**Ruminal tympany**

**Ruminal tympany**, also known as **bloat**, is a disease of [ruminant](https://en.wikipedia.org/wiki/Ruminant) animals, characterized by an excessive volume of gas in the [rumen](https://en.wikipedia.org/wiki/Rumen). Ruminal tympany may be primary, known as **frothy bloat**, or secondary, known as **free-gas bloat**.[[1]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Constable_2016-1)

In the rumen, food eaten by the ruminant is [fermented](https://en.wikipedia.org/wiki/Fermentation) by microbes. This fermentation process continually produces gas, the majority of which is expelled from the rumen by eructation ([burping](https://en.wikipedia.org/wiki/Burp)).[[2]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Reese_2013-2) Ruminal tympany occurs when this gas becomes trapped in the rumen.

In frothy bloat (primary ruminal tympany), the gas produced by fermentation is trapped within the fermenting material in the rumen, causing a build up of foam which cannot be released by burping.[[3]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Boden_2001-3) In cattle, the disease may be triggered after an animal eats a large amount of easily fermenting plants, such as [legumes](https://en.wikipedia.org/wiki/Legumes), [alfalfa](https://en.wikipedia.org/wiki/Alfalfa), [red clover](https://en.wikipedia.org/wiki/Red_clover), or [white clover](https://en.wikipedia.org/wiki/White_clover).[[1]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Constable_2016-1) Some legumes, such as [sainfoin](https://en.wikipedia.org/wiki/Sainfoin%22%20%5Co%20%22Sainfoin), [birdsfoot trefoil](https://en.wikipedia.org/wiki/Birdsfoot_trefoil%22%20%5Co%20%22Birdsfoot%20trefoil) and [cicer milkvetch](https://en.wikipedia.org/wiki/Cicer_milkvetch%22%20%5Co%20%22Cicer%20milkvetch) are not associated with causing bloat in cattle.[[4]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Majak_1995-4) In [feedlot](https://en.wikipedia.org/wiki/Feedlot) cattle, a diet containing a high proportion of cereal grain can lead to primary ruminal tympany.[[5]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Cheng_1998-5) The main signs of bloat in cattle are distension of the left side of the abdomen, [dyspnea](https://en.wikipedia.org/wiki/Dyspnea%22%20%5Co%20%22Dyspnea) (difficulty breathing) and severe distress. If gas continues to accumulate, the right side of the abdomen may also become distended, with death occurring in cattle within 3–4 hours after symptoms begin.[[1]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Constable_2016-1)

In free-gas bloat (secondary ruminal tympany), gas builds up in the rumen and cannot escape, due to blockage of the [esophagus](https://en.wikipedia.org/wiki/Esophagus).[[1]](https://en.wikipedia.org/wiki/Ruminal_tympany#cite_note-Constable_2016-1)

Treatment

1. Removal of gases through [trocar](https://en.wikipedia.org/wiki/Trocar%22%20%5Co%20%22Trocar) or [cannula](https://en.wikipedia.org/wiki/Cannula%22%20%5Co%20%22Cannula)
2. Use stomach tube and remove the ruminal digesta
3. Medi oral (antifoaming agent) 10ml+250ml warm water and drench to the animal. If antifoaming agent not available, vegetable oil can be used, 400–500ml per large animal
4. [Sodium bicarbonate](https://en.wikipedia.org/wiki/Sodium_bicarbonate)
5. [*Nux vomica*](https://en.wikipedia.org/wiki/Nux_vomica)
6. [Antihistamine](https://en.wikipedia.org/wiki/Antihistamine) is used to avoid lameness. One particular sign in [acidosis](https://en.wikipedia.org/wiki/Acidosis) is lameness. Because [lactic acid](https://en.wikipedia.org/wiki/Lactic_acid) accumulates in the coronary band, it causes irritation; [histamine](https://en.wikipedia.org/wiki/Histamine) is released which causes lameness, so antihistamine is used to avoid it.

**Hydrocyanic acid Poisoning**

 **Introduction**

Prussic acid poisoning (also known as cyanide poisoning) is a metabolic condition in livestock that producers may not see very often, but entire herds can be affected by it, and the resulting death rate can be economically devastating.

To protect livestock from prussic acid poisoning, livestock producers need to understand what causes this toxin, what its sources are, and how to recognize the symptoms of this type of poisoning. Producers should also know how to obtain an accurate diagnosis and apply effective treatment. Finally, producers should understand the risk factors and employ effective methods to prevent prussic acid poison­ing.

**Causes and Sources**

Prussic acid poisoning, sometimes referred to as hydrocy­anic acid poisoning or cyanide poisoning, occurs when livestock consume plant parts from specific forage plants, trees, and weed species that contain cyanogenic glycosides (Table 1). Cyanogenic glycosides are plant-specific and at least 55 cyanogenic glycosides have been identified so far (Knight and Walter 2001).

***Table 1. Examples of forages and plants that can accumulate prussic acid.***

|  |  |
| --- | --- |
| **Plant type** | **Examples** |
| Grains, forage crops, and grasses | Corn, flax, sorghum, sudangrass, arrow grass, velvet grass, white clover, Indian grass, birdsfoot trefoil, Johnson grass |
| Trees | Apricot, peach, cherry, chokecherry, elderberry, apple, wild black cherry |
| Landscape and house plants | Eucalyptus, hydrangea |

nitrate poisoning. There are some distinguishing character­istics to help differentiate between prussic acid and nitrate poisoning. Nitrate poisoning causes the animal’s tongue and eyes to turn blue and its blood to turn dark chocolate brown. In contrast, prussic acid causes the animal’s blood to turn a bright cherry red (Krantz 2011). Providing sodium nitrite to animals suffering from nitrate poisoning rather than prussic acid poisoning could make these animals worse.

To perform an accurate chemical analysis that leads to a correct diagnosis, quality test samples are important. Prus­sic acid levels increase in plants during the morning hours and are highest in leaves (Whittier 2011). Consequently, to obtain a good sample, it is best to sample leaves and regrowth at midday. Collect one or two pounds of forage that are representative of the plant material consumed. The sample must remain fresh. If it is allowed to dry, loss of prussic acid will result. Use a container that can be tightly sealed, such as a plastic bag, freeze the sample, and ship it to the testing lab in a cooler with an ice pack via overnight express (Sulc 2012). Contacting the testing lab to obtain complete shipping instructions will ensure a quality sample reaches the lab in a timely manner. To have feed tested for prussic acid content, contact the Washington Animal Disease Diagnostic Laboratory (WADDL) located in Bus­tad Hall, Room 155N, Pullman, WA 99164-7034 or email waddl@vetmed.wsu.edu.

**Treatment**

Treatment can be effective if initiated at the onset of symp­toms. However, severely affected animals usually die within 30 to 45 minutes following symptom onset. In cases where poisoning is less severe, your veterinarian may choose to employ intravenous therapy that includes sodium nitrite and sodium thiosulfate. Many times treatment comes too late and the animal cannot be saved. When this is the case, the focus should be on protecting the rest of the herd by keeping them away from affected feed and carefully moni­toring them for any symptoms of poisoning.

**Risk Factors**

Ruminants are at the greatest risk of poisoning because they have the ability to consume large quantities of for­age and other fibrous materials. However, other species, such as pigs, horses, and house pets, can be at risk as well. It is important to note that the following conditions can increase the risk of prussic acid poisoning.

• The potential for prussic acid poisoning is greatly increased after a frost. Thus, it is critical to delay grazing until reductions in prussic acid are con­firmed through forage testing (after regrowth has been frozen and wilted).

• A variety of grasses and trees (Table 1) common to the Pacific Northwest can place animals at risk.

• Excessive nitrogen fertilization can increase the hazard. Consider split applications of nitrogen fer­tilizer, and apply no more than 60–80 lb of N per acre at one time.

• Using herbicides to control weeds in the pasture can increase the risk of prussic acid accumulation after application.

• Plants under stress from drought or other condi­tions that inhibit regrowth can concentrate prussic acid in leaves that have been unable to mature.

• Grazing the regrowth of plant species susceptible to accumulation of prussic acid after the end of a drought can be hazardous. The new growth usually contains more prussic acid than old growth.

**Prevention**

Due to the severity of prussic acid poisoning and the potential for economic loss, prevention is certainly the best approach (Knight and Walter 2001; Collins and Hannaway 2003; *The Merck Veterinary Manual*). The following list pro­vides some preventive measures.

• Sorghum and sudangrasses should not be grazed when they are in an immature state. Allow these forages to attain a height of 15 to 18 inches before grazing.

• New varieties of sudangrass and sorghum x sudan­grass with lower prussic acid content should be considered when selecting seed.

• Make sure that animals have been provided suf­ficient feed, such as hay, so they are not hungry when they enter fresh pastures. This will reduce the amount of prussic acid consumed and allow more time for the animal to detoxify low levels of HCN.

• Do not provide animals with yard waste that may include plant material containing prussic acid. In addition, take care to ensure that there is no access to affected fruit and shade tree leaves when animals are allowed to inhabit areas adjacent to orchards and landscape plants. The risk increases when only limited feed is available, resulting in animals being more attracted to nearby leaves.

• Depending on the initial level of prussic acid, processing, such as chopping, haying, or ensiling, allows the prussic acid to volatilize, thus reducing it to acceptable levels in the feed. However, only laboratory testing can confirm these levels.

Diarrhoea and Indigestion:

Diarrhoea is a multifactorial disease entity that can have serious financial and animal welfare implications in dairy herds. It has been estimated that 75% of early calf mortality in dairy herds is caused by acute diarrhoea in the pre-weaning period

Diarrhoea is a common complaint in cattle and young ruminants (particularly in the first few months of life). Many of the pathogens and management practices that cause diarrhoea in calves also affect lambs and goats. Most herds are exposed to diarrhoea causing pathogens, and management practices will largely determine the health impact that those pathogens will have on the youngstock. In "real life", most young ruminant diarrhoea is caused by more than one factor or pathologic agent. It is important to be able to correctly diagnose and appropriately treat diarrhea in livestock, and to be able to suggest management strategies that will prevent further outbreaks of disease. Several pathogens are zoonotic agents (Salmonella spp., Cryptosporidium spp., Giardia spp., and certain types of enteropathogenic E. coli) so great care must be taken when handling diarrheic animals, contaminated bedding, and fecal samples to avoid contaminating yourself and others.

*When an animal passes watery droppings many times a day it has diarrhoea.* *Animals with diarrhoea have certain symptoms like*

Lose water and salt from their bodies.

Animals become weak and thin

Loss of appetite

Lose watery feaces contains mucous and sometimes blood comes along with feaces

Loss of milk production and animals can die if treatment gets delayed

**Common causes of diarrhoea**

Diarrhoea may be occurred due to Physiological and Pathological causes:

**Physiological causes of diarrhoea are mainly**

* Increase in intestinal peristalsis
* Hyper secretion of intestinal fluid
* Damage to intestinal mucosa
* Malabsorption
* In diarrhoea, the intestine fails to adequately absorb fluids, and/or secretion into the intestine is increased. Loss of fluids through diarrhoea produces dehydration and the loss of certain body salts. Diarrhoea causes a change in body tissue composition and severe depression in the animal.

**Etiology of diarrhoea-** On the basis of etiology diarrhoea can be classified as infectious and non-infectious diarrhoea

Causes of diarrhoea can be broadly divided into two categories

1. Infectious diarrhoea- Infectious diarrhoea can be caused by agents like virus, bacteria, parasites, mycotoxins etc.
2. Non infectious diarrhoea- Non infectious diarrhoea can be caused due to poor hygiene, stress, overfeeding, indigestion, faulty diet, intestinal injury and inflammation and malabsorption.
* Possible causes include bacterial and viral infections, certain chemicals, intestinal parasites, poor diet, overfeeding on milk or lush grass, poisonous, plants and other toxins, food allergies and even stress.
* In diarrhoea, the intestine fails to adequately absorb fluids, and/or secretion into the intestine is increased. Loss of fluids through diarrhoea produces dehydration and the loss of certain body salts.
* It causes a change in body tissue composition and severe depression in the animal.
* Death from scours is usually the result of dehydration and loss of body salts rather than invasion of an infectious agent.

The correct determination of the cause of diarrhoea is important in order to take effective preventive measures.

**Infectious diarrhoea and its management in calf**

About 80% deaths due to diarrhoea occur in the first 6 months of calf life. The main cause of death in acute diarrhoea is dehydration which results from loss of fluid and electrolytes in diarrhoea stools. Diarrhoea is an important cause of malnutrition. This is because calf with diarrhoea eat less and their ability to absorb nutrients is reduced; moreover their nutrient requirements are increased as a result of infection.

**Viral diarrhoea**

**Rotavirus diarrhoea**

1. Within 24 hours of birth, a germ called rotavirus causes this type of diarrhoea.
2. Infected calves are severely depressed. There may be drooling of saliva and watery diarrhoea.
3. The faeces will vary in colour from yellow to green.
4. Calves lose appetite and the death rate may be as high as 50 %.
5. There are no signs on dead animals; however, there is an increased volume of fluid in both the small and large intestine.

 **Coronavirus diarrhoea**

1. This occurs in calves that are over 5 days of age; the germ is called coronavirus.
2. The animal is not as depressed as in rotavirus diarrhoea.
3. The initial signs may be the same as in rotavirus, but later on the faeces may contain clear mucus that resembles the white of an egg.
4. Mortality is low (1-25 %).

 **Bovine virus diarrhoea (BVD)**

1. Diarrhoea begins 2 to 3 days after exposure to the germ and may persist for a long time.
2. Ulcers on the tongue, lips and in the mouth are the usual lesions found in the live calf.
3. Bovine virus diarrhoea is controlled by vaccinating all replacement heifers 1 to 2 months before breeding.
4. Pregnant heifers should not be vaccinated. Consult your state veterinarian before starting a BVD vaccination programme.

**Bacterial diarrhoea**

Colibacillosis (Eschericia coli)

Eschericia coli is a major cause of diarrhoea in young calves. E. coli germs attack the intestinal mucous membrane and other mucous membranes and produce toxins (poisons).

The toxins cause severe inflammation of the intestinal lining (enteritis) and can lead to death within hours. A less severe form of the disease is usually characterised by diarrhoea accompanied by progressive dehydration. Colibacillosis lasts 2 to 4 days and its severity depends on the age of the calf. E. coli inhabits the intestine and is excreted in the faeces. It can contaminate kraals, stables, floors, paddocks and even water supplies.

Control of E. coli scours can be difficult in a severe herd outbreak. Early detection (as well as isolation of affected animals) and treatment of scours help to prevent new cases. consult your veterinarian or animal health technician for advice on the use of the available remedies, which are usually mixtures of sulphas and antibiotics.

Animals may be vaccinated 6 weeks and 3 weeks before calving. However, the calves must get colostrum in the first few hours of life for the vaccine to be effective.

**Salmonella**

1. Salmonella germs produce a poison called an endotoxin. Calves are usually affected at 6 days of age or older (the same as in coronavirus diarrhoea).
2. Signs of salmonella scours include diarrhoea, presence of blood and fibrin (yellow clots) in the faeces, depression and elevated temperature.
3. Salmonella germs multiply in the intestine and many reach the bloodstream, causing blood infection and sudden death. Finding a membrane-like cover in the intestine of a dead animal suggests salmonellosis.
4. Tick-borne diseases and underfeeding of calves predispose them to salmonella scours. Heavily infected animals may become severely depressed following treatment with antibiotics because treatment causes the salmonella organisms to release toxins.

**Control and treatment of Salmonella diarrhoea**

1. Control of E. coli scours can be difficult in a severe herd outbreak. Early detection (as well as isolation of affected animals) and treatment of scours help to prevent new cases. Speak to your state veterinarian or animal health technician for advice on the use of the available remedies, which are usually mixtures of sulphas and antibiotics.
2. Animals may be vaccinated 6 weeks and 3 weeks before calving. However, the calves must get colostrum in the first few hours of life for the vaccine to be effective.
3. Guidelines for colibacillosis control consist of letting cows calve in an uncontaminated environment, e.g. in the veld; ensuring that calves consume enough colostrum and assisting weak calves; feeding or treating cows with a vitamin A preparation during dry periods (winter); keeping calves in clean pens and paddocks; feeding calves out of clean buckets; and the vaccination of pregnant cows.

**Enterotoxaemia (pulpy kidney)**

1. The disease usually starts quite suddenly. Affected animals become listless, display uneasiness, and strain or kick at their abdomen. Bloody diarrhoea may or may not occur.
2. It is usually associated with change in the weather, a change in the feed of the cows, or management practices that cause the calf to nurse for a longer period of time than usual. The hungry calf may overconsume milk which establishes an environment in the gut that is conducive to the growth and production of toxins by germs.
3. In dead animals the gut may be red in colour or have bloody, purplish areas.

**Other causes of calf diarrhoea**

**Coccidiosis**

Coccidiosis occurs in calves of 3 weeks of age and older, usually following stress, poor sanitation, overcrowding or sudden changes of feed.

A typical sign of coccidiosis in young calves is diarrhoea with faeces smeared over the rump as far around as the tail will reach. The symptoms are diarrhoea, with slimy and bloody faeces, emaciation, weakening and anaemia. The affected calves strain excessively when they defecate.