube is the best treatment for gassy bloat. Once the gas has been released, the cause of the obstruction should be looked for.

In a few cases a trochar and cannula punched through the side into the rumen will relieve gassy bloat when a stomach tube has not worked. But such cases are rare, and as the trochar provides a tremendous opportunity for introduction of infection, it should only be used as a last resort.

For frothy bloat, antifoaming agents that disperse the foam should be given by stomach tube. Old-fashioned remedies such as linseed oil and turpentine are effective but newer treatments such as dimethicone or polaxolene are easier to give as the effective dose is much smaller.

If an outbreak of frothy bloat occurs all cattle on that pasture should be removed immediately and put onto a high fibre diet (hay or straw), and any cows showing bloating signs treated with an anti-foaming agent. The pasture should not be grazed for at least ten days.

Rumen Acidosis

Rumen acidosis is a metabolic disease of cattle. Like most metabolic diseases it is important to remember that for every cow that shows clinical signs, there will be several more which are affected sub-clinically.

Acidosis is said to occur when the pH of the rumen falls to less than 5.5 (normal is 6.5 to 7.0). In many cases the pH can fall even lower. The fall in pH has two effects.

Firstly, the rumen stops moving, becoming atonic. This depresses appetite and production.

Secondly, the change in acidity changes the rumen flora, with acid-producing bacteria taking over. They produce more acid, making the acidosis worse. The increased acid is then absorbed through the rumen wall, causing metabolic acidosis, which in severe cases can lead to shock and death.

Cause

The primary cause of acidosis is feeding a high level of rapidly digestible carbohydrate, such as barley and other cereals. Acute acidosis, often resulting in death, is most commonly seen in ‘barley beef’ animals where cattle have obtained access to excess feed. In dairy cattle, a milder form, sub-acute acidosis, is seen as a result of feeding increased concentrates compared to forage.

Symptoms

Acute acidosis often results in death, although illness and liver abscesses may be seen beforehand. Cattle may become depressed, go off feed, have an elevated heart rate or diarrhea.

Sub-acute:

1) Reduced feed intake
2) Poor body condition and weight loss
3) Unexplained diarrhea
4) Temperature
5) Pulse rate and respiratory rate may rise
6) Lethargy

Treatment

Because subacute ruminal acidosis is not detected at the time of depressed ruminal pH, there is no specific treatment for it. Secondary conditions may be treated as needed.

Supplementing the diet with direct-fed microbials that enhance lactate utilizers in the rumen may reduce the risk of subacute ruminal acidosis. Yeasts, propionobacteria, lactobacilli, and enterococci have been used for this purpose. Ionophore (eg, monensin sodium) supplementation may also reduce the risk by selectively inhibiting ruminal lactate producers.

White Muscle Disease

White muscle disease is also known as nutritional myopathy of calves. It is normally is seen in young calves and is associated with deficiencies of selenium or vitamin E, or both.

Cause

There are two forms of white muscle disease; a congenital form that affects the cardiac muscle, and a delayed form that is associated with either cardiac or skeletal muscle.

Symptoms

Calves affected by the congenital form of white muscle disease usually die within 2-3 days of birth due to cardiac muscle degeneration.

On examination post death, the heart will show white, chalky subendochondral plaques that are most noticeable in the left ventricle. The result is damage to cardiac muscle cells and Purkinje fibers.

Cattle affected by the delayed form or white muscle disease may exhibit signs ranging from general unthrift and stiffness, to walking with an arched back and spending more time recumbent, depending on the level of selenium in the diet.

Often, the delayed form is brought on by vigorous exercise but if chronically affected, cattle can display splayed toes and a relaxation to the shoulder girdle. If a calf is affected severely it may die of starvation due to an inability to nurse properly due to weakness. The skeletal muscle lesions associated with the delayed from of white muscle disease are usually bilaterally symmetrical and can affect one or more muscle groups. The muscle will have white striations and feel dry and chalky due to abnormal calcium deposits.

Treatment

Cattle affected by white muscle disease have been treated with sodium selenite and vitamin E in sterile emulsion. This can be administered SC or IM, at 1 mg selenium and 50 mg (68 IU) of vitamin E per 18 kg (40 lb) body wt.

If necessary, the treatment may be repeated two weeks later, but no more than four doses total should be given. In calves affected with simple vitamin E deficiency, treatment with dietary supplementation using tocopherol or substances rich in vitamin E can be used. Calves have been cured using 600-mg of alpha-tocopherol initially; followed by daily doses of 200-mg. Any polyunsaturated fats should be removed from the diet as these may be causing the vitamin E deficiency.