Acidosis in Cattle

Rumen acidosis is a metabolic disease of cattle and occurs when the pH of the rumen falls to less than 5.5 (normal is 6.5 to 7). A large amount of highly fermentable feeds, such as grains, consumed in a short amount of time can result in the production of more lactic acid than can be buffered by the rumen. This results in water from the circulatory system being drawn into the rumen (body becomes dehydrated) and pronounced changes in the blood pH.

Acidosis is of two types:
• Subacute Ruminal Acidosis (SARA)
• Acute Ruminal Acidosis

Subacute Ruminal Acidosis (SARA)

SARA is characterized by repeated bouts of low rumen pH that recovers after ranging from few minutes to few hours. The long bouts (>3 to 4 hours) are of concern as they negatively affect fiber digestion (Russel and Wilson, 1966), decrease the absorptive capacity of ruminal epithelium (Harmon et al, 1985) and even damage the rumen epithelium thereby increasing the potential for bacteria, amines and toxins produced by bacteria to enter the portal circulation, causing lower abscesses and inflammation (Gohzo et al, 2005). There is increasing evidence that these toxins are implicated in Laminitis (Mungall et al, 2001).

Acute Acidosis

During acute ruminal acidosis, the pH in the rumen drastically drops (<4.8) and remain low for extended period of time (>2 hours). Acute acidosis is caused by build-up of lactic acid in the rumen, which results from an abrupt increase in the intake of rapidly fermentable carbohydrates. Clinical signs of acute acidosis include anorexia, abdominal pain, rapid beating of heart, abnormally fast breathing, diarrhea, lethargy and eventually death (Krause and Petzel, 2006). Cattle experiencing acute acidosis become “poor doers”, due in part to the damage of GI tract. Survivors of acute acidosis may have chronic problems such as fungal ruminitis, liver abscesses, bloat and laminitis.
Factors contributing to Acidosis:
• Roughage level fed
• Small roughage particle size
• Coarsely chopped forage
• Adaptation to ration (inadequate transition from dry cow to lactation ration)
• Excess grain in early post partum phase
• Rate of starch digestion (grain processing and moisture content)

The risk for acidosis is not the same for all cows. Individual dairy cows exhibit tremendous variation in the degree of acidosis they experience, even when fed and managed similarly (Dohone et al, 2008). A study (Penner et al) has provided evidence that difference in the absorptive capacity of the ruminal epithelia may account for a large proportion of the variation in ruminal pH among animals. Fresh cows are particularly vulnerable to acidosis because of the fairly abrupt change in fermentable carbohydrate intake that occurs after parturition. Grohn and Bruss (1990) observed that the number of cases of ruminal acidosis was greatest during the first months after calving.

The effect of day relative to parturition on mean ruminal acidosis (Penner et al 2007)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Day relative to parturition</th>
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<tbody>
<tr>
<td></td>
<td>-5 to -1</td>
</tr>
<tr>
<td>Minimum pH</td>
<td>5.74</td>
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<tr>
<td>Mean pH</td>
<td>6.32</td>
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<tr>
<td>SARA (pH&lt;5.8), h/d</td>
<td>1.1</td>
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<tr>
<td>Proportion of cow experiencing SARA %</td>
<td>5</td>
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Regulation of Ruminal pH

The digestion of large quantities of highly fermentable feed leads to rapid production of VFAs in the rumen. VFAs are removed from rumen by absorption through rumen epithelium. VFA absorption elevates the pH as a result of clearance of the acid from the rumen during passive diffusion of the protonated VFA and by the neutralization of acid within the rumen when ionized VFA are absorbed in exchange for bicarbonate. The rate of absorption of protonated VFA increases as the pH decreases, but there is likely a threshold at which decreased ruminal pH no longer increases the rate of passive diffusion due to damage to the rumen mucosa (Gabel et al, 1989), leading to further decrease of pH due to accumulation of the VFA, leading to acidosis.
Some of the problems associated with acidosis include (Dr Clell V. Bagley):

- Laminitis
- Poloencephalomalacia
- Ruminitis
- Poor immune function
- Reduced feed intake
- Reduced absorption
- Liver abscesses
- Grain bloat
- Clostridial infections
- Transient diarrhea
- Milk fat depression and poor milk production
- Lameness
- Moderate rumen distention, doughy content and week contractions
- Lung hemorrhages

**Prevention / Correction of Acidosis**

The causes can be grouped into three categories:

- Excessive intake of carbohydrates
- Inadequate buffering
- Inadequate ruminal adaptation

**Prevention**

The key to prevention is reducing the amount of readily fermentable carbohydrate consumed at each meal. This requires proper balancing of fibre and non-fibre carbohydrates. Feeding excessive quantities of concentrate and insufficient forage results in fibre deficient ration, which is likely to cause SARA. Including long fibre particles in the diet reduces the risk of SARA by increasing saliva production during chewing and increasing rumination after feeding. Ruminant diets should also be formulated to provide adequate buffering. A good buffer is that which has a short and long buffering action i.e. it increases the pH in short time and also maintains the pH at optimum level for a long time.

**Treatment**

To the animals showing signs of SARA or acute acidosis, feed a buffer having an appropriate ratio of Sodium Bicarbonate and Magnesium Oxide. Sodium Bicarbonate increases the pH giving immediate relief to the animal and Magnesium Oxide ensures a long time optimal pH.