EFFECT OF DIETARY BUFFERS ON RUMINAL AND BLOOD PARAMETERS OF LACTIC ACIDOSIS IN STEERS

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CHAPTER I

INTRODUCTION

The vast majority of cattle in the U. S. today are finished on high-concentrate rations. These diets are inducive to a metabolic disease alternatively known as "lactic acidosis," "acute impaction," "rumen overload," and "grain engorgement." The acute and chronic forms of this metabolic disorder differ only in the degree of ruminal production of lactic acid and volatile fatty acids and an animal's ability to cope with the stress caused by excessive amounts of these compounds. Because each animal differs in its response to having over eaten, it is difficult to distinguish between the two forms of this disease in experimental animals. Unless the rumen of an afflicted animal is manually cleared, washed and reinoculated with rumen fluid from a healthy animal, the acute form of lactic acidosis frequently results in death of the animal, and is certainly a source of economic loss to the feedlot cattle industry. However, deaths due to lactic acidosis constitute a very small percentage of total deaths in feedlot cattle. A study at Monfort Feedlot in Colorado indicates that of 426,000 cattle fed over a 14-month period, deaths due to lactic acidosis accounted for 0.95% and 0.0095% of the total deaths and total cattle fed, respectively (Braddy, 1976).

The chronic or subclinical form of lactic acidosis may cause a greater loss. Subclinical acidosis is initiated by some stimulus which

causes an animal to over consume a high-energy ration (e.g. feed bunk empty for a long period or changing weather conditions). This is followed by reduced consumption of the ration for a few days and then possibly again over eating. Continual pattern of this type can result in marked reductions in weight gains and feed efficiencies of feedlot cattle. When Uhart and Carrol (1967) substituted a grain ration ad libitum for a chopped alfalfa hay diet steers were observed to stop eating within 2 or 3 days and fasted for as long as 6 days. A reduction in intake for this period could cause a delay in finishing of up to 15 days and reduce the producer's profit considerably.

The most economical solution to this metabolic disorder is prevention. At present there appears to be three possible means of prevention. The gradual adaptation of animals from a roughage diet to a high-concentrate ration should prevent initial acidosis by allowing the ruminal microbial population to change to a type that is capable of digesting large amounts of starch. The intra-ruminal inoculation of both sheep (Huber, 1973) and cattle (Bond et al., 1975) with microbes obtained from animals fed high-concentrate rations reduces the probability of acidosis and increases feed intake, allowing a more rapid acceptance of a high-energy diet by the animals. Both methods have been shown to be satisfactory when adapting animals to high-energy rations; and the feeding throughout the finishing period of a lyophilized preparation of rumen fluid, as a form of continuous inoculation, may prove effective in preventing subclinical acidosis.

The use of dietary buffers has been suggested as a possible means of preventing lactic acidosis. Several types of buffers have been investigated for their effect on animal performance or their ability to

prevent acidosis (Bhattacharya and Warner, 1968; Emerick, 1976; Emery et al., 1964; Kay et al., 1969; Nicholson et al., 1963; Prigge et al., 1975). These studies have produced conflicting data as to the effect of dietary buffers on animal performance, but have shown that buffers tend to increase ruminal pH, and volatile fatty acid concentrations while preventing the accumulation of excessive amounts of lactic acid.

Many feedlots in the Oklahoma Panhandle utilize ground, ensiled high-moisture corn as the major component of finishing rations. Work by Johnson et al. (1974) has shown this processing method to be more conducive to producing digestive disturbances in feedlot cattle than whole shelled or ground corn rations. Recent studies by Prigge et al. (1975) have indicated sodium bentonite, potassium bicarbonate, and dolomitic limestone individually to be effective dietary buffers in high-moisture corn diets. An experiment was therefore undertaken to compare the effects of sodium bentonite, sodium bentonite plus potassium bicarbonate, or sodium bentonite plus dolomitic limestone on ruminal and blood parameters or subclinical lactic acidosis when steers were fed an excessive quantity of high-moisture corn.

CHAPTER II

REVIEW OF LITERATURE

Causes and Effects of Lactic Acidosis

A variety of feeds have been shown to be conducive to the development of lactic acidosis in ruminants. The most prevalent types of diets which cause acidosis are those composed of feedstuffs such as grains or fruits with readily digestible carbohydrates. As reported by Slyter (1976) those feeds containing large amounts of starch, sucrose, or glucose include wheat, barley, corn, unripe green corn standing in the field, oats, rye, milo, sorghum, molasses, cabbage, brewers dried grains, kiawe beans, potatoes, grapes, peaches, pears, fooder beets, and sugar beet leaves with the beet top attached. Although Dunlop (1972) appears to doubt that grain processing increases the incidence of lactic acidosis, work by Johnson et al. (1974) indicates that certain processing methods are more conducive to the development of lactic acidosis. Using steers fed 2.5% of their body weight in two equal 30-minute feedings daily Johnson et al. (1974) simulated subclinical lactic acidosis by feeding 50% of the evening meal on the day prior to sampling, then feeding 100% or 150% of the normal morning allotment of a sorghum or a corn based diet. If the quantity of feed offered was not consumed within 30 minutes any refusal was placed directly in the rumen through rumen cannulae. These researchers

compared (1) dry-rolled sorghum with increasing degrees or micronization and (2) whole shelled corn (WSC) with ground (GC), steam-flaked (SFC) and ground, ensiled high-moisture corn (HMC). In the first trial, ruminal pH was 0.5 units higher at 1 and 2 hours post-feeding with the dry-rolled than the micronized sorghum rations, although there was little difference in pH attributable to degrees of micronization. However, increasing micronization resulted in higher ruminal lactic acid concentrations. As one would expect from the pH values the dry-rolled sorghum diet produced the least amount of lactic acid. For the corn-based diets, the decline of the ruminal pH was much less with WSC while the GC, SFC, and HMC depressed pH to approximately the same level. However, the ruminal pH of cattle fed the SFC diet was the slowest to return to normal. All of the processed corn diets produced higher lactic acid levels than WSC with the high concentration persisting considerably longer in those steers fed the diet containing SFC. Research by Galyean (1975) supports the concept that grain processing increases the probability of lactic acidosis. When comparing dry-rolled corn with steam-flaked (SFC), ground, ensiled high-moisture (HMC) and propionic acid treated high-moisture corn (AHMC), he found that the greatest disruption of starch granules occurred in SFC, as indicated by in vitro gas production, and that HMC produced the largest in vitro dry matter disappearance, lowest ruminal pH and greatest total concentration of volatile fatty acids. Since foods with large quantities of starch, sucrose or glucose are more conducive to producing this disease, any processing method which increases the availability of lactic acid precursors would increase the likelihood of acidosis.

Ruminal Parameters of Lactic Acidosis

The introduction of an excessive amount of readily fermentable carbohydrate into the rumen may first result in an increase in the production of volatile fatty acids (VFAs) and lactic acid; both of which, due to dissociation, cause a tremendous increase in free hydrogen ions (Ryan, 1964a; Scarisbrick, 1954). Slyter et al. (1965) reported that at ruminal pHs below 6.0 VFA production was repressed. Corresponding with the reduced VFA production the initial signs of rumenitis have been observed at a pH of 5.85 with irreversible damage to the ruminal epithelium occurring at a pH of below 5.5 (Kay et al., 1969). Below a pH of 6.0 volatile fatty acids are the major buffering component of ruminal contents (Dunlop, 1972; Mackenzie, 1967). These prevent the continued decline of pH for a time; however, with an increasing acidity of ruminal ingesta their production decreases (Slyter, 1976) while the rate of absorption increases (Masson and Phillipson, 1951; Williams and Mackenzie, 1965). Thus, as the pH of the rumen declines past 5.0 the concentration of VFAs displays a pronounced decrease while the level of lactic acid remains high or increases (Ryan, 1964a).

The concentration of ruminal lactic acid has been indicated to reach a critical level at approximately 100 mM (Johnson et al., 1974). When Prigge et al. (1975) modified the technique of Johnson et al. (1974) by attempting to produce subclinical acidosis with the feeding of 200% of the morning allotment of a ground, ensiled high-moisture corn diet, they found a lactic acid concentration of 136 mM of strained rumen fluid associated with a pH of 6.0 at 2 hours post-feeding. At 4 hours after feeding the pH had dropped to 5.4. In light of Uhart and Carrol's work (1967) this level of lactic acid appears to be

unusually high at this pH. When Uhart and Carrol (1967) switched eight steers from a chopped alfalfa hay diet to an ad libitum rolled-barley and milo ration they observed that the animals stopped eating as the pH of the rumen ingesta was depressed to 4.81 and reported a level of lactic acid of only 100 mM at this pH. In one sheep engorged with 3 pounds of cracked wheat Ryan (1964a) observed that 16 hours before death the sheep had a ruminal pH of 4.22 with a lactic acid concentration of 100 mM. Dunlop and Hammond (1965) state that, although the ruminal pH of lactic acidotic animals may decrease to values very close to the pK of lactic acid, pH 3.88, with lactic acid concentrations up to 327 mM, pHs between 4 and 4.5 are more common with the level of lactic acid not exceeding 240 mM. Apparently, when animals eat excessively the concentration of lactic acid in the rumen is quite variable and may be influenced by several different ruminal factors. Two of the most important are (1) the effect of free glucose on the ruminal metabolism of lactic acid and (2) the rate of lactate absorption from the rumen.

A depression of rumen pH below 5 when animals over consume a high-concentrate diet is severely detrimental to many species of the rumen's microbial population, but selectively favors an increase of lactobacilli, a lactic acid producer (Hungate et al., 1952). The accumulation of lactic acid in the rumen is further enhanced by pH depression because some of the lactate-utilizing bacteria are also destroyed before the population of lactobacilli increases (Slyter, 1976). Papers cited by Slyter (1976) indicate that at pHs above 5.0, increasing the acidity of the rumen contents increases the activity of free ruminal amylase and reduces the rate of glucose fermentation. As the availability of glucose

increases, lactic acid production would increase (Ryan, 1964b). Ryan (1964a) observed levels of up to 15 mM glucose in rumen fluid from sheep which died as a result of engorgement with 3 pounds of cracked wheat. He also noted that this value is much higher than any previously observed. Slyter et al. (1974), after fasting heifers for four days and changing them from a forage diet to an ad libitum corn-based diet, noted free glucose levels peaking (.72 mM) between 1 and 4 hours post-feeding. Perhaps the concentration of free glucose in the rumen is not as important as its effect on lactate utilization. Hishinuma et al. (1968) have suggested that glucose possibly has an inhibitory effect on lactate metabolism. These researchers demonstrated that, when glucose was added to a lactate medium on which pure cultures of Selenomonas ruminatium were being grown, the utilization of lactate was almost completely inhibited for 4 to 7 hours depending on the level of glucose supplied. Recently this work has been indirectly supported by Bond et al. (1975). In heifers treated the same as those of Slyter et al. (1974), Bond et al. (1975) noted that if an abnormally high concentration of ruminal glucose was present at 7 hours after feeding, the level of lactic acid in the rumen increased and was maintained for more than 24 hours post-feeding. Thus, it appears that increased free ruminal glucose levels, due to the death of many species of microbes which cannot tolerate low pHs, not only results in increased lactic acid production, but also may inhibit the metabolism of any lactic acid already present.

Another factor which can influence the accumulation of ruminal lactic acid is the rate of absorption of the undissociated acid and its anion from the rumen. Free lactic acid is absorbed much faster than

its anion (Dunlop, 1972; Dunlop and Hammond, 1965; Williams and Mackenzie, 1965). Williams and Mackenzie (1965) reported that the rate of absorption was much faster at pH 4 than 5. This was probably caused by an increase in the quantity of the undissociated acid at the lower pH. At a pH of 4.79 as much as 90% of any lactic acid present is dissociated into the anion form while at a pH of 4.24 approximately 30% is free lactic acid (Dunlop and Hammond, 1965).

Several other factors also decrease the absorption of lactic acid from the rumen. Williams and Mackenzie (1965) noted that the absorption of lactate was depressed by the presence of VFAs at a pH of 5. These researchers also reported that an increase in the tonicity (osmolality) of the rumen of approximately 140 milliosmolar reduced lactate absorption by 10%. Perhaps the most noticeable factor affecting lactate absorption is rumen motility. The absorption of lactate from the rumen is dependent on motility (Huber, 1976). Juhasz and Szegedi (1968) demonstrated that when ruminal pH was lowered below 5 by lactic acid solutions, complete rumen stasis occurred after 2 hours. The infusion of lactic acid into the duodenum has been shown to almost immediately inhibit the amplitude and frequency of ruminal contractions (Bruce and Huber, 1973). Thus, the observation of Juhasz and Szegedi (1968), that two hours elapsed before complete ruminal stasis occurred, is probably an indication of the time necessary for sufficient quantities of lactic acid to pass from the reticulo-rumen to the duodenum for lactate absorption.

Acidosis increases the tonicity of the ruminal contents. A value of approximately 280 milliosmolar has been shown to be the fairly constant osmolality of the normal rumen (Warner and Stacey,

1965). Ahrens (1967), after fasting heifers for 24 hours and engorging them with 26 kg of cracked wheat, found that 16 hours later the ruminal pH had dropped to 4.0 while the lactic acid concentration had increased to 165 mM and osmolality increased to 523 milliosmolar. When the heifers were dosed with 25 kg of crushed green pears the ruminal pH, osmolality, and lactate concentrations were 4.28, 343 milliosmolar, and 119.4 mM, respectively, at 13.75 hours post-engorgement. Huber (1971) noted that in sheep in which the ruminal osmolality increased from 255 to 401 mosmoles per liter, 61% of the increase could be accounted for by lactic acid. Danelli et al. (1945) and Huber (1971) have shown that, when the osmolality of the ruminal contents greatly exceeds that of plasma, appreciable amounts of water enter the rumen from the plasma. This influx of fluid contributes to a common symptom of acidosis, diarrhea. The weight loss due to diarrhea can be considerable and require 2 to 3 weeks for an animal to recover (Huber, 1976.)

Two isomers of lactic acid, L and D, are normally produced in the rumen during lactic acidosis. Intraruminally, the metabolism of these isomers does not significantly differ and there is evidence that their interconversion is possible (Lee and Matrone, 1971). However, their systemic metabolism does differ critically and is of ultimate importance in lactic acidosis. These differences in systemic metabolism will be discussed in the next section. Of significance in the rumen is the difference in production of these isomers. Dunlop and Hammond (1965) state that the ratio of the D to L isomer of lactic acid may vary widely and cite papers indicating from .5 to 6 ratio. Elevated levels of a readily available carbohydrate substrate such as glucose

may be necessary for establishment of ruminal microbes capable of forming D-lactate (Ryan, 1964b). That L-lactate accumulates before D-lactate when a diet is changed from forage to concentrate has been reported by Slyter et al. (1974). These researchers also noted that if an animal remains on a high-energy diet and becomes acidotic, or if an animal is inoculated with rumen fluid from a concentrate-fed animal when changed from a forage to concentration ration, then the concentration of L and D lactate peak together. However, a wide variation in the ratio of the L isomer to the D was observed. Since the ruminal conditions necessary for the production of D-lactate and its quantitative relation to L-lactate have not been well defined, much work needs to be done to characterize this parameter of ruminal lactic acidosis.

Blood Parameters of Lactic Acidosis

The absorption of free hydrogen ions and dissociated fatty acids and lactate anions from the rumen combined with the transfer of water into the gastrointestinal tract during lactic acidosis results in serious systemic changes. Initially there is an increase in the hematocrit with a corresponding rise in hemoglobin concentration (Dunlop and Hammond, 1965; Jensen et al., 1954; Telle and Preston, 1971). The increase in hematocrit from normal values of 27 to 33 up to 40 to 55 (Dunlop and Hammond, 1965) is due to (1) the release of red blood cells from the spleen as a result of contractions caused by an acidotic stimulated adrenalin release (Kilburn, 1966) and (2) to the efflux of water from the blood into the hypertonic rumen causing dehydration and decreased plasma volume (Dunlop and Hammond, 1965; Mackenzie, 1967; Telle and Preson, 1971).

Another major effect of lactic acidosis is that on the blood bicarbonate buffering system and thus, the blood pH. Uhart and Carrol (1967) reported an initial blood pH of 7.37 and noted that animals stopped eating when the pH fell to 7.29, but resumed feed consumption at a blood pH of 7.31. Although the depression of blood pH in sheep infused intraruminally with lactic acid was not as great as that observed in the studies of Uhart and Carrol (1967), Telle and Preston (1971) observed that the rate of decline in blood pH reflected the concentration of blood lactate. The rate at which the pH declines depends initially on the ability of the bicarbonate buffering system to cope with increasing lactate levels.

The rate of absorption of D and L-lactate from the rumen does not differ appreciably (Dunlop, 1972; Dunlop and Hammond, 1965). As lactic acid is absorbed into the cardiovascular system both isomers dissociate to form the lactate anion and a free hydrogen ion. The hydrogen ion is either excreted by the kidneys or combines with bicarbonate to form carbonic acid which readily dissociates into water and carbon dioxide (Dunlop, 1972; Guyton, 1971; Huber, 1976). The first metabolic step in the utilization of most of the lactate in the body is believed to be the dehydrogenation in the liver of lactate to pyruvate (Dunlop, 1972). Work by Dunlop and Hammond (1965) shoed that L-lactate disappears rapidly and exponentially. This indicates that an animal has large amounts of available L-lactic dehydrogenase. However, they also observed that the decline of D-lactate in the blood was very slow, indicative of very low liver concentrations of D-lactic dehydrogenase.

A L-lactate anion may be metabolized in a manner which results in the net production of one bicarbonate ion and two molecules of carbon

dioxide. Large persistent increases in blood pH and bicarbonate have been observed when sheep were intravenously infused with L-sodium lactate, but not after D-lactate was infused (Braide and Dunlop, 1969). Therefore, the metabolism of D-lactate must be effected by the small amount of D-lactic dehydrogenase in the liver or kidney (Tubbs, 1965) or the concentration in the blood may be reduced by excretion into the urine (Dunlop, 1972).

The bicarbonate buffering system with a pK of 6.1 is not very powerful at normal blood pHs, but is very important because the concentrations of its components can be regulated easily, carbon dioxide by the respiratory system and bicarbonate ions by the kidneys. During acidosis the excretion of free hydrogen ions by the kidney tubules results in the production of sodium bicarbonate and thus increases the pH of the blood and attempts to correct acidosis (Guyton, 1971). If sufficient amounts of carbon dioxide, formed from the dissociation of carbonic acid, accumulate, the pH of the blood can be drastically lowered. However, an increased rate of carbon dioxide exhalation occurs due to stimulation of the respiratory system by the high blood carbon dioxide concentrations and lowered pH (Guyton, 1971; Huber, 1976). This activation of the respiratory system during lactic acidosis was shown by Juhasz and Szegedi (1968) by infusing intraruminally into sheep 16.2 g of glucose per kg of body weight. Before infusion the respiratory rate was 26 with a blood CO_2 value of 1.44 mM and 36 mEq of bicarbonate per liter of blood with blood pH 7.52. At 16 hours post-infusion the CO_2 content of the blood had risen to 2.07 mM and the respiration reached 86 as the bicarbonate level and pH declined to 32 mEq and 7.31, respectively.

The work of Uhart and Carrol (1967) is probably more indicative of the effect of lactic acidosis on the bicarbonate buffering system in animals which have over eaten. These researchers found that steers on an alfalfa hay diet had a blood pH of 7.37 with a bicarbonate level of 31.2 mEq per liter. After changing to an ad libitum rolled-barley and milo ration for a period of 2 to 3 days the animals stopped eating when the blood pH and bicarbonate concentration declined to 7.29 and 23.6 mEq per liter, respectively, and only resumed eating when the values returned to 7.31 and 28.3 mEq per liter. Values finally stabilized at 7.36 pH units and 34.4 mEq of bicarbonate per liter. The initial and stabilization values of blood bicarbonate concentrations of these animals are considerably higher than those of heifers fed a 37% concentrate diet by Bhattacharya and Warner (1968). They noted that the mean bicarbonate level was 21.6 mEq per liter of plasma and pH 7.38. Apparently the type of diet fed has an effect on the buffering capacity of the blood.

The Effect of Buffers on Animal Performance and on Ruminal Parameters of Lactic Acidosis

One proposed method for continued protection from the occurrence of acidosis is the addition of buffers to mixed, high-concentrate rations. The saliva of ruminants usually serves as a major buffering component of rumen contents. Normally, the rumen contents are well buffered between pHs 6.8 and 7.8 (Church, 1973), and are buffered to a greater extent when the ingesta tends to be acidic rather than alkaline (Broomfield et al., 1966). When high-concentrate rations are fed, the pH of the rumen lowers due to the quantity of organic

acids produced (Church, 1973). Oltjen et al. (1967) demonstrated that the type of concentrate fed affected the amount of saliva produced and, consequently, its buffering capacity. These researchers examined the stimulation of saliva flow by corn, wheat, barley and milo, and determined the amount of acid in milliequivalents required to lower the pH of saliva from pH 7.0 to 5.0. They found that the rate of saliva flow was significantly greater for corn than milo (1.6 vs 1.1 liter per hour), and that more acid was required to titrate the saliva of the corn diet than that of the milo. The rate of feed consumption has been shown to be inversely related to saliva stimulation (Bartley, 1976). Bailey (1958) noted that saliva production per g food fed for cows eating dairy cubes was one-fifth that of those eating hay, but pellets were consumed five times more rapidly. Sudweeks et al. (1975) have shown that increasing the amount of roughage in a concentrate diet increases the total chewing time. Since chewing is a primary stimulus for the secretion of saliva, the amount of saliva produced was probably increased which resulted in a greater buffering capacity of the rumen. Perhaps higher roughage levels in the diet do result in increased rumen buffering, but in meat animals this is usually not conducive to maximum production. Therefore, the possible effects of the addition of buffers to diets on animal performance and on acidosis prevention must be examined.

The effect of the addition of buffers to high-concentrate diets on animal performance has been extensively studied. However, it is difficult to compare some of these studies because of differences in diet and buffer types and quantities. The most studied buffer is sodium bicarbonate, although sodium carbonate, calcium carbonate, calcium hydroxide,

sodium acetate, sodium bentonite, combinations of sodium bicarbonate, ground limestone and potassium carbonate and of the potassium and sodium salts of bicarbonate have also been examined (Bhattacharya and Warner, 1968; Brethour and Duitsman, 1972; Emerick, 1976; Kay et al., 1969; Nicholson et al., 1963; Wise et al., 1965). The effect of sodium bicarbonate in high-energy rations on animal performance is at best inconclusive. The feeding of a ground shelled corn ration containing 2% sodium bicarbonate was shown to have no effect on feed intake or weight gains by lambs (Hoar et al., 1969; Hoar et al., 1970). However, Kromann and Meyer (1966) observed that, when a barley-based ration containing 5 or 12% sodium bicarbonate was fed to sheep, daily intake was reduced and total gain was significantly depressed. The feeding of 1 pound of sodium bicarbonate daily (approximately 5% of the total ration) to lactating cows reduced intake of a ground, shelled corn diet, had no effect on milk production, but significantly increased the percent milk fat (Emergy et al., 1964). When feeding a ground-oats and barley ration with 5.7% sodium bicarbonate Nicholson et al. (1962) found that steers ate more but gained less, causing a feed efficiency depression of 12%.

In contrast other researchers have shown that the addition of from 1 to 7.5% sodium bicarbonate to high-energy rations may be beneficial by increasing weight gains (Brethous and Duitsman, 1972; Kay et al., 1969; Nicholson et al., 1963; Wise et al., 1965). When steers were fed a rolled wheat ration containing 1% sodium bicarbonate Brethour and Duitsman (1972) observed an increase in daily gain and feed efficiency as a result of reduced feed consumption. Although Kay et al. (1969) noted an increase in intake when feeding a barley ration with 7.5% sodium bicarbonate to bull calves, an increase in feed efficiency and

average daily gain was also observed. Steers fed a ground shelled corn diet with 5% sodium bicarbonate have displayed increased feed consumption and daily gains, but depressed feed efficiency (Wise et al., 1965). Bhattacharya and Warner (1968) also found that a shelled corn diet containing 5% sodium bicarbonate increased feed intake; however, weight gains were not reported. When Nicholson et al. (1963) fed steers a rolled-barley ration containing 3% sodium bicarbonate, weight gains and feed consumption increased, but efficiency of gain decreased. Thus, the benefit of sodium bicarbonate addition to high-concentrate diets may or may not improve animal performance and efficiency of gain. Other compounds tested as buffers have produced similar conflicting results or have not been sufficiently examined to substantiate their effect on animal performance (Bhattacharya and Warner, 1968; Emerick, 1969; Emery et al., 1964; Hoar et al., 1969; Kay et al., 1969; Nicholson et al., 1962; Nicholson et al., 1963; Wise et al., 1965). Much work needs to be done to establish what effect different types and quantities of buffers in different diets have on animal performance before their addition to high-energy diets can be economically feasible.

Although the effectiveness of buffers in directly preventing lactic acidosis has not been extensively investigated, their effect on several parameters associated with lactic acidosis has been examined. Emery et al. (1964) reported that calcium carbonate slightly depressed ruminal pH in the lactating cow; however, the feeding of buffers to cattle generally increases the pH of the rumen contents (Bhattacharya and Warner, 1968; Emery et al., 1964; Kay et al., 1969). Prigge et al. (1975) demonstrated that all steers showed ruminal pH

depression when fed 200% of their morning allotment of buffered, high-moisture corn diets to simulate subclinical lactic acidosis. The steers fed rations containing either potassium bicarbonate, calcium carbonate, sodium carbonate, sodium bicarbonate, sodium bentonite or dolomitic limestone displayed a smaller depression in ruminal pH than those feed the unbuffered control diet. The potassium bicarbonate ration maintained pH the highest. When heifers were changed from a hay diet to a high-concentrate diet Tremere et al. (1968) found that intraruminal infusion of sodium bicarbonate, at 6% of the concentrates fed, extended the time before the heifers went off feed by one day to seven and prevented the ruminal pH from dropping below 6.0 before any reduction in intake was observed while the pH in animals not receiving the buffer fell to 5.4 before refusal of feed. However, the feeding of this buffer at 5% of the total ration rather than infusing it had no beneficial effects on preventing or delaying the animals from refusing their ration after changing from the hay diet.

A possible reason that the heifers of Tremere et al. (1968) went off feed lies in the effect buffers have on ruminal VFAs and lactate concentration. A variety of buffers have been shown to increase VFA concentrations in healthy animals (Bhattacharya and Warner, 1968; Emery et al., 1964; Kay et al., 1969; Van Campen and Matrone, 1960). The effect of the buffers on molar percentages of individual fatty acids varies greatly with the quantity and type of buffer fed and is not completely understood. However, the large increase in VFA appears to be of more importance. When Reid et al. (1957) intraruminally infused 750 ml of a 10% sodium carbonate solution into sheep fed a high-starch diet in an attempt to maintain the pH above 5.8, accumulation of lactic acid was obviated while a large increase in VFAs was observed.

observed. Tremere et al. (1968) reported that intraruminal infusion of sodium bicarbonate or potassium carbonate prevented the accumulation of lactic acid, but these researchers did not examine the response of VFA production to these buffers. Although ruminal VFA concentrations were not reported, Prigge et al. (1975) demonstrated that in steers fed an excessive amount of a high-moisture corn ration, the concentration of lactic acid was depressed by those diets containing buffers. As with the pH, the potassium bicarbonate ration was the most effective, showing a maximum level of 69 mM lactate. This quantity of lactic acid is well under the so-called critical level of 100 mM. These results indicate that adapted animals which refuse a buffered, high-concentrate ration may not be experiencing severe lactic acidosis. Instead the high concentrations of VFAs produced when animals over consume a buffered, high-energy diet may be acting as an intake depressant (Dowden and Jacobson, 1960; Montgomery et al., 1963). Thus, the buffers may be fulfulling their purpose in preventing lactic acidosis specifically, but not in preventing anorexia.

CHAPTER III

EFFECT OF DIETARY BUFFERS ON RUMINAL AND BLOOD PARAMETERS OF LACTIC ACIDOSIS IN STEERS[1,2]

Summary

A 4 x 4 Latin square design split twice on time was employed to study the effect of dietary buffers on ruminal and blood parameters of lactic acidosis in steers. Four Holstein steers fitted with permanent rumen cannulae were fed an 85% ground, ensiled high-moistured corn diet at a level of 90 g of dry matter per kg of metabolic body size in two equal meals daily. The feeding prior to the test meal was reduced by half and subclinical lactic acidosis was produced by feeding the daily allotment of feed in one meal, placing any refusal in the rumen through the cannulae. Effects of 2% sodium bentonite, 1% sodium bentonite plus 1% dolomitic limestone, and 1% sodium bentonite plus 1% potassium bicarbonate ($KHCO_3$) on ruminal pH, lactic acid, glucose, and volatile fatty acid (VFA) concentrations, and osmolality at 0, 1, 2, 4, 8, 12, and 24 hours post-feeding, and blood pH, pCO_2 and HCO_3 concentrations at 0, 2, 4, 8, 12 and 24 hours were examined. Differences from time zero across rations were compared.

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[1]Journal Article of the Agricultural Experiment Station, Oklahoma State University, Stillwater.

[2]J. L. Gordon, G. W. Horn, E. C. Prigge, F. N. Owens and D. E. Williams, Oklahoma State University, Stillwater, Oklahoma 74074.

The response in all parameters except ruminal pH fluctuated tremendously between the type of buffer fed. The ruminal pHs of all steers indicated that the most severe period for ruminal lactic acidosis was from 4 to 8 hours post-feeding. Steers fed the diet containing bentonite and $KHCO_3$ consistently displayed the highest pHs and had pHs at 4 and 8 hours (5.4 and 5.5 respectively) which were at least 0.2 units greater than steers consuming the other rations. By 24 hours after feeding the ruminal pHs of all steers had returned to levels above 6.0.

Maximum ruminal glucose and lactic acid concentrations and osmolality were observed 1 hour post-feeding. Steers fed the sodium bentonite plus dolomitic limestone ration had a significantly greater increase at 1 hour after feeding than the bentonite plus $KHCO_3$ fed steers ($P < 0.05$). By hour 2 the level of lactic acid in steers fed the bentonite plus dolomite diet had decreased so that the change from the zero hour for these steers was significantly less than the control steers ($P < 0.05$). No treatment effects were found in ruminal glucose and osmolality at any time or in lactic acid concentrations at subsequent times.

Time-course changes in ruminal total VFAs and individual acids were similar within steers consuming the same rations. Although the control steers had the highest total VFA concentration from 0 to 8 hours post-feeding, no differences between rations were found. Steers consuming the control, bentonite, and the bentonite plus $KHCO_3$ diets had maximum VFA concentrations at 2 hours post-feeding while the VFA level in the bentonite plus dolomite fed steers peaked at 1 and 4 hours after feeding.

Ruminally, the steers having eating the diet containing bentonite and $KHCO_3$ consistently had the highest pH and one of the lowest values observed in lactic acid, glucose, osmolality, and VFAs for up to 8 hours after feeding. Levels of the same parameters associated with the other 3 rations fluctuated widely and generally produced no consistent trends, although the steers consuming the bentonite ration had the second highest pH at all sampling times and lowest VFA level at 4, 8, and 12 hours post-feeding.

Because the respiratory system and kidneys can regulate the bicarbonate buffering system's components, the concentration of blood bicarbonate is an indication of an animal's ability to cope with systemic lactic acidosis. The bicarbonate decrease from time zero of steers consuming the sodium bentonite and the sodium bentonite plus dolomitic limestone diets was significantly greater than that of the control steers at 4 ($P < 0.05$) and 8 hours post-feeding ($P < 0.01$). Of most importance is that by 24 hours after feeding the steers fed the bentonite plus dolomitic limestone and the bentonite plus $KHCO_3$ diets had a blood bicarbonate concentrate which was physiologically more favorable to a resumed consumption of the high-moisture corn.

Introduction

The majority of feedlot cattle in the Oklahoma Panhandle are finished on ground, ensiled high-moisture corn. This processing method has been shown to be more conducive to producing lactic acidosis than feeding whole shelled or ground corn rations (Johnson et al., 1974). Recently it was observed that lactic acidosis causes approximately 0.0095% death loss in feedlot cattle (Braddy, 1976). Greater economic

loss may be realized from the subclinical form of this disease. Uhart and Carrol (1967) found that cattle suffering from lactic acidosis may fast for at least 6 days. Huber (1976) indicates that weight losses due to this disease may increase the feeding period for as long as 3 weeks, and thus, the producer's profit is reduced considerably.

The only economically feasible and effective means of treating this disease is prevention. Dietary buffers have been suggested as possible prevention agents. The work of Prigge et al. (1975) indicates that sodium bentonite, potassium bicarbonate and dolomitic limestone are fairly effective individually as dietary buffers for high-moisture corn diets. The purpose of this study, therefore, was to investigate the effectiveness of either sodium bentonite or combinations of sodium bentonite plus dolomitic limestone and sodium bentonite plus potassium bicarbonate in reducing ruminal and blood responses in steers fed a ground, ensiled high-moisture corn diet to simulate subclinical lactic acidosis.

Experimental Procedures

Four Holstein steers averaging 530 kg and fitted with permanent rumen cannulae were housed in a 6.1 x 6.1 m pen equipped with individual feeding stalls. A 4 x 4 Latin square design split twice on time was employed for feeding of ground, ensiled high-moisture corn rations containing either no buffer, 2% sodium bentonite, 1% sodium bentonite plus 1% dolomitic limestone or 1% sodium bentonite plus 1% potassium bicarbonate ($KHCO_3$). The buffers in the treatment diets replaced equivalent amounts of high-moisture corn (Table I). These rations were fed to each steer at a level of 90 g of dry matter per kg metabolic body size

TABLE I

COMPOSITION OF RATIONS

Ingredient[1]	Control	Sodium Bentonite	Sodium Bentonite & Dolomitic Limestone	Sodium Bentonite & $KHCO_3$
High-moisture corn	86.8	84.8	84.8	84.8
Supplement[2]	7.5	7.5	7.5	7.5
Cottonseed hulls	5.7	5.7	5.7	5.7
Sodium Bentonite	--	2.0	1.0	1.0
Dolomitic Limestone	--	--	1.0	--
$KHCO_3$	--	--	--	1.0

[1]Percentage of ration dry matter.

[2]Supplement contained the following ingredients in g/kg of supplement: cottonseed hulls, 300; dehydrated alfalfa meal, 300; soybean meal, 278.5; urea, 40; salt, 30; dicalcium phosphate, 25; calcium carbonate, 25; aurofac-50, 0.77; vitamin A supplement (300,000 I.U./g), 0.73.

in two equal feedings (8:00 a.m. and 4:00 p.m.) daily. The steers were trained to consume the total amount of feed offered in 30 minutes.

After adapting the animals to their assigned ration for 10 days a seven day sampling period was initiated with rumen and blood samples being taken on days 1 and 7. On the day prior to sampling the steers were fed 50% of their evening allotment of feed. At 8:00 a.m. the following morning, 100% of the daily ration was offered for consumption within 30 minutes. If any feed was not consumed after this time it was placed directly in the rumen through the rumen cannulae.

Rumen samples were obtained at 0, 1, 2, 4, 8, 12 and 24 hours post-feeding. Ruminal pH values were determined immediately after removal of the fluid from the rumen. Microbial action in the samples was halted by the addition of approximately 0.5 ml of 20% sulfuric acid per 100 ml of fluid following the straining of all samples through 4 layers of cheesecloth. The samples were then frozen, and quantitative determinations of lactic acid, free glucose, volatile fatty acids and osmolality were made at a later date.

An aliquot of strained rumen fluid was deproteinized with barium chloride, sodium hydroxide and zinc sulfate (Somogyi, 1945). Lactic acid concentrations in the protein-free filtrate were determined colorimetrically with ferric chloride (A.O.A.C., 1975). Quantitative analysis of free ruminal glucose was made by utilizing the "Glucostat Special" reagent set (Worthington Biochemical Corporation, Freehold, New Jersey). Centrifugation of rumen samples at 12,000 x g for 10 minutes was necessary for determination of the osmolality of the fluid by the freezing-point depression technique on an Advanced Osmometer (Advanced Instruments Incorporated, Newton Highlands, Massachusetts).

Volatile fatty acid (VFA) analysis of samples was conducted by the procedure of Erwin, et al. (1961) with a Bendix Series 2500 Gas Chromatograph. Column packing and gas flow specifications were reported by Hinman and Johnson (1974).

Blood samples were collected at 0, 2, 4, 8, 12 and 24 hours after feeding from the jugular vein of each steer utilizing heparinized needles and syringes. The blood samples were immediately placed in an ice slurry and analyzed for pH and pCO_2 within 30 minutes to an hour after collection. Quantitative determinations of blood pH and pCO_2 were done using a blood gas analyzer (Radiometer, Copenhagen, Denmark). These measurements were then utilized for determination of blood bicarbonate concentrations from a Sigaard-Anderson blood acid-base alignment nomogram (1963).

Statistical analyses on all data were done by standard procedures for Latin square designs split twice on time. Tests of significance between treatment means at the 0 hour were accomplished by the use of the least significant difference method protected by a preliminary F test. Differences between treatment means at subsequent times compared the changes from time zero for each ration using the above method. The statistical analysis of individual volatile fatty acids was performed on the quantitative amount of each acid.

Results and Discussion

There were no significant differences ($P > .05$) among rations, at zero time, for any of the ruminal or blood parameters. The response of ruminal pH to an abnormal amount of ground, ensiled high-moisture corn fed to simulate subclinical lactic acidosis is shown in Figure 1.

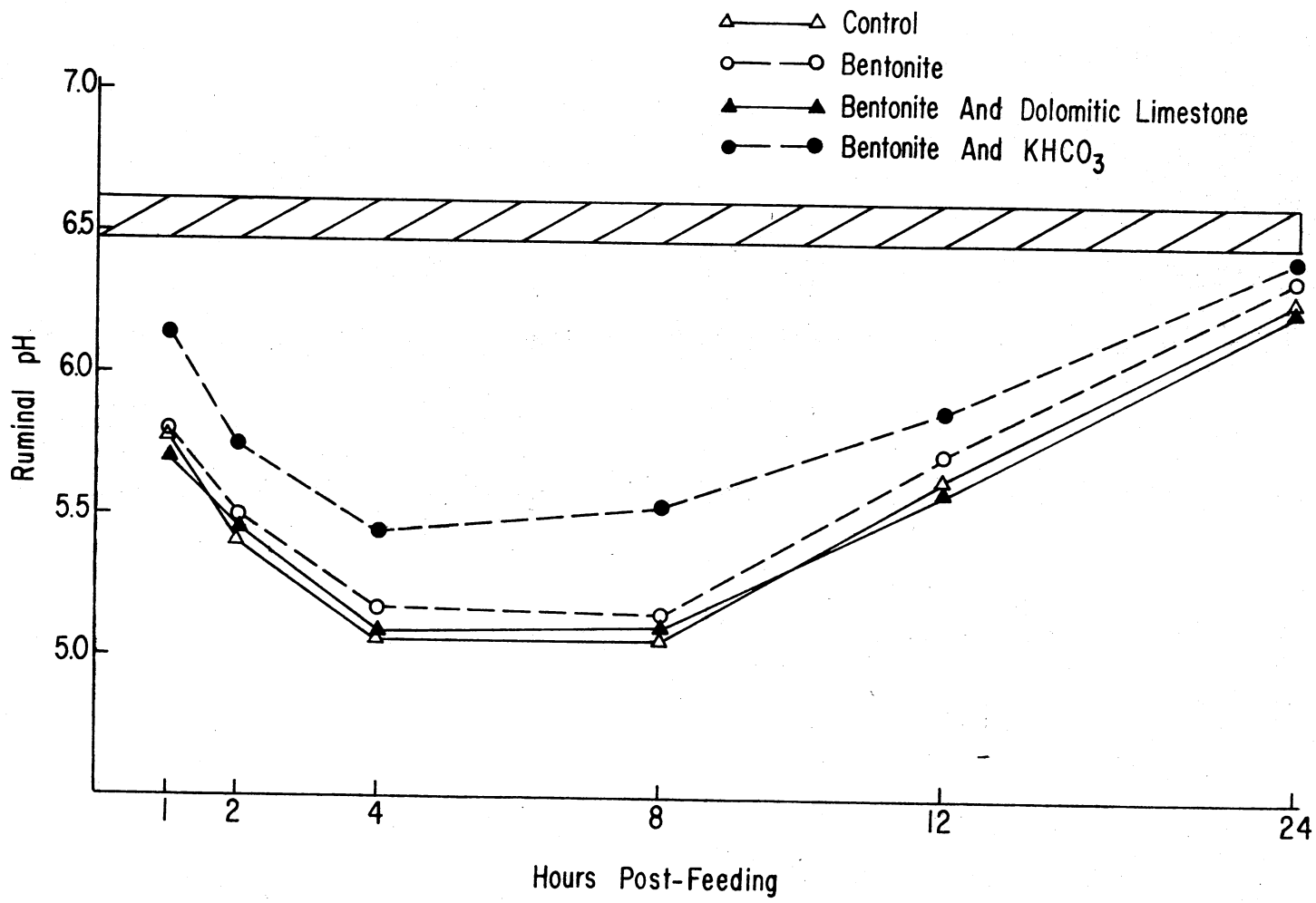

Figure 1. Time-Course Changes in Ruminal pH; Shaded Area Represents the Mean ± SEM pH of All Steers at Time Zero.

The rumen pH of steers fed the diet containing bentonite and $KHCO_3$ was consistently higher than that produced by any other ration. Maximum pH depression occurred at 4 to 8 hours post-feeding for all rations, indicating that ruminal lactic acidosis was most severe during this time period. During these times the pH associated with the bentonite plus $KHCO_3$ diet (5.4 and 5.5 at 4 and 8 hours post-feeding, respectively) was from 0.2 to 0.4 units greater than the ruminal pH of steers fed the other 3 diets. This may be of importance when one considers that irreversible damage to the ruminal epithelium begins to occur at a rumen pH below 5.5 (Kay et al., 1969). The pH produced by the bentonite plus $KHCO_3$ diet fell below this level only at 4 hours after feeding while pH values associated with the other rations were equal to or less than 5.5 at 2, 4 and 8 hours post-feeding. Thus, the ruminal epithelium of steers fed the control, sodium bentonite, and the sodium bentonite plus dolomitic limestone rations was exposed for longer periods of time to pHs capable of reducing the rumen's absorptive ability by damaging the epithelium. Differences in ruminal pHs between the steers fed either the control, sodium bentonite, or the bentonite plus dolomitic limestone diets were small at all hours post-feeding, although the bentonite fed steers had a consistently, but only slightly higher pH than the other steers. By 24 hours post-feeding none of the ruminal pHs had returned to the normal pH range of all steers at time zero; but all were well above 6.0. Means are reported in Appendix Table V.

The increase in ruminal lactic acid concentration (Figure 2) were substantial at 1 hour after feeding all rations, but did not exceed the critical level of 100 mmoles per liter quoted by Johnson et al.

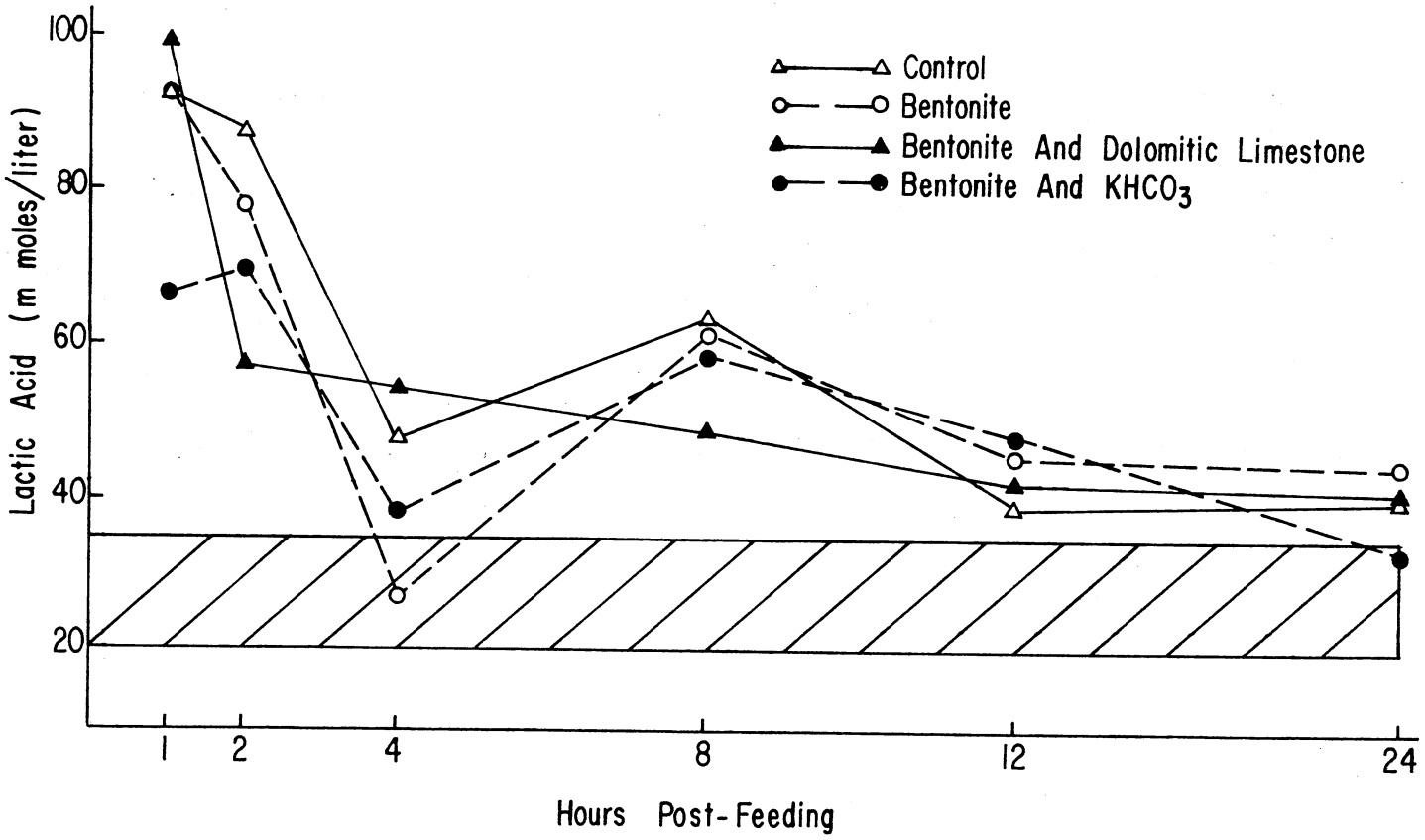

Figure 2. Time-Course Changes in Ruminal Lactic Acid Concentrations; Shaded Area Represents the Mean ± SEM Concentration of All Steers at Time Zero.

(1974). The concentration of lactic acid in steers consuming the control, sodium bentonite, and the sodium bentonite plus dolomitic limestone diets peaked at 1 hour post-feeding, but in those fed the diet containing bentonite and $KHCO_3$, the maximum lactate level was not reached until 2 hours after feeding. The lactic acid increase in those steers offered the sodium bentonite plus $KHCO_3$ ration was significantly smaller at 1 hour after feeding than that of steers fed the sodium bentonite plus dolomite diet ($P < 0.05$), but was not significantly different ($P > 0.05$) from that of steers fed the control or 2% bentonite diets. However, by 2 hours after feeding the change in lactic acid concentration from time zero was significantly greater for steers consuming the control diet than for those fed the bentonite plus dolomite ration ($P < 0.05$) with steers eating the other 2 rations being intermediate. Of interest is that 4 hours after feeding, the period which the ruminal pHs indicated that ruminal acidosis was most severe, the concentration of lactic acid in steers consuming the control, bentonite, and the bentonite plus $KHCO_3$ diets had decreased tremendously with the sodium bentonite ration producing a level well within the normal pre-feeding range of lactic acid concentrations. Since this decrease occurred with not one, but three rations it appears to be real. It is difficult to believe that this could be due to poor sampling technique because it would have had to occur in both replicates at 4 hours after feeding in all 4 periods of the Latin square. An increase in absorption of lactic acid due to decreased ruminal pHs could be a factor; however, the work of Williams and Mackenzie (1965) shows little effect of pH on the range of 7.5 to 5. Decreased production due to depressed ruminal pHs is also possible; but at 8 hours after feeding

when ruminal pHs were still relatively low the lactic acid concentration in steers fed the control and sodium bentonite diets had increased to a little more than 60% of the 1 hour value, while the steers consuming the ration containing the bentonite and $KHCO_3$ had 84% of their peak lactate concentration. Some other ruminal factor must have effected the production of lactic acid at 4 hours after feeding.

All steers displayed decreases in ruminal lactic acid concentration after 8 hours post-feeding. The lactate level in those steers fed the control, bentonite, and the sodium bentonite plus dolomitic limestone rations appeared to stabilize at 12 hours after feeding, but remained above the pre-feeding lactic acid concentrations. The sodium bentonite plus $KHCO_3$ fed steers showed a linear decrease in lactic acid concentration from 8 to 24 hours after feeding and had the lowest lactate level at the last sampling time. Means for each sampling time are shown in Appendix Table V.

The tonicity of the rumen contents increased by at least 150% in all steers at 1 hour after feeding (Table II). The rate of decline in rumen osmolalities was similar for steers fed all rations, and was characterized by a rapid decline from 1 to 4 hours post-feeding followed by a slower decrease during the 4 to 24-hour post-feeding interval. By 8 hours all ruminal osmolalities had essentially returned to the normal range of rumen tonicity at time zero.

Recently the concentration of ruminal glucose has received attention due to a possible inhibitory effect on lactic acid metabolism to acetic and propionic acids (Hishinuma et al., 1968; Slyter, 1976), thus, enhancing the accumulation of ruminal lactic acid (Bond et al., 1975). A marked increase from time zero and the maximum concentrations

TABLE II

RUMINAL OSMOLALITY[1]

Time Post-Feeding (hr.)	Ration			
	Control	Bentonite	Bentonite & Dolomitic Limestone	Bentonite & $KHCO_3$
0	258.1	246.7	221.2	250.5
1	395.4	394.7	386.5	378.1
2	337.0	343.5	309.6	337.0
4	279.6	251.7	293.0	271.6
8	234.7	247.6	244.7	260.6
12	239.0	243.7	241.5	248.6
24	247.7	245.6	229.9	232.0

[1]All values are reported as milliosmoles per liter.

of ruminal glucose in the steers of this study were observed at 1 hour after feeding (Figure 3). The control and the sodium bentonite plus dolomitic limestone fed steers had ruminal glucose concentrations at least twice that of the steers consuming sodium bentonite at 1 hour post-feeding with the ruminal glucose concentrations of steers fed the sodium bentonite plus $KHCO_3$ diet being intermediate. The glucose concentrations associated with the control diet were higher than those of the "buffered" rations for the first 8 hours after feeding. As with the lactic acid concentrations the steers fed the sodium bentonite and the sodium bentonite plus $KHCO_3$ diets showed a marked decrease in glucose concentrations at 4 hours and returned to higher levels by 8 hours post-feeding. Perhaps this decrease in glucose concentration at 4 hours after feeding allowed an increase in the ruminal metabolism of lactic acid by steers consuming these diets. However, the level of glucose in the control steers did not decrease at 4 hours with a subsequent increase at 8 hours as the lactate concentration of these steers did, while the ruminal glucose concentrations of steers fed the sodium bentonite plus dolomitic limestone diet responded in the opposite manner. Thus, the depression and subsequent increase of lactic acid in the steers consuming the control, sodium bentonite, and the sodium bentonite plus $KHCO_3$ diets can not be fully explained by corresponding levels of glucose and its possible inhibitory effect on lactate metabolism.

By 12 hours after feeding the level of glucose associated with all rations had returned to values slightly above the normal range at time zero and were well within this range of glucose concentrations by the 24 hour sampling time. Ruminal glucose concentrations are shown in

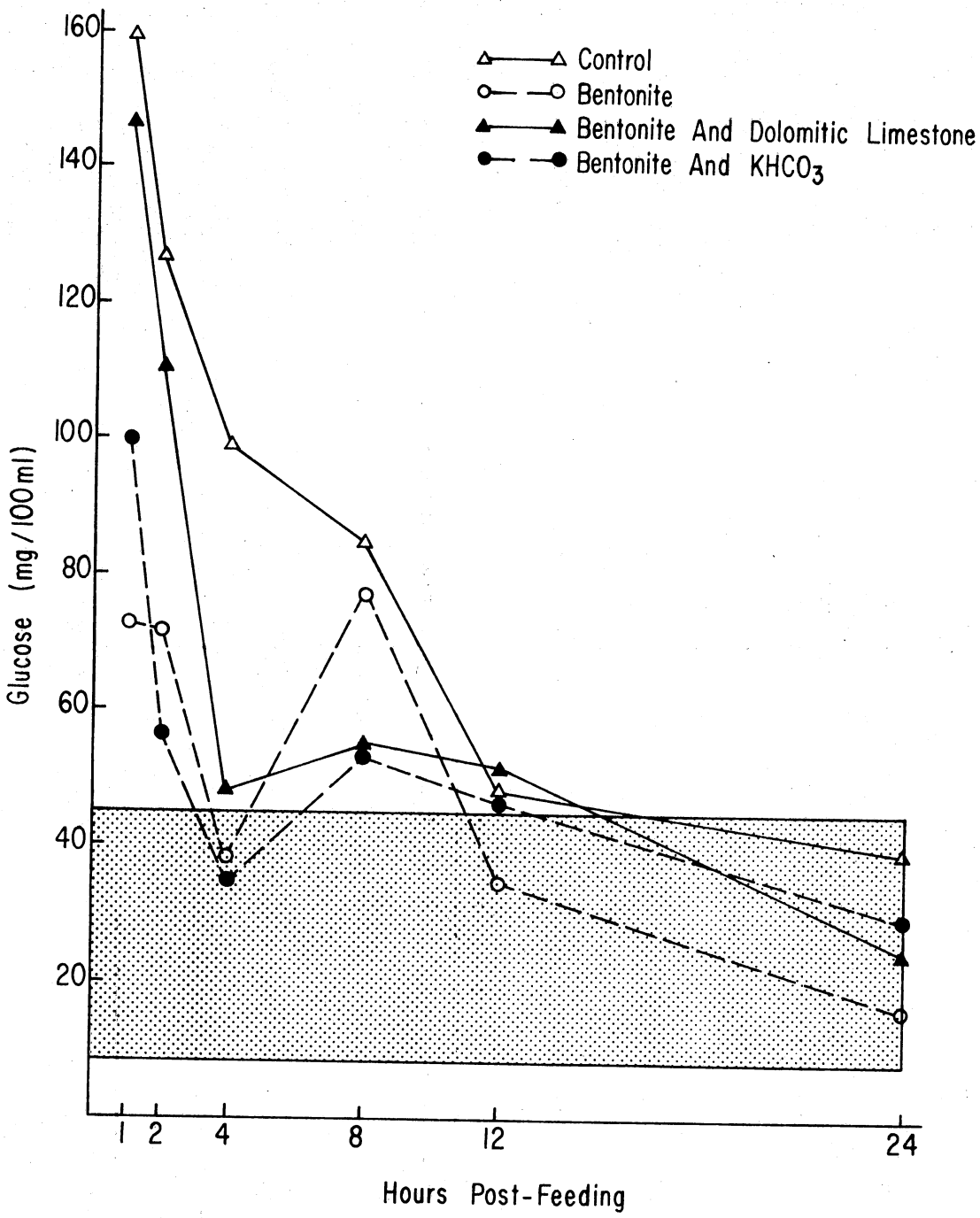

Figure 3. Time-Course Changes in Ruminal Glucose Concentrations; Shaded Represents the Mean ± SEM Concentration of All Steers at Time Zero.

Appendix Table V. The "normal range" of all the steers at time zero appears to be excessively high when compared to the values reported by Ryan (1964a) and Waldo and Schutz (1956). Therefore, a trial was conducted in which two of the four Holstein steers were fed the control diet as before and sampled to determine if the freezing and thawing of the strained rumen fluid caused starch hydrolysis of any remaining feed particles. A strained sample was centrifuges at 12,000 x g for 10 minutes for use as a control by removing all possible feed particles. Initial analysis of the fluid before freezing indicated concentrations of 2 and 5 mg of glucose per 100 ml of rumen fluid. Subsequent determinations at 2, 4 and 10 weeks after freezing indicated that the glucose concentration in the samples which were strained and centrifuged did not increase. However, one of the samples which was strained and immediately frozen showed an increase in glucose concentration of 750% or more on all analysis dates while the other sample increased 620% after 10 weeks. Although the results are not statistically conclusive, the possibility of starch hydrolysis in frozen rumen samples containing small particles of feed appears plausible.

Several researchers have reported that the feeding of buffers increases ruminal volatile fatty acid concentrations (Bhattacharya and Warner, 1968; Emery et al., 1964; Kay et al., 1969; Van Campen and Matrone, 1960). At time zero, the total ruminal VFA concentration of the steers fed "buffered" rations in this study was less than those consuming the control diet (Table III). After consuming an excessive amount of high-moisture corn steers fed the control diet displayed a higher concentration of VFAs for the first 4 hours after feeding than did those consuming the "buffered" diets (Figure 4). The maximum

TABLE III

EFFECT OF LACTIC ACIDOSIS ON RUMINAL VOLATILE FATTY ACIDS

Ration	Time Post-Feeding	Total	Volatile Fatty Acids					
			C_2[a]	C_3[b]	C_4[c]	C_5[d]	IC_5[e]	C_2:C_3
	(hr.)	(mmoles)/(liter)	(Molar %)					
Control	0	91.2	58.4	22.6	12.0	1.6	3.4	2.96
	1	160.5	57.6	24.0	11.4	2.5	2.7	2.89
	2	170.2	53.6	28.3	10.9	3.0	2.3	2.65
	4	154.9	54.2	28.1	11.0	2.6	2.0	2.95
	8	129.4	53.9	26.8	12.5	3.0	2.4	2.94
	12	100.1	51.5	25.6	14.9	3.2	2.1	2.64
	24	91.8	53.9	24.4	12.4	2.8	3.5	2.52
Bentonite	0	80.9	61.1	21.2	10.2	1.5	3.4	3.27
	1	150.5	58.4	23.2	11.7	2.0	3.1	2.87
	2	161.6	55.4	26.1	11.7	2.4	2.7	2.65
	4	130.7	53.7	25.9	13.0	2.8	2.5	2.61
	8	119.2	55.0	22.7	15.2	2.6	2.8	2.70
	12	100.6	53.6	23.0	15.2	2.7	2.7	2.52
	24	85.1	55.2	21.8	13.0	2.6	4.2	2.79
Bentonite & Dolomitic Limestone	0	76.1	57.9	19.6	14.5	1.9	3.5	3.08
	1	150.3	56.7	22.3	13.8	2.5	2.7	2.84
	2	127.8	54.1	22.1	15.3	3.0	3.0	2.68
	4	149.8	52.1	23.5	16.0	3.7	2.0	2.95
	8	125.1	53.8	19.3	18.2	3.2	2.4	3.26
	12	108.1	52.7	20.4	17.6	3.2	2.4	2.76
	24	93.8	53.9	20.1	15.9	2.8	3.6	2.78
Bentonite & $KHCO_3$	0	77.2	57.9	17.6	15.7	2.1	3.9	3.92
	1	147.1	56.9	21.3	13.4	2.5	3.5	3.11
	2	160.0	55.7	21.4	14.1	2.7	3.5	3.10
	4	135.3	53.6	21.5	15.9	3.2	2.7	3.26
	8	142.4	52.1	23.0	17.4	2.6	2.5	3.12
	12	111.9	54.3	22.0	15.3	2.9	2.4	3.12
	24	79.9	54.6	21.4	12.6	3.2	4.5	2.82

[a]Acetic Acid.

[b]Propionic Acid.

[c]Butyric Acid.

[d]Valeric Acid.

[e]Isovaleric Acid.

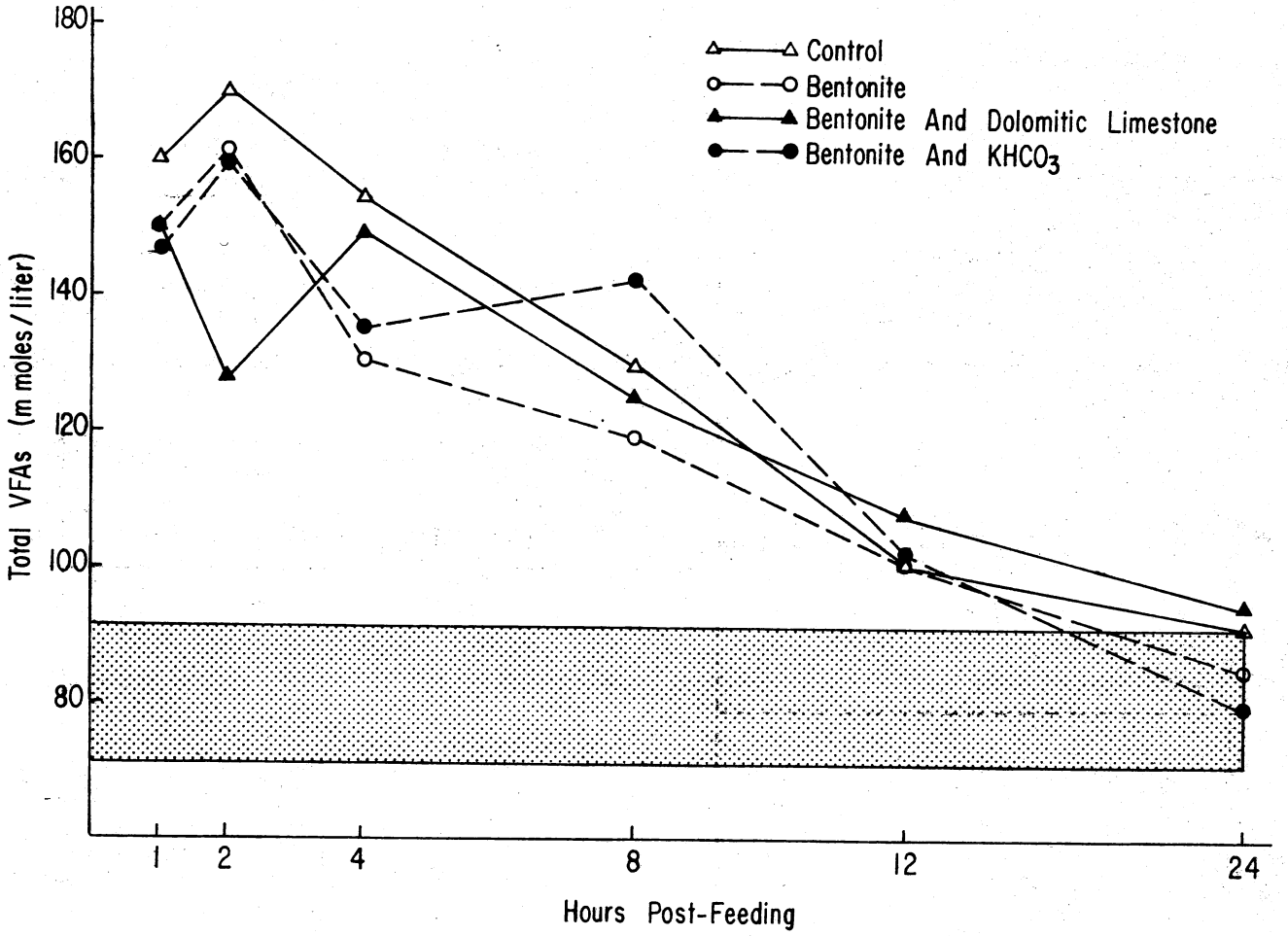

Figure 4. Time-Course Changes in Total Ruminal Volatile Fatty Acid Concentrations; Shaded Represents the Mean ± SEM Concentration of All Steers at Time Zero.

level of total VFAs associated with the control, sodium bentonite and the sodium bentonite plus $KHCO_3$ diets were observed at 2 hours post-feeding. The sodium bentonite plus dolomite diet fed steers displayed a peak at 1 hour post-feeding followed by a decrease at 2 hours and a very similar peak at hour 4. After 2 hours post-feeding the concentration of total VFAs in steers fed either the control or the sodium bentonite diets continuously declined to the last sampling time with the level in the control steers being higher at all times. This decline also occurred 4 hours after feeding in steers consuming the sodium bentonite plus dolomite rations, although the level associated with this ration was higher than the control at 12 and 24 hours. As with ruminal glucose and lactic acid those steers eating the ration containing the sodium bentonite and $KHCO_3$ displayed a decreased VFA concentration at 4 hours after feeding followed by an increased level at 8 hours. By the last sampling time the level associated with this ration was the lowest and well within the normal range of zero time.

Although time-course changes for individual acids are not shown, all steers displayed changes in acetic, propionic, and butyric acids very similar to those of the total VFA concentration. The molar percentages of the individual acids are reported in Table III. The ratio of acetic acid to propionic acid at time zero was lowest for steers fed the control ration (2.96) and the highest for steers fed the sodium bentonite plus $KHCO_3$ diet (3.92) (Table III). This could perhaps be an indication that the addition of these buffers to a diet would reduce an animal's efficiency of weight gain.

The effectiveness of dietary buffers in preventing excessive ruminal responses to the over consumption of a high-moisture corn diet

fluctuated tremendously at the different sampling times. The concentrations of ruminal glucose, lactic acid, and total volatile fatty acids, and the osmolality of rumen contents were maximum at either 1 or 2 hours post-feeding. The ruminal pH indicated that the most severe period of ruminal lactic acidosis was from 4 to 8 hours after feeding. For the period from 1 to 8 hours after feeding, the only consistently effective diet appeared to be that containing sodium bentonite and $KHCO_3$. The steers consuming this diet had the highest ruminal pH throughout the sampling period. The level of lactic acid associated with the bentonite plus $KHCO_3$ ration was the lowest at 1 hour and second lowest from hours 2 to 8, while the glucose concentration was next to the lowest at 1 hour and lowest of all rations until 12 hours after feeding. The steers fed the diet containing sodium bentonite and $KHCO_3$ also had the lowest or next to the lowest osmolalities, and acetic acid, propionic acid, and total VFA concentrations until 8 hours post-feeding. By the 8 hour sampling time the ruminal osmolality associated with all diets had essentially returned to the normal tonicity range of all steers at time zero. The increases of acetate, propionate, and total VFA concentrations at 8 hours post-feeding in steers fed the sodium bentonite plus $KHCO_3$ ration seems to indicate that the ruminal microbial population was still active and had not suffered as much damage as the microbial populations of steers fed the other rations. In most ruminal parameters the steers consuming the diet containing bentonite and $KHCO_3$ had returned to a more normal state by the last sampling time than steers consuming the other 3 rations. Although sodium bentonite fed steers consistently had the second highest ruminal pH and the lowest total VFA

concentrations during the critical 4 to 8 hour post-feeding period, the varied response in other parameters by steers fed the sodium bentonite and the sodium bentonite plus dolomitic limestone diets precludes positive statements about their effectiveness as dietary buffers in preventing or reducing ruminal lactic acidosis.

The steers fed the high-moisture corn diets in this study never experienced a blood pH of less than 7.37. Initially the blood pHs of steers fed the "buffered" rations were higher than those of steers fed the control diet (Table IV). After consuming an excessive amount of a high-moisture corn diet, the blood pH of the control steers increased to a peak at 8 hours post-feeding, then returned to normal by the last sampling time. Steers consuming the sodium bentonite plus dolomitic limestone diet also had an increase in blood pH at 2 hours after feeding, but returned to normal at the 4 and 8 hour sampling times with a second pH increase at 12 hours post-feeding. Those steers consuming the bentonite and the bentonite plus $KHCO_3$ diets displayed small decreases in blood pH. Due to the increase in blood pH of the control steers and the decrease in the pH of the bentonite plus $KHCO_3$ fed steers the changes from time zero of these diets at 4 hours after feeding were significantly different ($P < 0.05$). At 8 hours post-feeding the continued pH increase of the control steers resulted in a change from time zero which was significantly different from those steers fed the bentonite plus dolomite diet ($P < 0.05$). Because the blood pHs indicate that none of the steers were ever in danger of severe systemic lactic acidosis, a most interesting observation is that at the last sampling time the pH in steers fed the bentonite diet decreased below the zero time pH. This change was significantly different from the pH

TABLE IV

EFFECT OF LACTIC ACIDOSIS ON BLOOD PARAMETERS

Parameter	Time Post-Feeding	Ration			
		Control	Bentonite	Bentonite & Dolomitic Limestone	Bentonite & $KHCO_3$
	(hr.)				
pH	0	7.37	7.39	7.39	7.40
	2	7.37[a]	7.38[a,b]	7.40[a,b]	7.40[b]
	4	7.38[a]	7.39[a,b]	7.39[b]	7.38[a,b]
	8	7.39[a]	7.40[a,b]	7.41[a]	7.41[a,d]
	12	7.38[a]	7.39[b,c]	7.40[a]	7.41[a,d]
	24	7.37[a]	7.37[b,c]	7.40[a]	7.41[a,d]
			(mm Hg)		
pCO_2	0	43.5	44.6	44.0	42.4
	2	41.0	40.3	40.3	39.2
	4	42.0	40.9	39.9	40.9
	8	42.1	39.1	39.1	37.7
	12	39.6	39.3	39.0	36.5
	24	41.4	41.4	41.5	41.8
			(mEq/liter)		
HCO_3	0	24.2	26.4	26.3	25.3
	2	23.0[a]	23.1[b]	24.2[b]	23.1[a,b]
	4	24.0[a,c]	23.8[b,d]	22.9[b,d]	23.7[b]
	8	24.3[a,c]	23.4[b,d]	22.8[b,d]	23.2[b]
	12	22.7[a]	22.9[b,c]	23.9[a,b,c,d]	22.2[a,d]
	24	23.1[a]	23.2[b,c]	25.1[a,b,c,d]	25.9[a,d]

[a,b]Means in the same row with different superscripts differ in change from 0-hour value ($P < 0.05$).

[c,d]($P < 0.01$).

changes of steers fed the control and the bentonite plus dolomite diets ($P < 0.05$) and the bentonite plus $KHCO_3$ ration ($P < 0.01$); all of which returned to the zero time level or increased. The blood pH depression in sodium bentonite fed steers at this time could result in a delayed willingness to resume eating.

Because the bicarbonate buffering system of the body can be regulated by the respiratory and renal systems (Guyton, 1971) the quantity of bicarbonate in the blood is an indication of an animal's attempt to cope with systemic lactic acidosis. Work by Bhattacharya and Warner (1968) indicated that dietary buffers can influence to a small degree the concentration of blood bicarbonate. Steers consuming the sodium bentonite and the sodium bentonite plus dolomitic limestone diets had at least 2.1 mEq per liter more bicarbonate at time zero than the control steers (Table IV). For those steers consuming the control, bentonite, and the bentonite plus $KHCO_3$ diets the changes in blood bicarbonate concentrations indicated that the most severe systemic acidosis occurred at 2 and 12 hours post-feeding (Figure 5); but the bentonite plus dolomitic limestone fed steers were under the greatest stress from 4 to 8 hours after feeding. At 4 and 8 hours post-feeding the level of bicarbonate in the control steers was very similar to that observed at time zero. However, the concentration of blood bicarbonate ssociated with the other rations had decreases so that the change from time zero of the control steers was significantly less than that of steers consuming the bentonite and the bentonite plus dolomite diets at 4 ($P < 0.05$) and 8 hours post-feeding ($P < 0.01$). The bentonite plus $KHCO_3$ fed steers displayed a change at 8 hours which was significantly greater than that of the control steers ($P < 0.05$).

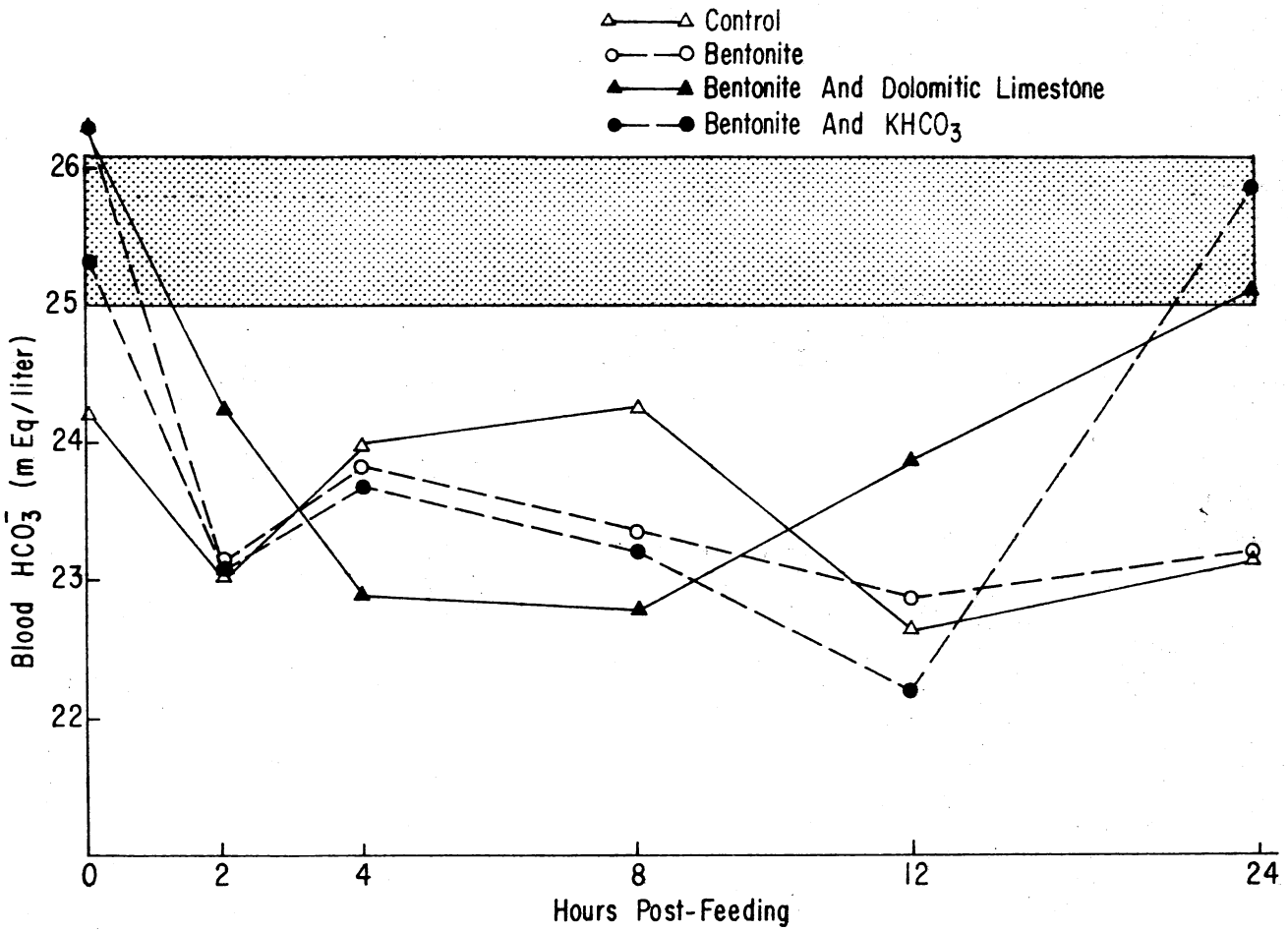

Figure 5. Time-Course Changes in Blood HCO_3 Concentrations; Shaded Area Represents the Mean $\pm$ SEM Concentration of All Steers at Time Zero.

The most important observation from the blood bicarbonate concentrations may be that at 24 hours post-feeding. The concentration of bicarbonate in the blood of steers fed the diet containing bentonite and $KHCO_3$ increased above that at time zero by 0.6 mEq per liter and was significantly different ($P < 0.01$) from the bicarbonate concentration of steers consuming the sodium bentonite diet which had a 3.2 mEq per liter decrease from the pre-feeding value. Steers fed the control and the bentonite plus dolomitic limestone diets also had decreased from their pre-feeding bicarbonate concentrations by 1.1 and 1.2 mEq per liter, respectively. However, the decrease was significantly greater for the steers fed the sodium bentonite ration than for those consuming the bentonite plus dolomite ($P < 0.10$) and the control diets ($P < 0.05$). Although the differences are not noted in Table IV for clarity, steers fed the diet containing sodium bentonite and dolomitic limestone also had a change from time zero which was significantly different ($P < 0.10$) from the change in bicarbonate concentrations of steers consuming the bentonite and $KHCO_3$ diet. Of most interest is that the blood bicarbonate concentrations from 12 to 24 hours post-feeding increased dramatically in steers fed the diets containing either bentonite and dolomite or bentonite and $KHCO_3$ while the control and bentonite fed steers displayed little change. Thus, 24 hours after consuming an excessive amount of high-moisture corn, the blood bicarbonate data indicate that steers fed the sodium bentonite plus dolomitic limestone or the bentonite plus $KHCO_3$ diets had returned to a more favorable acid-base status and that these steers would possibly have eaten more readily upon refeeding than steers consuming the control and the sodium bentonite supplemented rations.

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APPENDIX

TABLE V

EFFECT OF LACTIC ACIDOSIS ON RUMINAL PARAMETERS

Parameter	Time Post-Feeding	Ration			
		Control	Bentonite	Bentonite & Dolomitic Limestone	Bentonite & $KHCO_3$
	(hr.)				
pH	0	6.5	6.5	6.5	6.6
	1	5.8	5.8	5.7	6.1
	2	5.4	5.5	5.5	5.7
	4	5.1	5.2	5.1	5.4
	8	5.1	5.2	5.1	5.5
	12	5.6	5.7	5.6	5.9
	24	6.3	6.4	6.3	6.4
				(mmoles/liter)	
Lactic Acid	0	$28.8_{a,b}$	$28.1_{a,b}$	28.5_{a}	25.9_{b}
	1	92.6^{a}	$92.8^{a,b}$	99.1^{a}	66.6^{b}
	2	87.6^{a}	$78.2^{a,b}$	57.2^{b}	$69.5^{a,b}$
	4	47.8	27.2	54.2	38.4
	8	64.0	61.6	49.0	58.2
	12	39.0	45.7	42.2	48.0
	24	40.0	44.9	41.0	33.3
				(mg/100 ml)	
Glucose	0	36.7	25.0	36.9	8.8
	1	159.6	72.8	146.7	100.0
	2	126.7	72.0	110.7	56.8
	4	99.2	37.6	48.1	35.1
	8	84.6	77.7	55.2	53.2
	12	48.2	34.9	51.9	46.8
	24	39.2	16.2	24.3	29.4

^{a,b}Means in the same row with different superscripts differ in change from 0-hour value ($P < 0.05$).

VITA

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