Ruminal Metabolic Acidosis

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Ruminal acidosis is a metabolic disease of cattle, sheep and possibly other ruminants which is characterized by ruminal and generalized systemic acidosis resulting in anorixia, weakness, prostration, coma and frequently death. The condition has been called engorgement toxemia, rumenitis, toxic indigestion, rumen overload, grain overload, rumen acidosis or lactic acidosis. In the biochemistry of acid-base physiology, there are two broad classifications of acidosis. The first is respiratory acidosis which is usually a compensatory hypoventilation which increases the Pco_2, resulting in acidosis of respiratory origin. The second classification is referred to as metabolic acidosis. This is the type of acidosis we will be concerned with in this article.

Metabolic acidosis is a serious acid-base disturbance commonly encountered in cattle and sheep. The condition can be caused either by a loss of base such as found in cases of diarrhea or excessive loss of saliva, or by a gain of acid by the body. There are several ways that a gain of acid can occur in the ruminant. First, the absorption of acid from the rumen as in grain overload lactic acidosis. The second cause would be an incomplete oxidation of fat as occurs in acetonemia. The third cause of acid gain is the incomplete oxidation of carbohydrates resulting in the accumulation of organic acids due to anaerobic glycolysis. This normally occurs in severe dehydration resulting in diminished tissue perfusion and metabolic oxygenation. A fourth cause of acid gain is the excessive tissue catabolism which yields sulfates and phosphates resulting in the formation of inorganic acids. The last cause of acid gain is due to renal overload or impaired renal function. These are the major causes of metabolic acidosis in the bovine. We shall limit our discussion to the first, that being grain overload lactic acidosis.

Etiology

The cause of ruminal acidosis is overfeeding of the ruminant with readily fermented carbohydrates. This can occur by the sudden intake of a large quantity of concentrate feed such as when a cow gets into the grain bin by mistake. It may also result from a too rapid change in the diet resulting in the excessive intake of high energy feeds. Ruminal acidosis is frequently seen in animals that have been off feed for some time and then suddenly start eating. Acidosis may occur when the roughage intake is decreased but the concentrate intake remains the same.

Ruminal acidosis can occur in animals that have been on extremely high levels of readily fermented carbohydrates for an extended period. This occurs most frequently in heavy feeder cattle and in high producing dairy cows.

Whatever the source of the change, the primary cause of ruminal acidosis is the inadequate adaptation of the animal to a highly digestible carbohydrate source.

Physiopathology

Ruminant digestion is a highly specialized form of digestion in which fermentative digestion occurs on a massive scale. The reticulum and rumen form a fermentation vat where food enters and is held while bacteria and protozoa digest the food particles. These organisms digest carbohydrates, proteins and lipids by a method of fermentation, deamination and hydrolysis into simplified metabolic building blocks (monosaccharides, amino acids, fatty acids, etc.) and into the basic chemical ingredients of carbon, hydrogen, oxygen and nitrogen. These substances are then utilized by the microorganism for their own growth and reproduction. The organisms as well as the numerous breakdown products are then digested by the host animal either through ruminal absorption or by further digestion in the abomasum and intestine. Among the many products of microbial digestion are the volatile fatty acids (VFA) which are characteristic end products of this fermentation process. These volatile fatty acids; acetic, propionic, and butyric acids are normally present in the rumen fluid of animals fed a highly digestible roughage at molar percentages of approximately 70% acetic, 20% propionic, 8% butyric and 2% miscellaneous acids (formic, valeric, and succinic). The normal rumen pH on this diet would be approximately 6.5. Normal blood has a pH of 7.45 and the packed cell volume is approximately 35 percent. As the diet becomes higher in readily fermented carbohydrates, the volatile fatty acids are drastically changed in composition and the rumen is lowest when the diet consists of large quantities of hay or poor quality roughage (high in celluloses, hemicelluloses and lignin) and highest when the diet contains large quantities of soluble sugars or starches. The concentration of acetic acid is in reverse proportion to propionic acid. The amount of lactic acid in the rumen fluid of animals fed high levels of roughage is usually less than 2.0 percent.

In ruminal acidosis there is an initial decrease in rumen pH (from 6.5 - 5.5). This decrease is primarily due to an increase in organic acids such as formic, valeric and succinic, but not lactic. As the pH is lowered there is a change in the rumen microflora. First, the number of protozoa begin to decrease, then there is a decrease in the number of normal gram negative bacteria. These bacteria are replaced by an

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For clinical synopsis see following page.

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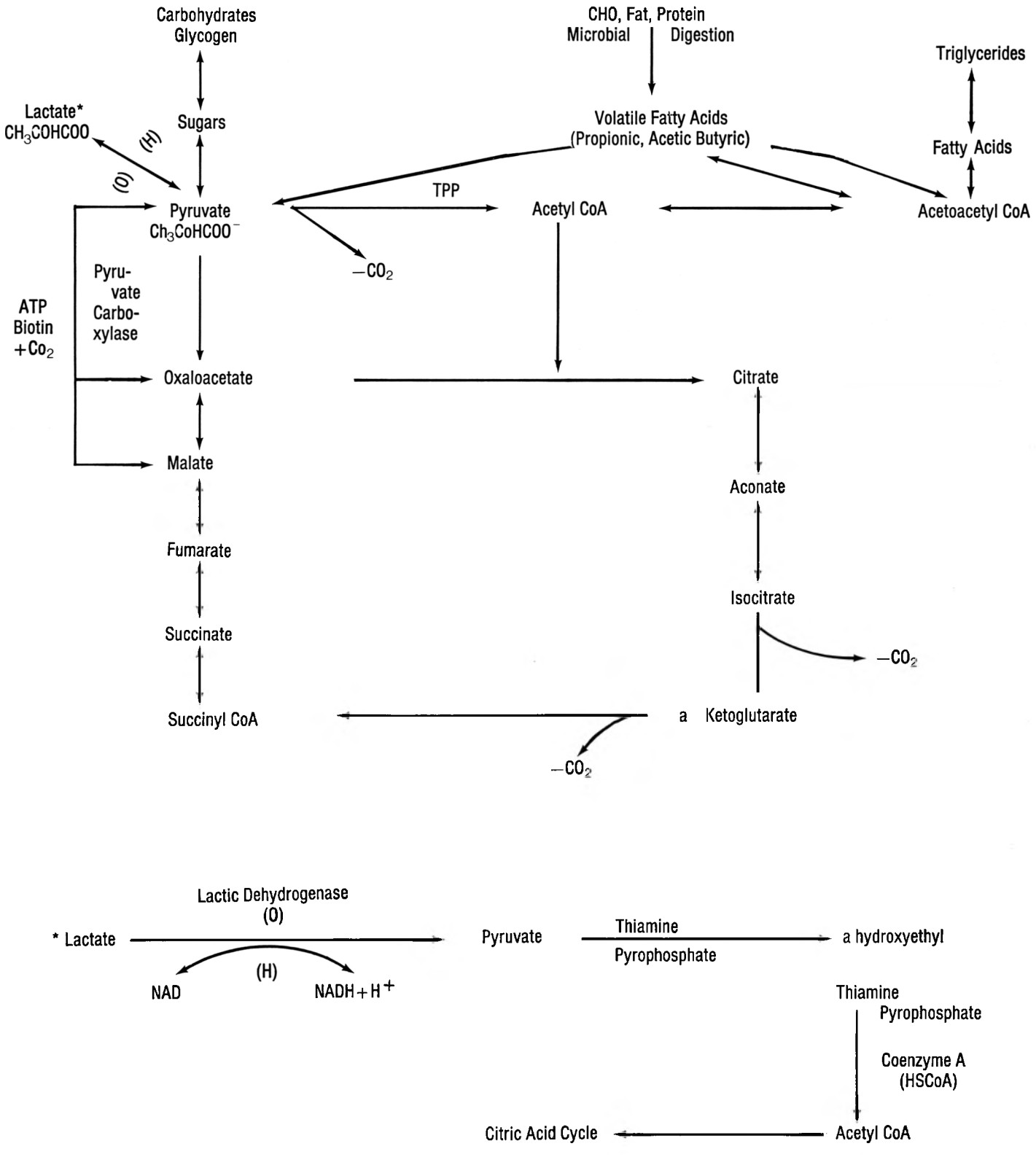
Chart No. 1

METABOLIC PATHWAYS



increased number of gram positive organisms (*Streptococcus bovis*). As the pH becomes lower (4.5 - 5.0) there is an increased production of lactic acid and a decrease in both gram negative bacteria and in *Streptococcus Bovis*. These organisms are replaced by lactobacilli species (gram positive rods) whose optimum pH is below 5.0. By 24 hours after overfeeding, lactic acid bacteria are the most numerous organisms in both the rumen and the cecum. The concentrations of coliform and *clostridium perfringens* are also significantly increased and are generally higher in the cecum than in the rumen. At this pH there are very few protozoa remaining. With an increase in the lactic acid concentration and a lowering of rumen pH, there is a depletion of intraruminal bicarbonate; when rumen pH reaches 4.0 or lower there is complete inhibition of rumen motility. There is also an increase in ruminal fluid osmotic pressure with a consequent increase in rumen fluid volume at the expense of body tissue fluids. Severe dehydration result, with hematocrit values often exceeding 50 percent.

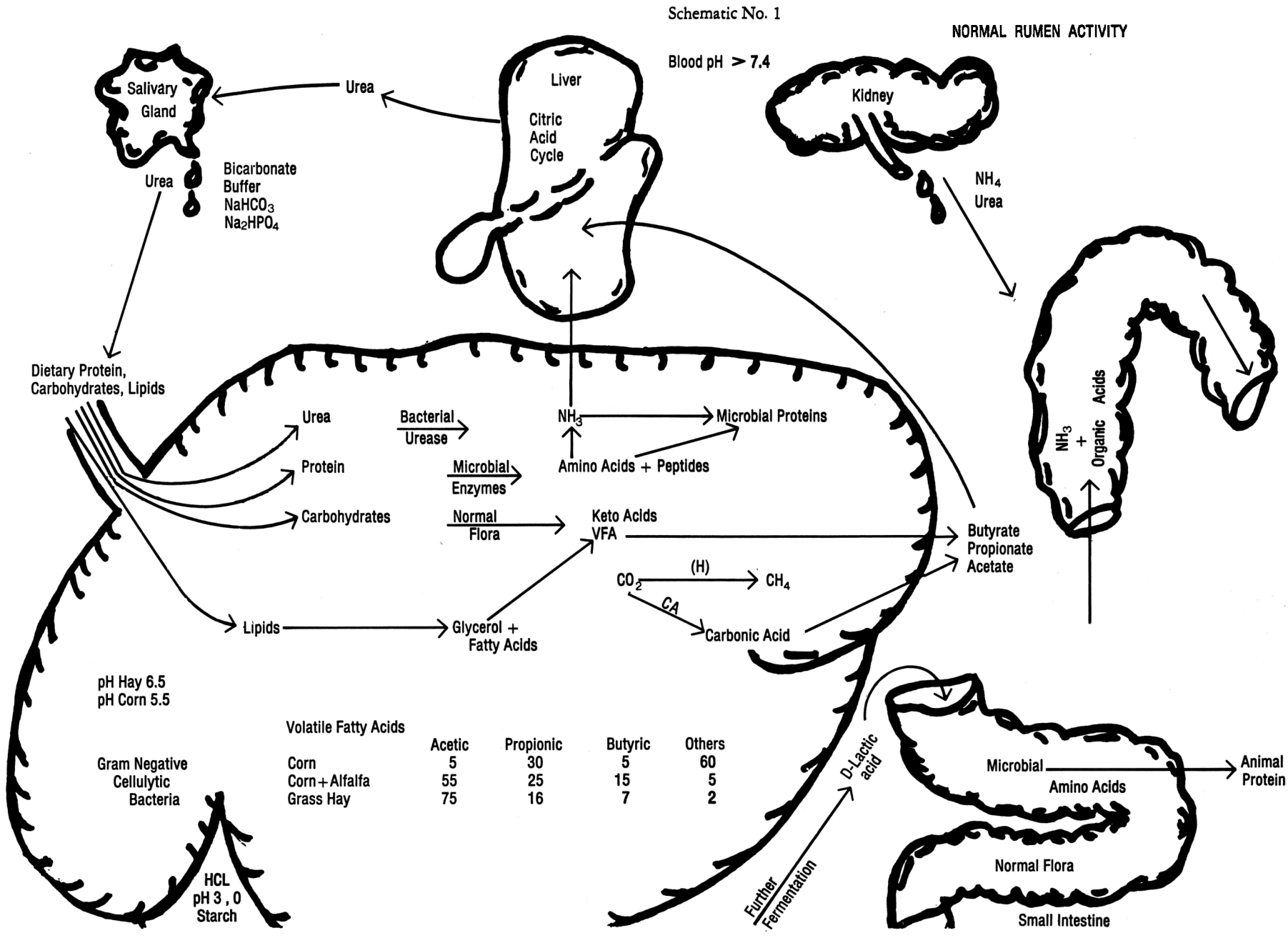
As the process continues, a set of chain reactions occur. As more lactic acid is produced, the pH is lowered which in turn gives an ideal medium (pH5) for the growth of *Lactobacilli species* bacteria which in turn produces more lactic acid. Lactic acid can be a source of blood glucose by first being oxidized to pyruvic acid which in turn can become glucogenic or the pyruvate can be decarboxylated to form acetyl CoA which enters the tricarboxylic acid cycle. In fact, blood glucose and pyruvate concentration may be increased two to three times normal in lactic acidosis. However, because of the extremely rapid production, the rapid absorption from the rumen and the lack of adequate oxidation, lactic acid concentration may increase to as high as 25 times normal levels. The total lactic acid (D+L lactic acid) concentration may be twice that measured because only the L-form is measured. The L(+) isomer of lactic acid is rapidly metabolized and the D(-) isomer is poorly metabolized. The plasma D(-) isomer may increase as much as ten fold. In addition to an increased absorption of VFA and lactic acid, there is an increased absorption of other organic acids (formic, succinic) and CO_2 which forms carbonic acid. The net result is the lowering of the blood pH to dangerously low levels. Because of the additional tissue destruction and dehydration, there is additional metabolic acid production and inadequate perfusion which lowers tissue pH even further. Very often in peracute cases, rumen pH is below 4.0 and blood pH is below 7.2. In these cases there is an uncompensated metabolic acidosis present. Any time the hydrogen ion concentration of the body results in a pH below 7.0 and it remains there for any extended period of time, the animal will die. In peracute cases, death may occur within 12 to 24 hours. For those which survive the peracute disease there develops a rumen stasis, increased rumen histamine concentration, and probably endotoxin formation which results in a severe chemical rumenitis with an increase in bacterial and toxin absorption. Endotoxin has been measured using limutus amebocyte, lysate, in sheep and a steer that were engorged with grain. Compounds considered as possible toxic factors include lactic acid, histamine, tyramine, tryptamine, ethenol, bacterial endotoxin and other unidentified toxic factors. There are varying degrees of rumenitis present, but in any case the ruminal epithelium becomes ulcerated and necrotic leading to increased absorption of toxins, bacteria (primarily *Fusobacterium necrophorium*) and fungi. Very often there is metastatic (either intraperitoneally or through the portal circulation) infection of other organs, principally the liver. The fungi are members of the family Mucoraceae (mucor, rhizopus and absida). Rumenitis caused by fungi is much more severe and extensive than bacterial rumenitis and is often fatal.

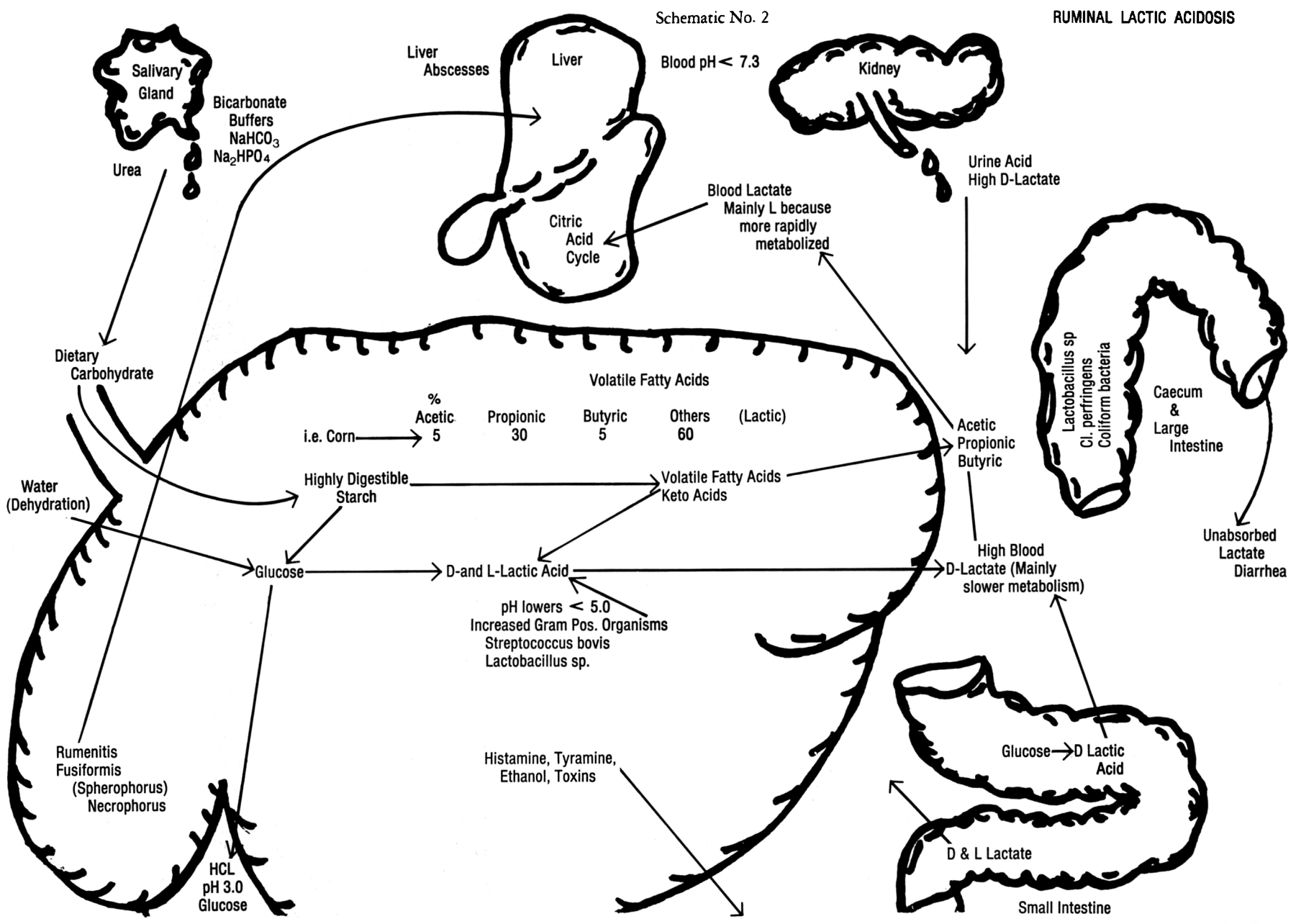
Clinical Signs

Peracute cases die within 12 to 24 hours. Signs in acute cases vary depending upon severity. Anorexia and lethargy are quite consistent findings. This is evidenced by the animal standing off by itself, showing little interest in food or water. The animal may be kicking at the abdomen. There is decreased rumen motility or in most cases a stasis. In mild cases there is a gray-green soupy diarrhea that becomes a watery, yellow-green and often foamy in more severe cases. An animal may lose up to 10 percent of its body weight due to diarrhea. Bloat and colic occur in approximately 25 percent of the cases. There may be an early fever, 40-41°C (103-106°F) but this is only transient unless there are severe ulcerations with subsequent peritonitis or hepatitis. Generally, the temperature is below normal, 37 to 38.5°C (99 to 101°F). The pulse rate is increased to 110 to 150 per minute and is often thready. Central nervous signs are manifested first by muscular tremors, followed by a drunken, staggering gait. The animals appear to be blind with no preservation (menace) reflex. However, the pupillary light reflex is not affected. These animals will become recumbent in 24 to 48 hours and they often take the typical parturien paresis (head turned into the flank) attitude. Many of these animals will go into a coma and then die. Some animals make a temporary recovery and then have a relapse in 2 to 3 days, apparently due to rumenitis and peritonitis. Many of these animals become chronically sick and are slow to recover. They may get up but have a tucked-up appearance due to pain, dehydration and rapidly developing emaciation. These animals show signs of lameness due to laminitis. Cases of chronic acidosis or "low-grade" acidosis show signs of chronic anorexia, lameness, rumenitis, peritonitis, hepatitis (liver abscesses) and decreased milk production with lower butter fat in dairy animals.

Clinical Pathology

The pH of the ruminal fluid will drop from a normal 5.5 to 6.5 (7.5 to 8.0 in sheep) to a pH of 4.5 to 5.5 in mild cases and below 4.5 in severe cases. A rumen pH of 4.0 or below is extremely critical. The pH of the blood will drop in





proportion to that of the rumen. Blood pH will drop from a normal of 7.45 to 7.0 or less. Blood pH of 7.0 or less almost invariably terminates in death of the animal. Urine pH will fall to about 5.0. The hemocrit will rise from a normal of 30 to 32 percent up to 50 percent or higher, depending on the degree of dehydration. In the terminal stages, there is an extreme elevation in blood glucose. Milk butterfat may fall from a normal of 4.0 to 5.0 percent or more to a low of 3.0 percent or less.

Hematology results will vary from normal to a neutrophilic leucocytosis with a left shift, depending upon the degree of rumenitis, abomasitis or hepatitic involvement. The SMA-12 profile will also vary considerably depending upon secondary involvement of other organ systems. As previously mentioned, there may be a hyperglycemia. When there is rumenitis or hepatitis (abscess formation) there may be an elevated serum glutamic oxalacetic transaminase (SGOT), serum alkaline phosphatase (SAP) and occasionally an elevated lactic dehydrogenase (LHD). Because of thiaminase activity in the rumen with a subsequent decrease in thiamine pyrophosphate (TPP), there is a decrease in pyruvate decarboxylase, which results in elevated blood pyruvate levels.

Pathological Changes

The pathologic changes of acute ruminal acidosis are congestion and edema of the lungs, congestion of the meningeal blood bessels, and swelling, edema and dilation of the ventricles of the brain, myocarditis with suffusion and ecchymotic hemorrhages of the epicardium. The rumen papillae are swollen and edematous. There is a mucous exudate with hyperemia and hemorrhage of the mucosa. There are areas of detached mucosa and epithelial necrosis. Frequently, there are rumen ulcers and occasionally peritonitis and adhesions in the peritoneal cavity. The kidneys are enlarged with congested medullas. Microscopic changes in the brain include perivascular and perineuronal edema, increased congestion and vascularity and death of neurons. Vascular changes of the extremities with evidence

Chart No. 2

RUMINAL LACTIC ACIDOSIS
PHYSIO-PATHOLOGICAL CHANGES

	Low CHO High Roughage	High CHO 2.3 Grain ⅓ Hay 60 - 40	Excess CHO 90% Grain
Acetic	75%	55%	0-10%
Propionic	16%	25%	25-35%
Butyric	7%	15%	0-10%
Others (Lactic, Formic, Succinic)	2%	5%	50-90%
Blood Pyruvate	<2.0 mg%	<2.0 mg%	2-8 mg%
Rumen Protein	Normal	Normal	Histamine, Tyramine formation
Rumen pH	6.5	5.5	<4.0
Bicarbonate Buffer	Adequate	Adequate	Depletion
Blood pH	7.45	7.30	<7.10
Urine pH	8.00	7.50	<6.00
Butterfat	4.3%	3.8%	3.0%
Ruminal Osmotic Pressure	Normal	Slightly Increased	Extreme increase
Rumen Motility	3 every 2 minutes	Same	Rumen Stasis
Rumen Epithelium	Normal Rough	Some Redness	Smooth Inflamed Epithelium Clinical Rumenitis
P.C.V.	35%	40%	>50%
Rumen Flora	Protozoa →	Still Adequate	Protozoa Die
	Gram-Negative Bacteria →	Minor Change	Increase in Strep. bovis, Lactobacilli, Cl. perfringens & Coliforms (Endotoxins)
Abomasum	Normal	Normal	Abomasitis
Liver	Normal	Some Fatty Changes	Fatty Infiltration Hepatic Abscesses Peritonitis
Extremities	Normal	Normal	Laminitis

of laminitis is rather common. Pododermatitis occasionally occurs.

Diagnosis

A diagnosis is usually made from the history and symptoms. Confirmation is usually made by clinical pathology and in terminal cases by necropsy. The major diseases that may be confused with lactic acidosis are enterotoxemia (or other clostridial diseases), polioencephalomalacia, and meningoencephalitis. Very often these conditions are sequela to acidosis. Hemophilus septicemia may be differentiated by respiratory signs, and elevated temperature. Parturient paresis is restricted to those animals near parturition. The condition may be confused with acute hepatic insufficiency but there is usually jaundice associated with primary hepatitis.

Treatment

The treatment of ruminal acidosis will depend to a large extent upon the acuteness and severity of the condition. In peracute or acute acidosis when the animal is in critical condition, the first thing to consider would be rumentomy and an emptying of the rumen contents. Fill the rumen with 15 to 20 liters of fluid and then re-empty. An alternative to rumenotomy and emptying the rumen is oral lavage. Pump out the rumen contents with an all purpose pump (Marine bilge pump BM-124P, Beckson Mfg., Bridgeport, Connecticut) or siphon off rumen contents with a Kingman stomach tube. Fill the rumen 2 to 3 times with several liters of fluid and re-empty. With either of the above procedures the rumen must be reinoculated with approximately 15-20 liters of fresh rumen contents. An alternative to flushing the rumen is to administer 20 to 40 liters of water orally to alter the osmotic pressure and dilute the acidic state.

The oral administration of antacids and mild cathartics such as magnesium oxide, magnesium hydroxide, magnesium carbonate or magnesium sulfate are indicated in addition to intravenous infections. Magnesium oxide is the most effective antacid followed by magnesium hydroxide. Magnesium sulfate or sodium sulfate is the most effective saline cathartic. Care must be taken with the use of magnesium oxide and magnesium hydroxide because of their strong alkalizing effect and the resulting electrolyte imbalance. Alkalosis can result with the excessive use of magnesium oxide or magnesium hydroxide and they are contraindicated in animals in the absence of acidosis. Serum potassium may be significantly depressed with the excessive use of magnesium sulfate.

Initial doses of antacids are given at the rate of 1 g. per kg. of body weight. This can be followed by repeated doses of ¼ to ½ the initial dose every 6 to 12 hours. The antacid is best dissolved in 8 to 12 liters of warm water to insure adequate dispersion in the rumen.

The oral administration of antibiotics has been found effective in lowering total volatile fatty acid production and particularly lactate production. The most effective antibiotics for reducing lactate are bacitracin methylene disalicylate, capremycin disulfate, novobiocin and oxamycin. Erythromycin, hygromycin B and tylosin tartarate have been moderately effective in lowering lactate. Chlortetracycline, streptomycin sulfate, penicillin and monensin have not been found very effective in reducing lactate. Monesin, a biologically active compound produced by *Streptomyces cinnamonesis*, increases propionic acid and decreases both acetic and butyric acid. However, the total ruminal volatile fatty acid concentration is unchanged.

In acute lactic acidosis, systemic dehydration and blood pH must be corrected with supportive therapy. The most important factors to consider are dehydration and low blood pH. Therefore fluids containing balanced electrolytes and sodium bicarbonate are given intravenously. For moderate to severe acidosis, sodium bicarbonate should be given intravenously at the rate of 2.0 to 4.0 mEq/kg of body weight.

An isotonic sodium bicarbonate solution can be prepared by dissolving 13 g of $NaHCO_3$ in 1 liter of sterile water or 5 percent dextrose. This solution will contain 156 mEq/L HCO_3 and 156 mEq/L Na+. An isotonic solution of bicarbonate in saline can be prepared by dissolving 26 g of sodium bicarbonate in 4 liters of half normal (0.425 percent) saline or by mixing 1 liter of isotonic sodium bicarbonate (13 g $NaHCO_3$/L and 1 liter of isotonic (0.85 percent saline). This provides a solution containing 78 mEq/L HCO_3, 78 mEq/L Cl-, and 156 mEq/L Na+. This is an isotonic solution. Stronger solutions can be used with caution.

A simplified formula has been devised for field calculation of the amount of bicarbonate deficit in metabolic acidosis. The formula is:

0.3 X Body wt. in kg X base deficit (-base excess)

The average base excess (B.E.) measurements in animals with metabolic acidosis are:

Severity of Clinical Signs	Base Excess
mild	-4 to -6
moderate	-8 to -11
severe	-14 to -16

The majority of cases fall in the -8 to -11 range.

Therefore a calculation of the mEq of bicarbonate of a 450 kg animal with moderate clinical acidosis (10 percent dehydration) would be:

0.3 X 450 X -10 = 1350 mEq of bicarbonate required

The above solution of bicarbonate in isotonic saline contains 78 mEq/L. Therefore it would take $\frac{1350}{78}$ = 17.3 liters of this fluid.

Care must be taken not to overdose with sodium bicarbonate because of the danger of producing alkalosis which can be just as fatal as acidosis. Also be careful not to use a lactated ringer's solution in cases of lactic acidosis.

A 450 kg animal with 10 percent dehydration would require 45 liters of fluid for rehydration. The balance of fluids can be provided by normal saline or 5 percent dextrose solutions.

Calcium-magnesium solutions have been given subcutaneously with apparent benefit in some cases.

Thiamine hydrochloride at the rate of 5 to 10 mg/kg

administered intravenously or intramuscularly as an initial dose and followed by the same dose for 2 to 3 days will help prevent polioencephalomalacia which often accompanies severe ruminal acidosis. B-complex vitamins may be given orally and parenterally on an empiracal basis.

The administration of antihistamines at the rate of 0.5 mg/kg is usually considered to be of some value in helping to prevent the laminitis associated acute rumenitis. Systemic and oral antibiotics are indicated to help control secondary bacterial infections associated with the rumenitis and hepatitis.

Field experience has shown that oral thiabendazole in normal anthelmintic dosage is helpful in controlling the development of secondary mycotic rumenitis.

Prevention

A multitude of management factors are involved in the increased incidence of ruminal acidosis in feedlot cattle. Management is most critical during the following periods: 1) starting cattle on feed, 2) graduating cattle to higher concentrate rations, 3) changes in the weather, 4) long periods on a finishing diet, and 5) when cattle are extremely hungry due to feeding problems or errors.

As a first line for prevention, avoid a sudden change from roughage feed to highly fermentable rations. Drastic changes in ration will cause severe alteration in rumen microflora and microfauna. Abrupt increases in concentrate causes increased ruminal concentrations of propionate and total acids, and decreases the concentration of butyrate, and acetate:propionate ratio. It also decreases the percentage of milk fat, total solids, and ruminal pH. Therefore it is important for ruminants to be adapted from roughage feed to high-energy rations by a gradual reduction in the roughage-to-grain ratio. Begin with a mixture of 25 percent grain and 75 percent roughage. Gradually change this ration over a period of 30 days to a 85 percent grain (concentrate) and 15 percent hay (roughage) ration. Occasionally rations are formulated that contain 95 to 100 percent concentrate. The management consideration in feeding full concentrate rations is extremely critical. Attempts to rear calves on rations completely devoid of roughage have been unsuccessful. The ideal ratio of concentrate to roughage for beef cattle on full feed is 88:12; for heavy producing dairy cows 60:40; and for lambs on full feed 70:30.

It is generally believed that slow adaptation procedures allow the development of adequate numbers of lactic acid-utilizing bacteria in ruminal ingesta. It has been observed that 3 ruminal lactic acid-utilizing bacteria, *Megasphaera elsdenii, Peptococcus asaccharolyticus,* and *Selenomonas ruminatum*, became more plentiful in the rumen ingesta of an animal slowly adapted to a high-energy feed. However, the feeding of rumen inoculum containing these organisms have not proven beneficial in preventing lactic acidosis.

Animals should be separated and fed according to their size, condition, state of health and possible even breed. Heavier cattle or lambs that are accustomed to eating grain will be more likely to overeat when exposed to an excess than will young weaning animals or pasture fed animals. Also, healthy animals are more likely to overeat than sick or stressed animals. The breed of animal may be a factor in that it has been shown that there are differences in breed susceptibility to acidosis. Brahman cattle develop laminitis more frequently than cattle of the English breeds. Also blood lactate levels increase more rapidly in the Brahman than in Herefords and Angus, whereas Holstiens appear to be more resistant to acidosis and laminitis than the standard beef types.

Coarse roughage should be provided when animals are first given access to high concentrate feeds. Adequate bunk space should be provided in order than animals have an opportunity to feed normally. The type of grain (oats, barley, sorghum grain, corn, wheat) has little influence on the volume or composition of volatile fatty acids except barley increases butyrate and decreases propionate. However, the mill process does affect the speed of fermentation and the finely ground grains are more likely to produce excess acid production, the feeding of high concentration of which results in the condemnation of more livers than the feeding of high concentrations of corn.

The addition of alkalinizing agents to the ration of heavy grain fed cattle has not been reported as preventing rumen acidosis but it has significantly increased feed consumption and improved feed efficiency. Alkalinizing agents such as magnesium oxide, magnesium carbonate, and bentonite and whey products appear to have little effect on rumen pH although they significantly reduce the percent rumen propionate which in turn leads to an increase in milk fat percent in dairy cattle. The optimum pH for cellulolytic activity is between 6.7 and 7.1 and fiber digesting activity is reduced considerable by lowering the pH. Limited research indicates this alteration in cellulolytic activity is a result of the altered metabolism of ruminal microorganisms or by the reduction in the proportion of fiber-digesting microorganisms. This may explain the increased digestibility of feeds when buffers are consumed by ruminants. Alkalinizing agents have appeared to be more beneficial in dairy cattle than in feedlot cattle on high concentrate rations.

The addition of limestone $CcaCO_3$ and dolomitic limestone appear to have little effect upon rumen fermentation. However, they may be beneficial by increasing the digestibility of starch in the lower gut.

Field experience has demonstrated that the oral supplementation of broad spectrum antibiotics such as the tetracycline group have been helpful in controlling lactic acidosis. Antibiotics definitely lower the amount and severity of rumenitis and liver abscessation.

The addition of fat such as tallow as a replacement for a portion of the grain in the ration appears to lower the production of lactic acid and therefore may be of benefit in helping control ruminal lactic acidosis.

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