The Pharma Innovation Journal 2023; 12(4): 976-980

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277-7695
ISSN (P): 2349-8242
NAAS Rating: 5.23
TPI 2023; 12(4): 976-980

www.thepharmajournal.com
Received: 27-02-2023
Accepted: 30-03-2023

Akanksha Gupta
Ph.D. scholar Department of Dairy Cattle Physiology, National Dairy Research Institute Karnal, Haryana, India

Swati Thakur
Ph.D. scholar Department of Veterinary Physiology and Biochemistry, Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar, Haryana, India

Shubham Thakur
Ph.D. Scholar Department of Animal Nutrition, Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar, Haryana, India

Corresponding Author:
Akanksha Gupta
Ph.D. scholar Department of Dairy Cattle Physiology, National Dairy Research Institute Karnal, Haryana, India

Rumen dysfunctions and their management

Akanksha Gupta, Swati Thakur and Shubham Thakur

Abstract
Rumen dysfunctions are one of the major threats to the health and welfare of animals worldwide. The most common ruminal disorders/dysfunctions are usually defined as indigestion. The term indigestion is a general term for a group of diseases that describe mainly the reticuloruminal dysfunction. Ruminal disorders have a high morbidity that can results in great losses both in production and costs of treatment of affected animals. In addition, rumen dysfunctions cause a huge economic loss due to high mortality, feed wastage, delayed marketing, unthriftiness of the recovered animals, incomplete utilization of disease-provoking food, and extra labor costs of preventive and therapeutic measures. Management of ruminal dysfunctions has relied on agents and mixtures that restore the normal ruminoreticular environment, reduce foam stability and promote release of free gas such as ruminal acidifying and alkalinizing agents, antifoaming agents, and motility modifiers.

Keywords: Rumen, dysfunctions, bloat, acidosis, impaction

Introduction
The rumen is one of the most extensively studied gut ecosystems, because of the importance of ruminants for human nutrition and the major role played by rumen microbes in the nutrition and health of the ruminant animal. Indeed, ruminants are in a symbiotic relationship with the microorganisms in the rumen, degrade and use fibrous feed as a source of energy and nutrients. The rumen is a fermentation vat *providing* an anaerobic environment, constant temperature, pH and, good mixing.

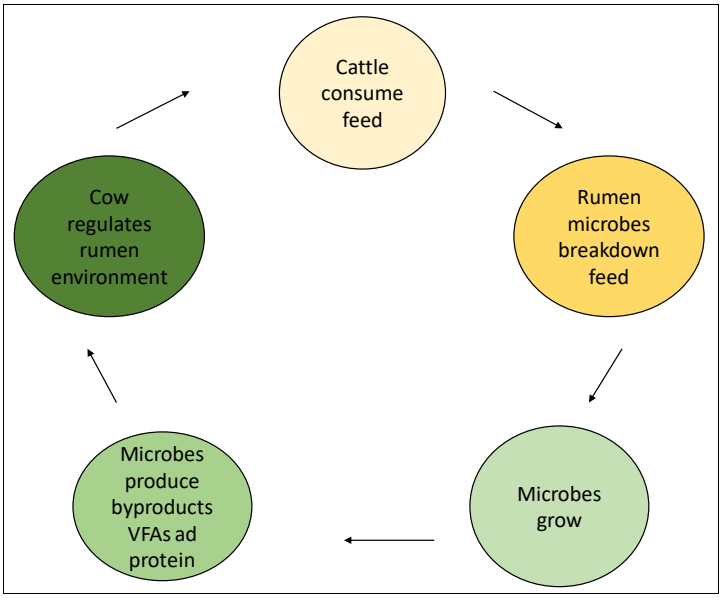

Fig 1: Basic scheme for rumen function

The basic scheme of rumen function (Fig 1) includes that cattle consume feed rumen microbes breakdown that feed, various byproducts like VFAs and proteins are produced and the ruminal environment is regulated, Disturbance in any of these activities results in various dysfunction in the rumen. Following are the common rumen dysfunctions;

1. Ruminal acidosis
2. Ruminal tympany/ bloat

3. Vagus indigestion
4. Ruminal impaction
5. Ruminal drinkers
6. Ruminal parakeratosis

Ruminal acidosis

Ruminal acidosis is frequently defined as a decrease in the ruminal pH. It may be better to define ruminal acidosis as a fermentation disorder in the rumen characterized by a lower than normal ruminal pH, but reflecting an imbalance between microbial production, microbial utilization, and ruminal absorption of volatile fatty acids (VFA) Ingestion of a large amount of fermentable carbohydrate. Cattle that accidentally gain access to large quantity of readily digested carbohydrate particularly grains. Change in microbial population particularly Gram+ bacteria like *Streptococcus bovis.* Lactic acid increases, pH decreases to 5 which destroy protozoa, cellulolytic, and lactate utilizing organisms and impairs rumen motility. Osmotic pressure increases, movement of excessive quantity of fluid in the rumen resulting in dehydration. (Jaramillo *et al.*, 2017) [14].

Some 30% to 50% of the acid in the rumen is neutralized by salivary buffers or bound to ammonia generated from urea entering across the ruminal wall. A smaller quantity passes on into the lower gastrointestinal tract. However, even the most conservative estimates leave a significant proportion of about 30–50% of the acid that is ruminally produced and that has to be absorbed by the ruminal wall, and one of the most important reasons for the appearance of ruminal acidosis would be a decrease in the absorptive capacity of the rumen which is thus unable to maintain a stable pH. Absorption of VFA, by removing unionized acid and by the exchange of ionized VFA for bicarbonate during the absorption process, aids in maintaining pH near neutrality. Consequently, a reduced rate of VFA absorption causes ruminal pH to drop for two reasons: ruminal VFA accumulates and bicarbonate input from the bloodstream is decreased. The severity of acidosis allows us to classify ruminal acidosis considering different factors, among others, like ruminal pH threshold, predominant acid (VFA or lactic), and ruminal population bacteria, in two forms: acute and subacute acidosis. In a brief summary, and starting with clinical signs, in acute forms symptoms will appear in the animal, more or less noticeable, and will be absent in a subacute form. Taking into account ruminal parameters, ruminal pH will be low in acute form, and this fact will imply an important difference in bacterial species, with gram negative bacteria appearing, with lactate consumers bacteria, and high amount of VFA. Meanwhile, in an acute form, we will find gram positive bacteria, with lactate producer bacteria, like the amount of serum lactate and decreasing the presence of lactate in the rumen. In conclusion, we can define acute ruminal acidosis as a metabolic status defined by a decrease in blood pH, parallel to blood bicarbonate decrease, which is caused by a D-lactic ruminal overproduction. *Streptococcus bovis* or even, in ruminal pH below 4.8, *Lactobacillus* spp. In this severe form, with pH next to the isoelectric point of lactic acid (around 3.8), we will find metabolic acidosis, with a decrease in blood pH and blood bicarbonate, increasing.

Subacute ruminal acidosis

Periparturient cows are at risk

a) Concentrate intake increases
b) The time required for rumen microflora to adapt
c) Deficiency of fibre in the diet
d) Error in delivery of ration

Milk Fat suppression

- SARA is considered to reduce milk fat to protein ratio (FPR) below 1.
- In acidic rumen fermentation products such as Trans 10, cis 12 conjugated linoleic acid are produced which are considered to be potent inhibitors of mammary de novo fat synthesis (Bauman *et al.*, 2008) [2].

Manipulation of acid–base balance

A basic understanding of acid–base balance suggests that additional dietary sodium bicarbonate could be beneficial to counteract a high risk of subacute rumen acidosis (SARA). However, as in dietary cation anion balance (DCAB), sodium itself has an alkalinizing effect, as more complete theories of acid–base balance suggest that pH itself is merely an easily measured proxy of a more complex ionic balance. This may explain the reported beneficial effects of positive DCAB diets on the dry matter intake of postparturient cows, and provides an alternative strategy to counteract a high risk of rumen pH depression.

Hepatic oxidation theory (HOT)

In cattle, satiety has been proposed to depend on the balance between rumen stretch and hepatic oxidation, primarily of non-esterified fatty acids (NEFAs) or propionate to ATP. According to this theory, intakes in the first 2 weeks of lactation are suppressed by hepatic oxidation of NEFAs and rumen stretch limits appetite after this initial period and before peak, dry matter intake has been reached. After peak intake is reached or during the dry period, propionate may be absorbed surplus to requirements, increasing hepatic propionate oxidation and stimulating satiety before rumen stretch becomes limiting. Since rumen stretch is an important stimulus for rumination, if this controversial theory is proven true, balancing ruminal and post ruminal starch digestion at different stages of lactation may offer new avenues for the dairy nutritionist to influence the risk of subacute rumen acidosis (SARA), dry matter intakes, milk yield, lactation persistence, and body condition change (Allen *et al.*, 2009) [1].

Clinical findings

- Enlarged rumen, hypermotility
- Abdominal pain
- Hydrorumen; accumulation of organic acids and glucose increasing the osmotic pressure inside the rumen, resulting in water flux from bloodstream across rumen wall.
- Simple indigestion- rapidly fatal acidemia and strong metabolic acidosis.
- Laminitis- altered hemodynamics peripheral and microvasculature

↓

Vasoactive substances (histamine)

↓

Bacteriolysis and tissue degradation

↓

Injured microvasculature

↓

Ischemia of extremities
(Kleen *et al*., 2003)

Clinical findings

- Enlarged rumen, hypermotility
- Abdominal pain
- **Hydrorumen:** Accumulation of organic acids and glucose increasing the osmotic pressure inside the rumen, resulting in water flux from bloodstream across rumen wall.
- **Simple indigestion:** Rapidly fatal acidemia and strong metabolic acidosis.
- **Laminitis:** Altered hemodynamics peripheral and microvasculature.

Management

- Slowly degradable starch sources such as maize pose a lower risk than sugars, wheat or barley.
- More rapidly digestible fibre encourages high dry matter intake and allows higher milk yield to be maintained, lowering the risk of acidosis.
- Sufficient dietary sodium is required to absorb VFAs and the requirement is likely to increase when lactate is pre
- Restricted water intake for 24 hrs (Russell *et al.*, 2017) [12].

Treatment

- 5% Sodium carbonate solution- intravenous.
- Balanced electrolyte solution.
- Antibiotics, Vit B complex.

Ruminal tympany / Bloat

Distension of ruminoreticulum by accumulated gases, because of failure of eructation mechanism.

a) Primary Bloat; Inability to eructate because of froth and foam.
b) Secondary bloat Choke (esophageal obstruction, neoplastic growth, disease affecting vagal nerve functions.

Primary bloat/ Frothy bloat pathophysiology
Intake of legumes alfa alfa (saponins)
↓
Entrapment of gases in a stable foam
↓
Uniting of small gas bubbles
↓
Intra ruminal pressure
↓
Receptors in cardia signal medulla
↓
Esophageal sphincter will not relax
↓
Eructation reflex cant complete
↓
Gases cant escape

Factors affecting Pasture bloat

1. A highly digestible high-protein forage (i.e., alfalfa, clover, wheat) that results in rapid gas production and proliferation of ruminal microbial populations.
2. Presence of fine plant particles that promote the coalescence of gas bubbles in rumen contents that restrict the release of fermentation gases. These fine particles arise from the fragmentation and rupture of chloroplast.
3. Conditions favourable for ruminal bacteria to produce an excessive amount of exopolysaccharides or bacterial slime that contributes to the establishment of a stable foam consisting of fine particles and trapped fermentation gases (Majak *et al.*, 2003) [9].

Factors affecting Feedlot bloat

1. Feedlot bloat occurs most commonly during the finishing period, when cattle are fed diets high in grain and low in roughage. (Herrera *et al.*, 2009) proposed that certain feeding regimens in ruminants can lead to the proliferation of *S. bovis* in the rumen, resulting in the production of high levels of lactate and capsular polysaccharide (slime).
2. Frothy feedlot bloat is frequently, but not exclusively associated with ruminal acidosis. The viscous, frothy nature of rumen contents is almost a continuous condition in feedlot cattle fed high grain diets and although not always acidotic, the ruminal pH is invariably lower than that observed with pasture bloat. Feedlot bloat usually occurs within the first 14 days that an animal has been introduced to a high-grain diet.
3. Finely ground feed particles along with bacterial slime serve as the nuclei for the development of the stable froth associated with feedlot bloat. Additionally, fine grinding increases the feed surface area available for microbial digestion, accelerating both acid and bacterial slime production. Increases in rumen fluid osmolarity can further promote the lyses of bacterial cells and the release of cell contents including endotoxins, factors that can further contribute to the increased viscosity and frothiness of rumen fluid. The lower rumen pH associated with these rapidly fermentable diets may also play a role in the stabilization of the froth (Hall and Majak, 1988) [10].
4. An increase in *S. bovis* has been suggested to play a role in the aetiology of feedlot bloat.

Clinical findings

- Sudden death.
- Left flank so distended that contour of paralumbar fossa protrudes above vertebral column.
- Dyspnea
- Protrusion of tongue
- Extension of head and neck
- Frequent urination
- Tympanic resonance over the dorsal abdomen

Management

- Pasture management; Grasses or bloat resistant legumes such as sainfoin, birdsfoot trefoil.
- Use of grass or silage–legume mixtures.
- Crop maturity; risk is highest at the vegetative and pre-bud stage of growth (Thompson *et al.*, 2000) [13].
- Feed additives; ionophores like monensin and lasalocid, various mineral mixtures, poloxalene prevent bloat (Majak *et al.*, 2015) [11].

Treatment
- Trocarisation releases a large quantity of gas.
- Antibloat agents like bloatosil.
- Oils and non-absorbed surfactants.
- Newer treatment- Dimethicone, Poloxalene.

3 Ruminal impactions
- Accumulation of the indigestible materials (polythene bags, nylon rope) in the rumen interferes with the flow of ingesta.

Clinical signs
1. Absence of rumination.
2. Reduced ruminal motility.
3. Distension of rumen.
4. Passing of scanty or no feces.

Treatment
Exploratory rumenotomy is the only choice for both diagnosis and treatment

4 Vagal indigestion/ Chronic indigestion
The gradual development of abdominal distension as a result of lesions affecting the vagus nerve.
Aetiology;
a) Injury, inflammation, or pressure on the vagus nerve.
b) TRP (vagal nerve damage absent).

Foster *et al*., (2017) [4] suggested types of vagal indigestion.

Type 1: Localized peritonitis, chronic pneumonia, inflammatory lesions in the vicinity of the vagus nerve, and failure of eructation.

Type 2: Failure of omasal transport, conditions preventing ingesta from passing through an omasal canal into the abomasum.

Type 3: TRP, abomasal volvulus, secondary abomasal impaction.

Type 4: Indigestion of late gestation enlarged uterus shifts the abomasum cranially Inhibits normal functioning.

Clinical findings
1. The gradual development of abomasal distension secondary to ruminoreticulum distension.
2. Distension of dorsal and ventral sac L shaped rumen on rectal examination
3. Left dorsal and left and right ventral distension of abdomen Papple shaped abdomen (Pear + Apple) from behind
4. Sticky feces and fecal output
5. Milk production decreases.
6. Strength of ruminal contraction decreases,

Treatment
- Left flank rumenotomy for type 1 and type 2 vagal indigestion
- Right flank rumenotomy for type 3 and type 4 vagal indigestion.
- Transfaunation
- Surfactancts such as poloxalene can be used.

5. Rumen drinkers
- Ruminal drinkers refer to calves that develop chronic indigestion because milk is deposited into the rumen as a result of the failure of the reticular groove reflex during drinking (Herrli-Gygi *et al.*, 2006) [7].
- The esophageal groove allows milk and milk replacers to bypass the rumen, reticulum, and omasum and to flow directly into the abomasum.
- Here are several conditions that can make calves more susceptible to rumen drinking caused by the failure of esophageal groove closure. These factors include;

1. Neonatal diarrhea
2. Irregular feeding times
3. Low quality milk replacer
4. Milk or milk replacer fed at too cold temperature
5. Drinking from an open bucket
6. Tube feeding
7. Stressors such as long-distance transportation.

Calves are indeed creatures of habit, so feeding management and changes in housing and environment are key factors that can affect the closure of the esophageal groove. Care must be taken to avoid changes, provide high quality milk replacer and feed at the proper temperature (approximately body temperature of 101°F or39 °C). Stress such as transport can result in rumen drinking, so it's very important to be aware of the potential for rumen drinkers in arriving calves.
Aetiology and pathogenesis;
- Common in bucket fed calves 2-8wk old.
- Calves that "gulp" milk rather than "sip" milk are at the greatest risk.
- Fermentation of milk retained in the rumen, production of acetic acid, lactate and butyric acid therefore pH decreases Parakeratosis of ruminal mucosa develops.

Clinical signs
- Calves show in appetence, ventral abdomen distension.
- Sticky clay-like feces with a white, putty-like appearance. The color, pH, smell, consistency, and presence of casein coagula are indicators of rumen drinking. Rumen fluid from rumen drinkers will be light or white in color with low pH and sour milk smell. Conversely, normal rumen fluid will be darker in color with pH between 6 and 7. It will smell of fermentation but not of sour milk. Rancid-smelling grayish white fermented material can be obtained by stomach tube from the rumen.
- Ruminal pH less than 6 leads to a systemic acid-base balance disturbance.

Diagnostic feature
Fluid-splashing sounds, audible on auscultation over the left flank while the calf is drinking.

Management
- A rubber nipple floating on the surface of bucket-fed milk may prevent the syndrome.
- Sodium bicarbonate effective alkalinizing agent.

6. Ruminal Parakeratosis
- Characterized by hardening and enlargement of papillae of the rumen.
- Common in animals fed on; high concentrate ratio, heat-

treated alfalfa pellet, calves having prolonged ruminal acidosis

- Papillae are enlarged and hardened.
- Papillae of the anterior ventral sac are commonly affected.
- Affected papillae contain excessive layers of keratinized epithelial cells.
- Interfering with feed absorption, the efficiency of feed utilization.

Prevention: Finishing animals with ration 1 part roughage 3 part concentrate.

References

1. Allen MS, Bradford BJ, Oba M. BOARD-INVITED REVIEW: The hepatic oxidation theory of the control of feed intake and its application to ruminants. Journal of Animal Science. 2009;87(10):3317-3334.
2. Bauman DE, Perfield JW, Harvatine KJ, Baumgard LH. Regulation of Fat Synthesis by Conjugated Linoleic Acid: Lactation and the Ruminant Model. The Journal of Nutrition. 2008;138(2):403-409.
3. Berchtold J. Treatment of Calf Diarrhea: Intravenous Fluid Therapy. Veterinary Clinics of North America: Food Animal Practice. 2009;25(1):73-99
4. Foster D. Disorders of Rumen Distension and Dysmotility. Veterinary Clinics of North America: Food Animal Practice. 2017;33(3):499-512.
5. Hall JW, Majak W, Ryswyk ALV, Kalnin CM, Howarth RE. The relationship of rumen cations and soluble protein with predisposition of cattle to alfalfa bloat. Can J Anim Sci. 1988;68(2):431-437.
6. Herrera P, Kwon YM, Ricke SC. Ecology and pathogenicity of gastrointestinal *Streptococcus bovis*. Anaerobe. 2009;15(1-2):44-54.
7. Herrli-Gygi M, Hammon HM, Zbinden Y, Steiner A, Blum JW. Ruminal Drinkers: Endocrine and Metabolic Status and Effects of Suckling from a Nipple Instead of Drinking from a Bucket. Vet Med Series A. 2006;53(5):215-224.
8. Kleen JL, Cannizzo C. Incidence, prevalence and impact of SARA in dairy herds. Animal Feed Science and Technology. 2012;172(1-2):4-8.
9. Majak W, Garland GJ, Lysyk TJ. The effect of herbage mixtures of alfalfa and orchardgrass on the incidence of bloating cattle. Can. J Anim. Sci. 2003a;83:827-829.
10. Majak W, Garland GJ, Lysyk TJ. The effect of feeding hay before fresh alfalfa on the occurrence of frothy bloat in cattle. Can. J Anim. Sci. 2008b;88:29-31.
11. Majak W, McAllister TA, McCartney D, Stanford K, Cheng KJ. Bloat in cattle. Kaulbars, C. (Eds.) Alberta Agric. Food and Rural Development, Edmonton, AB, Canada, 2003. p. 22–26.
12. Russell S. An update on rumen dysfunction: What are the new thoughts on SARA? Livestock. 2017;22(2):73-79.
13. Thompson DJ, Brooke BM, Garland GJ, Hall JW, Majak W. Effect of stage of growth of alfalfa on the incidence of bloat in cattle. Can J Anim Sci. 2000;80(4):725-727.
14. Jaramillo-López E, Itza-Ortiz MF, Peraza-Mercado G, Carrera-Chávez JM. Ruminal acidosis: strategies for its control. Austral j vet sci. 2017;49(3):139-148.