Acidosis in Feedlot Cattle and Lambs

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Introduction

Ruminants are adapted to digest and metabolise predominantly forage diets. The ruminal microflora of ruminants entering a feedlot consists mainly of cellulolytic gram negative bacteria with protozoa. Fermentation occurs under anaerobic conditions. As a consequence sugars are metabolised predominantly to volatile fatty acids.

The principle volatile fatty acids are acetic, propionic and butyric acids, which collectively provide for the ruminants’ energy needs. The ratio of these VFAs varies with the diet although the majority product is always acetate. On a diet high in the molar ratio of acetic to propionic to butyric acids is roughly 70:20:10. Volatile fatty acids are produced in large amounts in the rumen and continuous removal of these VFAs from the rumen is important not only for distribution, but to prevent excessive and damaging drops in the pH of rumen fluid.

However, growth rates are increased substantially when ruminants consume high grain diets in a feedlot. High concentrate rations favour the production of amylolytic gram positive bacteria such as Streptococcus Bovis and Lactobaccilli. Calves and lambs entering a feedlot are suddenly exposed to diets high in concentrates as opposed to their accustomed high roughage diets and this results in the ruminal pH dropping from 7-7.4 to 5.5 to just above 6. The VFAs in the rumen on a high concentrate ration change to whereby the molar concentration of the acetic to propionic to butyric is 40:40:20. Rumen protozoa disappear through day 9 to 15 with the addition of concentrates to the diet.

The calves and lambs require specifically formulated adaptation rations that are stepped up (roughage percentage is decreased and concentrate percentage is increased) over a period of at least 3 weeks. The adaptation of the ruminal microflora and papillae from a system appropriate for forage (cellulolytic) to a system capable of utilising high energy ration (amylolytic) requires a gradual change over a period of at least three to five weeks.

In general calves and lambs that are in a poorer condition are more prone to acidosis problems and will require a longer adaptation period. Calves and lambs suffering from severe intestinal parasitism and coccidiosis – which was a major problem in lambs arriving at the feedlots following the good summer rains that we experienced in South Africa this year – and protein-energy malnutrition seen in calves and lambs arriving during and after the winter will require a longer adaptation period.
Passive absorption is enhanced by finger-like papillae, which project away from the rumen wall. The rumen is lined with stratified squamous epithelium similar to skin, which is generally not noted for efficient absorption. Nonetheless this squamous epithelium has a structure which functions similarly to the columnar epithelium in the small gut and performs efficient absorption of volatile fatty acids, lactic acid, electrolytes and water. Ruminal papillae increase in length when calves and sheep are fed high grain diets; this presumably increases ruminal surface area and absorptive capacity, which protects the animal from acid accumulation in the rumen. In contrast animals that have been under nutritional deprivation have small, blunted papillae and require time on a high quality diet to allow for development of their papillae and absorptive capacity. Volatile fatty acids are absorbed passively across the rumen wall. If the absorptive capacity of these cells is impaired (chronic rumenitis with fibrosis) it becomes more difficult for an animal to maintain a stable ruminal pH following a meal.

Many calf and lamb feeders attempt to rush the animals onto a finishing diet within two weeks of arrival, often with disastrous effects. The sudden change to a high concentrate diet or a sudden change in quantity of feed consumed leads to acute/sub-acute indigestion- acidosis.

Pathogenesis
Acidosis is categorised as acute or sub-acute, primarily on the basis of the presence or absence of various symptoms. With sub-acute acidosis decreased and/or erratic feed intake is often the sign that is seen and this may be difficult to see in individuals in group-fed cattle. Sub-acute acidosis is the most prevalent form and is definitely more difficult to detect and treat.

Clinical acidosis and the sequelae that follow are discussed below.

When ruminants consume excessive amounts of readily fermentable carbohydrates, ruminal pH drops below 5.5 (the normal physiologic nadir). Any additional intake puts the ruminant at risk of ruminal acidosis. Intake suppression is the animal’s last resort for regulating ruminal pH. Depressed dry matter intake becomes especially evident if ruminal pH falls to about 5.5 and below. Intake depression may be mediated by pH receptors and/or osmolality receptors in the rumen. Inflammation of the ruminal epithelium (rumenitis) causes pain and contributes to feed intake depression during ruminal acidosis.

Unfortunately lactate production at low ruminal pH can offset gains from VFA absorption. As the pH drops, the gram negative bacteria in the rumen begin to die and lyse resulting in the release of massive amounts of endotoxin (Lipo-Polysaccharide). Gram positive bacteria such as *Streptococcus Bovis* and *Lactobacillus* become predominant and lactic acid (both D- and L- isomer) becomes the principal fermentation end-product. *Streptococcus Bovis* begins to ferment glucose to lactate instead of VFA. This is a dangerous situation since lactate has a much lower pH than VFA(3.9 vs 4.8) and lactate is 5.2 times less dissociated than VFA at pH 5.0. As a result lactate stays in the rumen longer and contributes to the downward spiral in ruminal pH.
Additional adaptive responses are invoked if lactate production begins. Lactate-utilising bacteria such as *Megasphaera elsdenii* and *Selenomonas ruminantium* begin to proliferate. These beneficial bacteria convert lactate to VFA which then easily protonate and are absorbed. However, the turnover time of lactate utilisers is slower than that of lactate synthesisers, thus this mechanism may not be invoked quickly enough to fully stabilise ruminal pH. Low ruminal pH during ruminal acidosis also reduces the number of species of bacteria in the rumen and protozoal populations are destroyed as the pH approaches 5.0. When no protozoa and fewer species of bacteria are present the rumen microflora are less stable and less able to maintain normal ruminal pH during periods of sudden dietary change.

Lactic acid production increases osmotic pressure within the rumen so that fluid is drawn into the rumen from the circulatory system as well as other tissues. The rumen pH drops resulting in rumen stasis. The D- and L- lactic acids are converted to sodium lactate. In the rumen they contribute to hyper tonicity. D-lactate, because of its slower rate of metabolism, is absorbed into the circulation and contributes to the depression of blood pH. There is some absorption of lactate from the omasum and abomasums and some as a result of continuing fermentation in the small intestine. An osmotic gradient is established within the intestinal tract, drawing fluid into the rumen, contributing to the profuse diarrhoea.

Ruminal epithelial cells are not protected by mucus, so they are vulnerable to chemical damage by acids. Low ruminal pH leads to rumenitis, erosion and ulceration of the ruminal epithelium. Once the ruminal epithelium is inflamed, bacteria may colonise the papillae and leak into the portal circulation. *Fusobacterium necrophorum*, a gram negative obligate anaerobic bacterium and a component of normal rumen microflora, is the primary etiologic agent. Two biovars have been implicated. Biovar A (*F. necrophorum necrophorum*), the more virulent, is the most predominant biovar in the rumen microflora and is isolated usually in pure culture, from most cases of liver abscessation. Biovar B (*F. necrophorum fundiliforme*) is commonly isolated from micro-abscesses in the rumen wall and is less commonly isolated from liver abscesses, in which it is always found in mixed culture with Biovar A, or other bacterial species. *Arcanobacterium pyogenes*, *Streptococci*, *Staphylococci* and *Bactroides* species are most frequently recovered from mixed cultures.

These bacteria may cause liver abscesses, which may eventually lead to peritonitis around the site of infection. Most liver abscesses are occult lesions that regress to a sterile abscess. Untoward sequelae include peritonitis after abscess rupture into the peritoneal cavity. If the ruminal bacteria go through the liver undetected (or if bacteria from the liver infections are released into the circulation) this may result in the caudal vena cava syndrome. They may colonise the lungs, heart valves, kidneys or joints, resulting in pneumonia, endocarditis, pyelonephritis or arthritis. The infection of the lungs (chronic suppurative pneumonia) as a result of the septic emboli, may lead to the invasion of pulmonary blood vessels, causing them to rupture into the airways. This leads to hemoptysis, epistaxis and peracute deaths due to massive pulmonary haemorrhage.

The other sequelae of acidosis are mycotic rumenitis where fungi such as *Mucor Absidia* and *Rhizopus* invade the rumen wall. Damage to the rumen wall may result in the translocation of rumen LPS into the prehepatic bloodstream, initiating an acute
phase response. The elevated LPS levels result in an increased serum haptoglobin concentration within three days, as well as serum amyloid A within 24 hours. Serum haptoglobin and serum amyloid A are two proteins used as inflammatory markers in ruminants – their concentration is elevated as a result of tissue damage.

With the drop in pH and the proliferation of gram positive anaerobes, two thiaminase 1 producing bacteria namely Bacillus thiaminolyticus and Clostridium sprogenes and a thiaminase 2 bacteria namely Bacillus aneurinolyticus proliferate and result in a vitamin B1 deficiency otherwise referred to as polio encephalomalacia, or cerebral cortical necrosis. Thiamine is necessary for the production of thiamine diphosphate as a coenzyme, which plays a role in the activation of transketolase. Transketolase found in glial cells and erythrocytes is an important enzyme involved in glucose metabolism (eg elevated blood pyruvate concentrations). Since the brain is glucose dependant, glial cell transketolase plays an important role in brain metabolism.

Ruminal acidosis has been associated with laminitis and hoof overgrowth. This is thought to relate to the relative levels of histamine produced in the rumen. The severity of the laminitis depends on the duration and frequency of the metabolic insult. These foot problems generally do not appear until weeks or months after the initiating event.

The frothy bloat associated with acidosis is due to an alteration in the viscosity of the ruminal fluid; the viscosity of the ruminal fluid is markedly increased due to the production of insoluble slime by certain bacterial species that proliferate on high carbohydrate rations. The slime may entrap the normal rumen fermentation gases. The physical form of a grain ration appears to be related to the frothy bloat: fine particular matter can markedly increase foam stability and the feeding of ground grain of fine particular size (geometric mean particle size of 388 micron) was associated with more froth production than the use of coarse particle size (715 micron). The pH of the rumen fluid also plays an important part in the stability of the foam; maximum stability occurs at a pH of about 6.

The time required for the ruminant’s intestinal microbes to be restabilised after a digestive disturbance (once conditions favour such stabilisation) has not been clearly established. A week or more is required for cattle not inoculated with ruminal ingesta to attain the diet intake levels of cattle inoculated with two litres of ruminal ingesta. Results from in vitro studies have shown that 10 to 15 volume turnovers, or about 6 to 10 days, may be required to bring a rumen microbial population receiving glucose in a haemostat to steady state conditions.

Redgut, or clostridium enterotoxaemia, is another sequelae of acidosis. Redgut is not always seen in cattle that have died from acidosis, but animals that are diagnosed at post mortem with clostridium enterotoxaemia invariably have a degree of acidosis. The most frequent basis for diagnosis for enterotoxaemia is reddened or haemorrhagic areas in the small intestine; these frequently may also arise from other phenomena such as post mortem redistribution of blood, salmonellosis or other segmental or diffuse congestion. Clostridium perfringens will invariably be identified because they are part of the normal flora of the bovine intestine. Clostridium Type A is the organism isolated from all cases of suspected enterotoxaemia.
Clinical findings
The rapidity of onset of clinical signs varies and depends on the nature and quantity of feed consumed and the adaptation of the animal to the diet. Unadapted animals may die from quantities of feed that are readily consumed by animals conditioned to the feed. On the other hand even animals on “full feed” will overeat and develop lactic acidosis under certain conditions. Usually clinical signs will become apparent in 12 to 36 hours following engorgement on grain or similar material. Incoordination and ataxia are first noticed, followed by profound weakness and depression. Total anorexia will be apparent, heart rate is elevated (90 to 120 beats per minute) and respiratory rate is increased (50 to 80 breaths per minute); affected animals will appear to be blind. The rumen is splashy with complete stasis, with abdominal pain evidenced by abdominal grunting and grinding of teeth. Loose pasty grey manure is often observed and the manure very often contains gas. Significant dehydration will become apparent within 24 to 36 hours. Affected animals are weak and may become recumbent.

Treatment
Animals with mild cases may recover without treatment and in more severe forms the damage to the animal may be so extensive, that even with intensive therapy, only limited success will be achieved. Emptying of the rumen by oral lavage or rumenotomy is indicated if circumstances permit – this is not practical in a feedlot environment. It is important to firstly correct the acidosis and restore the hydration status of the animal: the oral administration of magnesium oxide, together with 8 to 12 litres of lukewarm water, is essential and at the same time one can administer monensin bullets (2 grams in cattle only) through the stomach tube. The volume of water allows for the distribution of the magnesium oxide and the monensin within the rumen.

One should avoid the use of the carbonates as an antacid as this can aggravate the bloat situation – the carbonates break down into carbon dioxide and a single oxide ion. The parenteral administration of penicillin at a dose of 20ml (stat dose) also aids in destroying the vast number of gram positive lactate producing bacteria in the rumen. Thiamine administration is essential and should be given at least twice and even better three times in the first twenty-four hours. The first dose should be given intravenously if possible (10ml stat dose), followed by 10ml intramuscularly. Administration of antihistamines has been found to be beneficial by certain people.

Prevention and control
Primary control is by controlling ruminal acidosis through the method of feeding, diet composition and diligent feedbunk management, as well as the use of ionophores such as monensin, salinomycin and lasalocid. Only monensin limits feed intake and the use of buffers in the diet such as feedlime. Fewer ruminal lesions develop when the ratio of concentrate to roughage is decreased and when the transition from a roughage-based to a concentrate-based diet is lengthened. Increased roughage in the ration, particularly in the starter ration, gradual step ups in terms of starches and sugars and multiple daily feedings increase the time of mastication and saliva flow. The pH of saliva is between 8.1 and 8.3 – the best bluffer we have and in copious volumes. This increases buffer to the rumen and provides a continuous and uniform
fermentation that reduces intraruminal acidity, which in turn lowers the number of ruminal lesions and indirectly the number of liver abscesses. There are other technologies such as direct fed microbials available that may assist in preventing ruminal acidosis, especially for cattle that have been preconditioned or backgrounded and are accustomed to feed, but of reasons such as transport, revaccination, etc are away from feed for a protracted length of time and may overeat when given feed.

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