Rumen Malfunctions – Acidosis Problems with High Grain Rations

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Introduction

Ruminants differ from other species in that their food is exposed to microbial degradation in the forestomachs prior to biochemical digestion by the secretions of the host. The various microbial species in the rumen fluctuate in numbers with changing dietary conditions. Such changes can become unfavourable to the host when excesses or deficiencies of certain nutrients occur in the diet. An important example of an undesirable excess of a dietary constituent is overfeeding of cereal grains which are rich in starch. By promoting the growth and multiplication of some organisms, an unbalanced population results with accumulation of their metabolic end-products which may be different from those produced by the normal population and may have a detrimental effect upon the host.

The modern feedlot operator is confronted with the dilemma of feeding high-concentrate rations to achieve maximal return which is accompanied by the risk of triggering off an unfavourable fermentation in the rumen that can lead to financial losses from death and the unthriftiness which may affect survivors. Episodes of acid indigestion may occur whenever ruminants ingest an excessive dose of grain or other feed rich in starch or sugar. The problem is frequently associated with faulty management: animals may gain access to the feed supply as a result of

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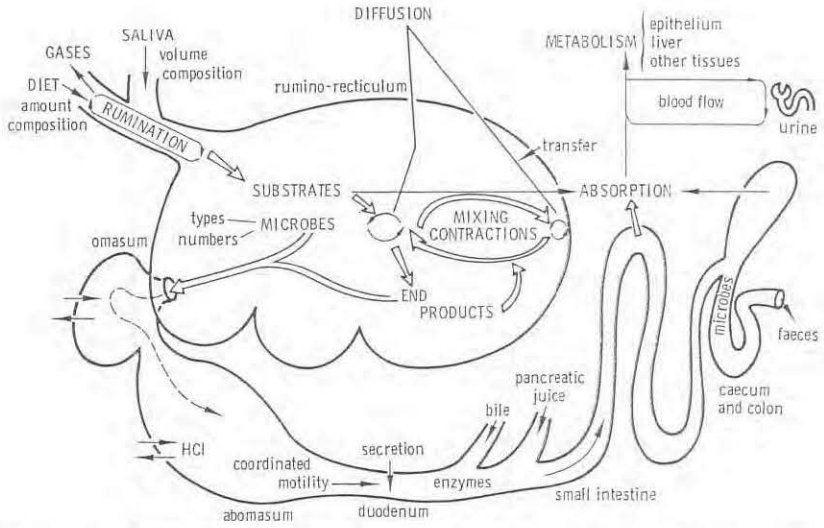

Figure 1. Diagram to illustrate the functions of the gastro-intestinal tract of the healthy ruminant.

defects in fencing, storage containers, pen, stanchions etc.; alternatively, animals may be too rapidly brought on to high levels of concentrate feed or they may be rendered susceptible to excessive intake by missing a feed or two, by letting self-feeders become empty for a period then refilled, by unfavorable climatic conditions, by sudden changes in feed composition, or by having unevenly-sized animals in a lot with a resulting hierarchy that affects feeding behavior.

The general functions of the ruminant digestive tract are illustrated diagrammatically in Figure 1 to provide some orientation for subsequent discussions.

Toxic and lethal doses of feeds

The entry of an excessive amount of sugars (e. g. glucose, sucrose, lactose, maltose) or starch into the rumen of cattle or sheep can lead to a major change in the rumen fermentation regardless of the source of such carbohydrates. Any of the common cereal grains, ripe or green ear corn, various root crops such as sugar beet and fodder beet, and various fruits such as apples, grapes and pears, as well as a number of processed foods such as energy-rich concentrate feedstuffs, flour and bakery by-products, molasses, and even certain dairy by-products such as whey have been incriminated. There are differences between feeds that appear to be attributable to several factors, including the proportions of the various carbohydrates that are present, the fineness of divi-

sion and the presence and proportions of other ingredients or dietary components including water.

It is not clear what the range of doses of the various carbohydrates would be that can trigger the abnormal fermentation. The practical value of knowing the lowest dose that might have this effect is obvious but as yet it has not been studied adequately. It is not even clear whether dose should be referred to body weight or to metabolic size or to some other estimator. In a brief report of incompletely described experiments, Australian workers stated that the dose of crushed wheat that would consistently produce the disease in well-nourished Merino sheep under laboratory conditions was 75-80 g/Kg body weight (49). However voluntary consumption of this amount was not achieved and the dose was added via a rumen fistula. Sheep in poor condition, on the other hand, succumbed after a dose of 50 g/Kg. The only experiment in which voluntary ingestion of a toxic dose occurred was when sheep were gradually brought on to a wheat diet, reaching 80% wheat by the 12th day. The animals were then starved for 24 hours, following which wheat was presented *ad libitum*. Nine out of 25 head died within the following two days. It is of interest to note that, in this case, the animals were group fed and the psychological stimulus of competition between hungry animals that had been starved for a day may have been a key factor in the success of the experiment. Unfortunately, it was not known how much the individual animals consumed, hence no comparisons could be made between those that died and those that survived. The weights of the sheep and the composition of the grain were not given in the report. However the mean wheat consumption was only 36 g/Kg. The experiment was of considerable practical importance, however, because it stressed the significance of controllable factors such as a period of starvation and competition between animals. It also raised questions about the significance of previous exposure to the feed and the possibility of an adaptation process within the rumen. One conclusion is that, although the dose of grain required to produce the engorgement syndrome appears to be very high, under appropriate conditions a proportion of the animals in a group will be sufficiently hungry or greedy to consume the dangerous amount. If animals of mixed sizes are present, e.g. in a feedlot, the larger ones may dominate the feed trough and be the ones affected. Alternatively, when a farmer tries to avoid trouble by feeding hay before filling the feed bunks with grain or concentrate, the larger animals may fill up on the hay and leave the smaller ones to over-indulge on the concentrate. The availability of roughage seems to be a significant factor, if only by reducing the total amount of the concentrated feed ingested. In areas where corn is readily grown, chopped corn ensilage appears to be a valuable diluent of this type.

Recent studies at the Canada Department of Agriculture Research Station at Melfort, Saskatchewan (7) have indicated that chopped hay and cereal straw can be used for a similar purpose of controlling the rumen fermentation in self-fed steers. All the roughage was ground through a one inch screen. The diets were adjusted from 90% roughage plus 10% dry rolled wheat at the start by either 10% or 20% increments in the proportion of wheat every 8 to 10 days until the 70% wheat level was attained. After a longer period at 70%, adjustment was made to 75% and, one week later, to 80% for half the steers (days 71-77 and 77-119 of the trial respectively) while the other half were switched directly to the 80% ration at a later date (for days 107-119 of the trial). In general weight gains and feed conversions were favourable during the trial with the exception of the last two weeks when an unexplained decline in performance was noted in some steers. It was noteworthy that the group that was switched by 20% increments in the proportion of wheat developed digestive disturbances when switched from 50% to 70% wheat. Two steers required treatment and the remainder of the group developed extremely watery diarrhea followed by recovery. These steers had been fed the 70% wheat ration by day 28 of the trial. Since the starting weights averaged 700 lb and daily gains varied from 3.4 lb (days 11-71) to 5.5 lb (days 0-10), the steers would have weighed an average of about 815 lb at the time of the digestive crisis. The average daily feed intake for the critical period was not stated but, for the overall trial, the average was 26.8 lb/day. This would be equivalent to 18.8 lb wheat on the 70% diet or an average intake of only 23 g/Kg. Unless there was a large increase in consumption during this period, this calculation indicates that the hazardous dose of grain in these steers was appreciably lower than that reported for sheep in Australia. Also it indicates that, while there was definite evidence for some type of adaptation in the rumen to high intakes of grain, it would be difficult to predict the safe and hazardous rates of changes of the diet. In this trial it appeared that a sudden change from about 16.4 g/Kg to 23 g/Kg was a critical step in leading to an imbalance in the rumen microbial populations and an abnormal fermentation.

Hironaka (26) reported the results of some interesting experiments on the use of starter rations to bring beef cattle in feed lots on to high intakes of finishing diets rapidly. The principle used was to formulate a starter ration having a digestible energy content at a tolerably low level and feed it for two days then mix it with increasing proportions of finishing ration. Although no definite cases of engorgement or founder occurred, it was observed that a sharp decline in feed intake occurred during the first week if the DE concentration of the feed exceeded 2800 kcal/kg of feed when fed *ad libitum*. It was concluded that the rate of

increase of DE concentration of the ration was a critical factor in getting animals on to full feed safely. Animals were taking in an average of 24 g/kg of finishing ration by nine days on feed without controlling feed intake when the proportions of starter to finishing ration were changed by 25% increments at 2 day intervals from 100% starter to 100% finishing ration, in which the major ingredient was barley. The starter rations contained alfalfa and either beet pulp or brewers grains in addition to oats, barley, molasses, minerals, vitamin A and chlortetracycline.

Tremere *et al* (48) reported the results of a study on adaptation to high concentrate feeding in hay fed dairy heifers. This paper is of particular interest since the major ingredient of the diets used was ground wheat. Dosages of grain were expressed as a function of body size. Daily increments in concentrate intake of 7 g per unit of body size ($W_{kg}^{0.75}$) led to accumulation of lactic acid in the ingesta, a fall in rumen pH and the animal going off feed. Frequency of feeding was an important factor, twice a day providing more protection than once a day.

Microbial changes consequent upon overfeeding on grain

The Australian workers noted a virtually complete replacement of the ruminal and intestinal microflora with lactobacilli after excessive intake of wheat by sheep. They also showed that chlortetracycline orally provided protection from an other wise lethal dose of grain (49). Hungate *et al* (30) showed that the numbers of cellulolytic organisms declined while gram-positive species proliferated and protozoa disappeared. They also identified *Streptococcus bovis* and a species of *Lactobacillus* as components of the flora of engorged sheep and suggested that they were responsible for the development and persistence of increased acidity of the rumen ingesta. These results were confirmed and amplified by Krogh (34, 35, 36) who characterized several species of lactobacilli and the responses to a variety of carbohydrate substrates. He gradually increased the daily dose of the substance until an excessively acid fermentation was triggered. The range of dose required varied from 200-600 grams for sucrose and from 600-1200 grams for lactose, which was given as a partial suspension because of its low solubility. He made quantitative measurements of the microbial concentrations of several genera with respect to time. The results indicated that the sequence of events was as follows: a) proliferation followed by decline of *S. bovis* b) reduction of cellulolytic species and protozoa c) proliferation of lactobacilli and d) in some cases only, proliferation of yeasts. Unfortunately, the lactate-utilizing bacteria were not studied. Krogh reported the lactobacilli isolated were sensitive to antibiotics but not to sulfathiazole. Bullen and Scarisbrick (11) also noted that sodium penicillin, given within 6 hours

of dosing with an otherwise lethal dose of grain, as one to four doses of 5×10^5 Units each into the rumen greatly reduced the degree of accumulation of acid in the rumen. They also showed that rumen acidosis could be distinguished from *Clostridium welchii* type D enterotoxemia which may occur under similar dietary conditions.

Lactate utilization and removal can be accomplished by a variety of species of rumen organisms. Baldwin concluded from isotope labelling patterns that the production of propionate from lactate in rumen contents taken from the cattle fed on high carbohydrate diets occurs mainly via the acrylate pathway (6). This finding points to a role for *Peptostreptococcus elsdenii* in such utilization since it is the only rumen organism known to metabolize lactate via this pathway. However Hobson *et al* (27) reported that this organism occurs in low numbers in the adult rumen. There may be other species capable of utilizing this pathway and the possibility of a role for the protozoa in lactate utilization has not been clearly demonstrated. Certainly the microbiological aspects of lactate removal merit further study since they could be a key to the process of adaptation to high grain diets or to convalesence after an outbreak has occurred.

The phenomenon of adaptation should be studied from the standpoint of the various possible regulatory factors, such as concentrations of substrates (starch or sugar), hydrogen ion concentrations, ammonia concentration, redox potential, osmolality, inhibitory or promoting substances and lactate isomer concentrations in the ingesta.

Chemical changes in the rumen contents

The consistency of the ingesta changes profoundly after the abnormal fermentation of grain overfeeding develops. The contents became milky and a yellowish-green or even grayish color, gas formation is greatly reduced after several hours and the floating or suspended roughage is greatly reduced.

The most dramatic changes in chemical composition are the large increases in hydrogen ion concentration (100-1000 fold) and lactate concentration (21). The lactic acid is a mixture of the two isomers, D-lactic acid and L-lactic acid. The proportion of the two isomers varies considerably with L-lactic acid tending to be predominant initially, following which D-lactic acid increases to equal or exceed the concentration of L-isomer. The pK_a of lactic acid is about 3.7, hence even if the pH falls to 4.0 the majority of the lactic acid that is formed is buffered by alkaline salts in the rumen. However the small proportion of undissociated lactic acid that persists is of great significance because this acid has a corrosive action on the rumen epithelium. Also at pH 4.0 almost all the volatile

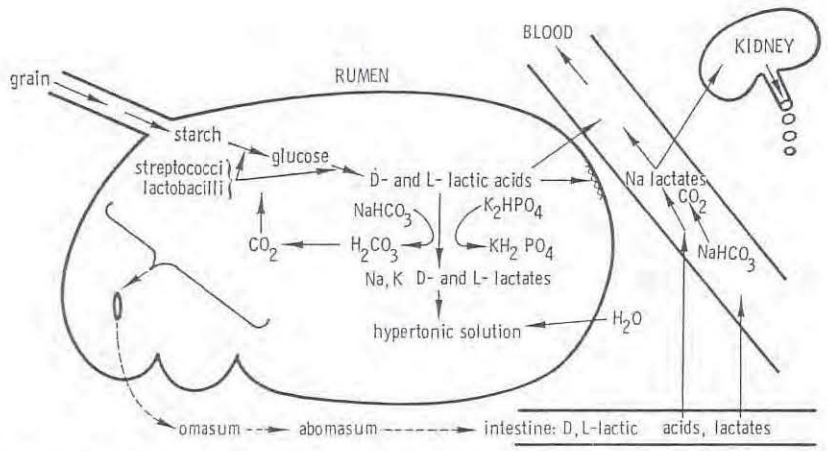

Figure 2. Diagram illustrating some of the significant changes that occur following engorgement on grain if a lactic acid fermentation takes place. The symbol _ _ indicates damage to the ruminal epithelium.

fatty acids present will exist in the undissociated acid form in which they readily penetrate the mucosa of the rumen. A diagram representing these changes is shown in Figure 2.

Many other changes occur in the composition of the ingesta after a ruminant overeats on grain. There is a major rise in osmolality which can be as much as double the normal value. This causes water to move from blood to lumen (10, 15) and thus could be a significant dehydrating factor (internal dehydration since the fluid is trapped in the forestomachs unless diarrhea occurs). A preliminary report suggests that the degree of hypertonicity of rumen contents is correlated with reductions in feed intake and the rate of cellulose digestion (8).

Other electrolyte concentrations change. Potassium and phosphate levels increase while bicarbonate and volatile fatty acid concentrations decline. Sodium may diminish while ammonium ion increases. Free sugar may become detectable and, later in the course, unusual acids, such as succinic and formic, may appear (42, 43). Chloride may increase in the later stages as well, particularly if gastro-intestinal statis persists.

Macromolecular products from microorganisms appear in the contents in increased amounts. This is probably attributable to the increased rate of destruction of protozoa and bacteria under acidic conditions. Some of these components contain endotoxins that can be demonstrated by studying their pyrogenic activity in rabbits after intravenous injection (21). However, the significance of this finding for the well-being of the affected ruminant has not been determined. Endotoxins are less active via the oral route but the presence of very large amounts in the gastro-

intestinal tract might be a contributing factor. The failure of steroids having glucocorticoid activity to have a significant beneficial effect in treatment may indicate that the role of endotoxin is a relatively minor one (37). It is possible that such toxins could account for the non-histamine toxic factor demonstrated to occur in the ingesta of affected animals (16).

Other toxic factors for which a toxic, and possibly lethal, role have been proposed are histamine (12) and alcohol (3, 4). The available data on histamine analysis are very variable and it seems unlikely that histamine consistently plays a major role in the lactic acidosis syndrome (44, 45). Alcohol has not been studied adequately up to the present but the data from Hungary suggest that extremely high blood levels may be attained under some circumstances (31, 46) following ingestion of grain meal (up to 670 mg/100 ml reported) or glucose (up to 1600 mg/100 ml reported blood alcohol concentration). Further studies on the role of ethanol are needed.

Consequences of ruminal changes for the functional status of the animal

The changes in rumen microbes, chemical composition and dynamic processes, such as rates of gas production, can have profound effects on the wellbeing of the animal. The first effect is a local one on the epithelial surface of the rumen and other parts of the gastro-intestinal tract. Lactic acid, hypertonicity and, probably, other factors participate in the damaging effect upon the epithelium which is characterized by microvesicle formation, loss of keratin, vacuolation, invasion by poly-morphonuclear leukocytes, desquamation and death of epithelial cells and small hemorrhages (1, 32, 47). In later stages damage, and even ulceration may occur in the omasum, abomasum and duodenum (18). These effects upon the epithelium probably lead to derangements in the strength and coordination of contractions of these organs (5), partly as a direct action and partly by changes in the function of the receptor component of visceral reflexes. The net effect is rumen stasis which may be accompanied or followed by diarrhea.

While motility is becoming impaired, absorption of lactic acid proceeds and leads to a systemic acidosis of the metabolic type (17, 21, 51). Large increases in lactate concentration of the blood are observed while plasma bicarbonate falls. The effect on these parameters usually reaches a maximum between about 24 and 36 hours after the overfeeding episode and is followed by a return towards normal values, even overshooting to produce a metabolic alkalosis in some cases (18, 20). The blood lactate of normal ruminants is comprised of about 100% of L-isomer but the en-

gorged animals often show D-lactic acidosis with the D-isomer accounting for the majority of the lactate present (21, 22). In animals which deteriorate into a shock-like state L-lactate and pyruvate may rise and this is usually an ominous sign of failure of the circulation to supply adequate amounts of oxygen to the tissues. It is still unresolved whether other toxic factors are absorbed and play a role in producing some of the signs of the disease. It seems unlikely that histamine would be absorbed to a significant extent at the pH of the rumen ingesta of acidotic animals because most of its molecules have two positive charges at pH 4. If histamine is involved it is presumably attributable to absorption from the intestine. The changes in osmotic pressure of the rumen contents lead to dehydration into the rumen from the other body fluids. This causes the hematocrit and the concentration of plasma proteins to rise. The declining blood volume and acidosis lead to circulatory difficulties and inadequacies of regional perfusion with blood. It is thought that these circulatory disturbances may account for some of the other signs of deranged function that are observed. These include reductions in the rates of salivary secretion and urine formation as well as a number of manifestations of disturbed performance of the central nervous system. Signs that may have a partly neurological basis are anorexia, ataxia, reduced muscular tone and strength, lowered rate of respiration, abnormal body temperature (may be high or low, depending upon environmental factors and stage of the disease) increased pulse rate, depressed gastric motility, and reduced behavioral and reflex responses to environmental stimuli. Similarly, depression of function of many organs can be predicted to occur. The liver may be an important site because of its important role in lactate utilization. It should be noted that ruminants have a limited capacity to utilize D-lactate (9, 21, 25, 28). Consequently, the continuing entry of D-lactic acid by absorption in the face of decreased renal function (29) creates a particularly hazardous situation.

The outcome of the detrimental effects may be a progressive deterioration, followed by coma and death. If the animal survives the acute phase of acidosis, it may recover promptly if normal gastrointestinal function is restored or, more frequently, it may be affected by a chronic phase of convalescence and unthriftiness. The acid conditions in the rumen often persist for several days, following which restoration of function occurs very slowly and death may occur during this period from causes that are as yet poorly characterized. This period of unthriftiness can be a major cause of economic loss in outbreaks affecting substantial numbers of cattle as in feedlots.

An example of some of the biochemical changes that may be observed in a case of lactic acidosis as a function of time are shown in Figure 3.

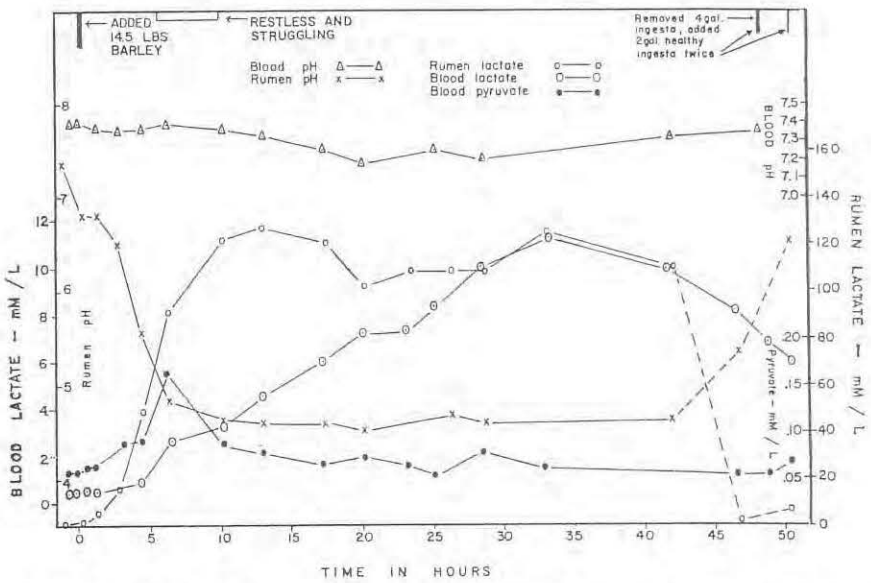

Figure 3. Typical data from an experimental case of lactic acidosis in a Holstein steer weighing about 130 kg.

The dose of barley was approximately 50 g/kg.
The animal was equipped with a rumen fistula and recovered after the ingesta was removed.

Production of similar detrimental changes to the acidosis syndrome by simple chemical solutions

The Australian workers attempted to reproduce the acute disease by adding lactic acid to the rumen to maintain the rumen pH at 4 for a long period but this only led to a mild acidosis with no hemoconcentration. In subsequent experiments they introduced lactic acid solutions at pH 3 or abomasal contents adjusted to pH 3 with lactic acid directly into the duodenum. This procedure produced severe acidosis, lactic acidemia and some reduction of blood volume followed by recovery after termination of the infusion. In neither of the two groups of experiments were the lactate concentrations and isomeric compositions reported. Finally, they incubated ground wheat with ruminal fluid for 24 hours at 37° C *in vitro*. The product had a lactate concentration of 150 mM/l and pH 4.4. When infused into the duodenum the sheep developed typical signs of wheat engorgement and was moribund within 20 hours. Changing the infusion solution to normal fluid restored the animal to health (49).

In the only other study of this type (17, 21) an attempt was made to study the deterioration in function that resulted when a lactic acid

solution of known isomeric composition (57L:43D) was introduced into the emptied washed rumen of otherwise healthy rumen-fistulated cattle. Three experiments were conducted in one of which a lactic acid solution (dose of total lactate 47.1 mM/kg at pH 3.60) was used alone, in another the lactic acid solution was rendered very hypertonic by the addition of mannitol and, in the third, the effect of hypertonic mannitol alone (72.5 mM/kg) was studied. The first animal developed D-lactic acidosis and became depressed but recovered. The second animal developed signs and chemical changes comparable to those of the acutely engorged animal and died within nine hours while the third developed severe hemoconcentration and moderate L-lactic acidosis (21). The latter animal died after the experiment was terminated. It was concluded that dehydration and lactic acidosis may be critical components in the pathogenesis of the disease. In animal No. 2 the total dose of lactic acid and lactate in the form of its salts was 42 mM/kg but the dose of undissociated D-lactic acid was estimated to be only 11.7 mM/kg (pH was 3.60) and the dose of mannitol was 49 mM/kg.

Treatment

There have been few controlled studies of the treatment of lactic acidosis. In one such study prednisolone was found to be ineffective (37). A preliminary report of another study indicated that most medical approaches involving drugs are ineffective, including several for which extravagant claims have been made (19). The best chance of recovery follows complete emptying of the reticulo-rumen either by surgical means or by use of repeated flushing via a large diameter stomach tube. A transplant of ingesta from a healthy animal, if available, seems to promote recovery and restoration of the rumen epithelium (17). Alternatively, a conservative approach to treatment using oral antibiotics and water in repeated doses plus parenterally administered electrolyte solutions and other drugs has been recommended (13, 14, 19). There is only limited documentation of the efficacy of this approach, however.

Complications of acidosis; related problems

Treatment is not the answer to the problem of acidosis because it is expensive and invariably followed by a set-back and an unthrifty period. Some of the pathological sequelae have been reported. These include a variety of lesions of the gastro-intestinal tract including rumenitis with invasion by fungi or Spherophorus organisms, ulceration of omasum, abomasum or duodenum, and also problems in other body organs such as the liver, kidneys, and central nervous system.

Other syndromes have been considered to be attributable to or associated with the acidosis syndrome. Among these is laminitis which has been tentatively attributed to the absorption or release of histamine (40). Although laminitis or some type of soreness of the feet does occur in some cases of acidosis it may also be observed in grain-fed animals in which there is no evidence of acidosis. The term, "founder", applied to the acidosis syndrome appears to be a misnomer since severe laminitis in conjunction with acidosis is rare. In young intensively-fed animals profound changes in the structure of the foot may occur in laminitis but this condition does not appear to be attributable to acidosis (38, 39).

Another related problem is the serious economic problem of liver abcesses. This problem is believed to be secondary to some degree of acidic damage to the ruminal epithelium which may enhance the entry of microorganisms and their passage to the liver via the portal circulation. This problem has been reviewed in an attempt to evaluate the efficacy of antibiotic given with the feed to reduce the incidence of liver abcesses (50). Further studies on the pathogenesis and control of this important problem are indicated.

Feedlot bloat is not directly related to acidosis but has some interesting facets that may bear on the latter syndrome.

Of particular interest was the finding that *Peptostreptococcus elsdenii* increased in numbers in the rumens of bloated animals in one study (24). Since it was noted earlier that this organism is normally present in low concentrations, this finding raises the question that it may serve a protective role against acidosis. *Streptococcus bovis* was also present in increased numbers so it can be assumed that appreciable amounts of lactate were being formed and utilized. However the organism appeared to generate a slime that led to foam formation and increased the risk of bloating. This is but one of a large number of theories on the etiology of bloat and is by no means confirmed.

The control of ruminal fermentations by inoculation of pre-adapted ingesta (23) or by antibiotics (33) afford promising leads for future developments. Unfortunately the available data on the spectra of antimicrobial drugs against rumen organisms is limited (2). New methods of processing animal feeds and the increasing use of additives may create new problems to be characterized and controlled.

As ruminants are pushed harder on concentrated feeds under intensive conditions, a condition that is intermediate between a normal fermentation and lactic acidosis may develop. This state has not yet received adequate study but it may be accompanied by abnormally acidic conditions (pH about 5.0-5.5) and excessively high concentrations of volatile fatty acids rather than lactic acid. Very high levels of VFA have been observed in the rumen contents of some animals that died suddenly on high grain diets (41). Also abnormal accumulation of butyrate has been

recorded in cattle on a diet of sugar beet crowns and tops (14). Butyric acid is the most toxic of the VFA, hence this finding may have significance in the pathogenesis of the disease on some diets. It was reported earlier (42) that VFA concentrations decline in the rumen if a lactic acid fermentation develops. The possibility that there may be some forms of abnormally acidic fermentation associated with unusually rapid production of VFA merits investigation.

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