Rumen Acidosis and Related Complications

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Milk and meat production have become more dependent on the increased use of concentrates in the ration and whether these practices will continue with the known problems that exist and altered rumen fermentation that occurs, is dependent upon the continued advantage of use and solutions to modify the problems of high-concentrate rations.

Rations that consist of primarily roughage provide enough energy to maintain the average annual milk production of approximately 10,000 lbs. per cow in the United States, but most cows have a genetic potential for more milk production and this can be obtained by increased use of grain. Kansas Dairy Herd Improvement records indicate the advantage of concentrate feedings for increased milk production (Table 1).

Table 1
Dairy Herd Improvement Association
Records of Kansas Dairy Herds
for 1976-77 (5)

	Group 1	Group 2	Group 3
Avg. lb. milk/cow	9,029	13,073	17,153
Avg. lb. BF/cow	334	477	636
Lbs. concentrate fed	4,884	5,750	7,061
Lbs. of silage or haylage fed	6,453	9,675	11,482
Lbs. dry roughage fed	4,593	4,173	4,705
Feed cost per cwt. milk	$5.93	$4.70	$4.28

The excessive feeding of grain to obtain maximum milk production may also predispose to disease problems of the dairy cow such as simple indigestion or "off feed," acute rumen acidosis-rumenitis, mycotic rumenitis and abomasitis, laminitis, liver abscesses, and low milk-fat syndrome. Others to be considered but which are not discussed are abomasal displacement and fatty-liver syndrome. Such diseases will be expressed with increased incidence when extreme grain feeding is practiced, especially before or near the time of parturition.

Pathogenesis of Rumen Acidosis

The consequences of feeding high-concentrate diets is a decreased flow of saliva to the rumen and this decreased flow is important in regard to the bicarbonate buffering system of the rumen. Bicarbonate and phosphate are equally important components of saliva and account for most of the buffering in the normal pH range of the rumen (3).

A significant difference in saliva production can be attributed to the physical characteristics of the feed and to the different types of rations. Considerably more saliva is secreted by animals fed hay than those fed concentrate or other types of rations (Table 2).

One of the most important changes that occurs during a transition from a ration high in roughage to a ration high in fermented carbohydrates is the ruminal microbial change. Gradual dietary change is less likely to cause the development of rumen acidosis. Adaptation of ruminal microorganisms to high-concentrate rations involves the selection of organisms which produce volatile fatty acids from starch.

Figure 1 demonstrates a simplified scheme of starch and roughage breakdown in the rumen by microorganisms.

If high-roughage diets are being fed, the pH of the rumen remains relatively high and acetate is the predominant fatty acid. As more concentrates are fed, propionate and butyric increase relative to acetate. If concentrates are increased to the limit, lactic acid and sometimes propionic acid concentrations increase (Figure 2).

Rumen pH is usually within the normal range of 5.8-7.00 and is dependent on the ratio of roughage to concentrate (30). The possibility of disease is relative to the diet and if appreciable amounts of rapidly fermentable sugars or starch in a grain diet are fed, a marked decrease in rumen pH may occur. Values of 5.5 or less in pH are usually associated with lactic acid fermentation occurring in the rumen (13,14,24). Some grass and corn silages may contain in addition up to 8% lactic acid in the dry matter.

An adapted population of ruminal microorganisms competes with or prevents overpopulation by lactic acid-producing bacteria when a high-starch ration is fed, while an unadapted population cannot compete. If an animal is fed a ration of readily-fermented car-

Table 2
Saliva Production in Relationship
to Type and Physical
Form of Ration (2)

Ration	Saliva/10 kg Ingested Food
Hay	50.2 kg
Concentrates	
Meal	13.0 kg
Cubed	10.1 kg
Flaked corn	13.7 kg
Fodder	5.0 kg
Grass	9.3 kg

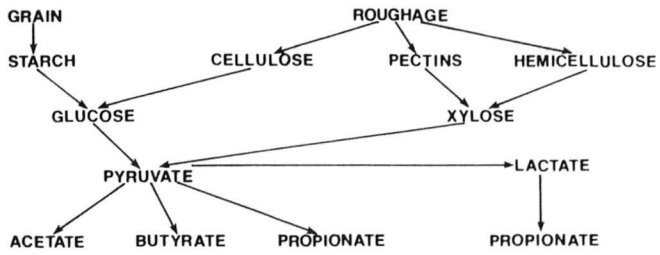

Figure 1. Grain and roughage breakdown in the rumen (21).

bohydrates which is beyond the fermentative capacity of the adapted bacterial population of the rumen, free glucose accumulates. This leads to the rapid growth of *Streptococcus bovis* with production of lactic acid, reduced ruminal pH, and is followed by the growth of lactobacillus organisms and development of excessive rumen lactic acid (19,22). Endotoxins from dying normal rumen bacteria and absorption of lactic acid and other abnormal compounds of the rumen caused local ruminal and systemic effects. Major clinical and pathological changes are found in cardiovascular, digestive, brain and urinary systems (36,37).

Simple Indigestion or "Off Feed"

Not all cases of simple indigestion should be considered as caused by rumen acidity because there are multiple reasons for this syndrome. Dairy cattle may be fed and adapted to continuous levels of high concentrates, but because of daily fluctuation in amount, type, or physical nature of concentrate fed, ruminal pH may temporarily decrease below normal. A mild case of rumen acidosis may exhibit a reduction or loss of appetite for a few days with diminished rumen motility. Feces are sometimes mucoid grey-green in color with a pasty to loose consistency. Milk production will be reduced.

In this regard, Uhart and Carroll (34) changed the ration of 400-lb. calves from primarily alfalfa hay to a ration containing 90% grain with no adaptation period and no limit on consumption. The steers went "off feed" with the period of anorexia varying from 2 to 6 days. Table 3 demonstrates the rumen pH and lactic acid concentration associated with these changes.

Table 3
Rumen pH and Lactic Acid Levels
Associated with Changing From
Alfalfa Hay to a High-Grain Diet (34)

Sampling Time	pH	Lactic Acid m M/L
Initial	6.98	.10
Eating stopped	4.81	99.96
Eating resumed	6.78	.10

Acute Rumen Acidosis-Rumenitis

Severely affected animals that ingest quantities of grain above their adapted capacity, such as occurs in a cow that accidentally gets to the grain bin or group of steers exposed to a self-feeder without adequate adaptation, will exhibit marked intoxication within 12-24 hours after overeating. The signs are complete anorexia, sudden drop in milk production and, sometimes, colic signs. The feces are grey, sometimes yellowish in color, and vary from pasty to watery consistency. Affected animals are reluctant to move, some appear blind and others appear dehydrated as evidenced by sunken eyes and, lastly, fullness of the abdomen. The rumen is static, full and ballottement indicates a watery content. Urination is markedly reduced in quantity.

Others are found in recumbency in semicoma or coma with a "milk fever" attitude and a pool of fluid feces behind them. Differentiation from hypocalcemia, coliform mastitis, toxic metritis, or torsion of a right displaced abomasum would have to be made.

Mycotic Rumenitis and Abomasitis

Fungal agents such as mucor, absidia, and rhizopus have been associated with gastrointestinal disease, especially if the predisposing factors which allow their establishment are present (23). When pH in the rumen decreases to 5.0 or less, many of the normal rumen bacteria and protozoa are killed and this low pH allows fungal agents to have a selective advantage of growth (18). These organisms are also likely opportunists which invade damaged ruminal or abomasal mucosa (32). Extensive use of oral antibiotics is also known to inhibit normal bacterial growth and some antibiotics such as oxytetracycline may actually

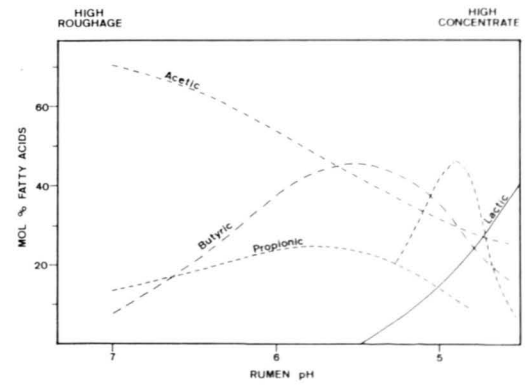

Figure 2. Volatile fatty acids, lactic acid, and pH changes associated with transition from high-roughage to high-concentrate rations (35).

stimulate the growth of fungal agents (28).

The hyphae of fungal agents are invasive and demonstrate a special tendency to infiltrate blood vessel walls of the rumen or abomasum producing thrombosis of vessels or causing disseminating infection of other major organs such as the liver. Infarction due to vascular occlusion is frequently observed (9). Animals severely affected with rumenitis but not responsive to treatment may exhibit continued signs of rumen atony, depression, anorexia, emaciation, and occasionally, black tarry stools. This would be suggestive of mycotic rumenitis. Acute death due to perforating ulcers and diffuse fibrinohemorrhagic peritonitis may be observed.

Decreased Milk Fat

The conditions leading to low concentrations of milk fat were reviewed by Davis and Brown in 1970 (12). The features of low milk fat are a ration high in digestable carbohydrates and low in long fiber roughage. Reduced concentrations of milk fat with excessive grain feeding are more marked in early lactation than compared to late lactation (7). A change from excessive to more restricted grain feeding in early lactation will increase milk fat concentration moderately. In regard to the proportion of rumen volatile fatty acids, a decrease in acetate concentrations and increased propionate and blood glucose are found with low milk fat. Forty percent of the fatty acids of milk fat are synthesized in the mammary gland from acetate, hydroxybutyrate, and triglycerides of the plasma.

Liver Abscesses–Peritonitis

Rumen acidosis and resulting liver abscesses is a common syndrome observed in cattle being fattened for slaughter. The events leading to liver abscesses are a result of rumenitis which allows microorganisms such as *Fusiformis necrophorus* or *Corynebacterium pyogenes* to enter the portal circulation and reach the liver. *Fusiformis necrophorus,* which is present in the rumen of healthy cattle, is known to produce abscesses in the liver and has been produced experimentally (20). It has also been reported that the introduction of a high-grain diet not only requires adaptation of rumen microorganisms, but there is some suggestion of temporary hepatic cell damage and liver dysfunction. It has been shown from biochemical profiles that a metabolic adaptation is needed following the start of grain feeding (4,29).

Clinical signs observed with liver abscesses are usually subclinical, but frank rupture of an abscess and spilling of the contents into the anterior right quadrant of the abdomen can result in a variety of clinical signs, many of which are suggestive of traumatic reticulitis (hardware). Abdominal pain and rumen stasis are often exhibited; decreased milk production and elevation of white blood cell counts are features. Abomasal atony and impaction, and omasal impaction sometimes are found.

Upon exploratory laparotomy and rumenotomy no evidence can be found for metal nor is there close proximity of peritonitis near the reticulum or rumen. Abscesses may be felt on the liver surface, or scars of abscesses and relocalization of abscesses ventral to the medial aspect of the liver or just regional peritonitis ventral to the liver in the region of the omasum or abomasum have been findings.

Laminitis

Bovine laminitis has generally been associated with high-grain diets and is believed to be related to rumen acidosis and lactic acid build-up in the rumen (13). MaClean (26) also related laminitis to other conditions such as mastitis, retained fetal membranes, metritis, abscesses, peritonitis, udder edema, and acetonemia. Histamine is often found in increased levels in the rumen of animals with rumen acidosis, and because of the histopathological findings of increased capillary permeability and arteriolar dilation which are similar to the circulatory effects of histamine, it has been related as the cause of laminitis (10). Serum histamine was found to be slightly elevated with acute laminitis of cattle on high-concentrate rations, but was found to be elevated even more if laminitis progressed to the chronic stage (26).

While rumen acidosis and laminitis appear to be related, the biochemical relationship remains obscure. Laminitis may not be caused by histamine (6). Merritt and Riser (27) described laminitis in Jersey cattle of possible hereditary origin. It has been suggested also that subacute to chronic laminitis and sole ulceration occurs from an allergic reaction, especially if the grain ration is composed of nearly 50% high-protein feed (8).

Both acute and exacerbated chronic laminitis is more common at six years of age or later when the dairy cow reaches her highest lactation and is more prone than in its earlier ages to metabolic and parturient diseases such as metritis and mastitis (26). Mild laminitis is often unnoticed or unremarkable by the owner. More acute laminitis cases will be observed shivering, having muscular tremors, with feet located more under the body or commonly, with treading of the hind feet. More time is spent lying down and severely affected cows may even lie flat on their sides. Some may refuse to rise or do so reluctantly, often resting on their knees before completing the movement. They often move clumsily or are stubborn and refuse to move. While walking, their steps are short and heavy and preference is given to walking on soft ground rather than concrete. When the front feet are more severely affected, they are sometimes crossed in an attempt to take more weight off the medial claws which are often more severely affected than the lateral claws.

Heat may be felt in the feet and pulsation of superficial arteries of the lower foot and a greater degree of distension of the veins of the lower leg are sometimes observed.

Visible changes in form of the hooves are observed in chronic laminitis with overgrowing being a prominent sign. Isolated patches of reddish-brown hemorrhages at the wall-sole junction and heel-sole junction are often seen when pared down. The lateral walls are usually convex and curl over the sole ventrally. The anterior part of the claw is concave and longer than normal. Transverse growth lines are well marked.

The third phalanx can be severely deviated and atrophied when examined by sagittal sectioning of the foot (25). In addition, sole ulceration of the hind feet may be found at the bulb of the heel. Such ulcers are immediately surrounded by a region of soft, yellowish, necrotic-appearing horn. If the horn has grown excessively, fistulous tracts extending from the sole surface to the ulcer can be found.

Prevention With Rumen Buffers

When dairy cows are fed high levels of concentrates, major changes in the rumen environment occur. Some of these changes are detrimental as described in the previous disease conditions. Numerous attempts have been made to use rumen buffers to counteract some of these negative effects (15,16,17,31).

The effects of these buffers on volatile fatty acids, lactate production, and metabolism have been variable. If appropriate levels of supplementation can be determined, they may be useful in combating the negative effects of high-concentrate feeding.

Supplementing rations of dairy cattle with buffers has generally reduced feed intake but in a few trials feed intake has been increased. Reduced concentrations of milk fat with excessive grain feeding leads to the use of sodium bicarbonate for prevention, and the success with its use has lead to investigation of other alkaline materials as feed additives. Some of the buffers that have been supplemented into diets have been sodium bicarbonate, potassium bicarbonate, calcium hydroxide, magnesium oxide, and sodium bentonite. Buffers have no nutritional value and thus there has been some hesitation to recommend widespread use of buffers in dairy rations. If the primary ration consists of heavy grain feeding and finely-chopped forages and the owner doesn't have adequate hay or is not set up to feed it conveniently, buffers could be added to good advantage. The addition of 0.1 to 0.3 pounds of buffer daily to the ration of each cow (either sodium bicarbonate, potassium bicarbonate, or magnesium oxide or a combination of these) might be helpful (11). The cost at this recommended level would be between 1 and 3 cents per cow per day.

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