3-1986

Effects of excess potassium by bolus and pasture fertilization on mineral metabolism in lactating beef cows

Francis John Mueller

To the Graduate Council:

I am submitting herewith a thesis written by Francis John Mueller entitled "Effects of excess potassium by bolus and pasture fertilization on mineral metabolism in lactating beef cows." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

M.C. Bell, Major Professor

We have read this thesis and recommend its acceptance:

R.N. Heitmann, J.K. Miller, S.P. Oliver

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EFFECTS OF EXCESS POTASSIUM BY BOLUS AND PASTURE FERTILIZATION ON MINERAL METABOLISM IN LACTATING BEEF COWS

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Francis John Mueller

March 1986

ACKNOWLEDGMENTS

The author wishes to express his graditude and appreciation to the following:

To the Animal Science Department at The University of Tennessee, Knoxville, for the opportunity to pursue the Master's degree.

To Dr. M. C. Bell for his advice and guidance in conducting the research and preparation of this manuscript.

To Dr. R. N. Heitmann, Dr. J. K. Miller and Dr. S. P. Oliver for serving as committee members, and their advice, suggestions and criticism of this manuscript.

To Jim Bacon and Nancy Ramsey for their assistance in collection and chemical analysis of samples.

To John Burnett and Jim Tweed for their assistance in care and handling of experimental animals.

To fellow graduate students for their assistance in sample collection, as well as their friendship and advice.

To my family for their support and encouragement throughout the author's collegiate studies.

Finally to his wife Kristen for her love and support throughout the author's studies.

ABSTRACT

The objectives of this research were to compare potassium (K) fertilization and oral administration of KCl as sources of excess K and to determine the effects of K sources on plasma, milk, fecal and urine minerals. Four pasture trials two weeks in length, using beef cows with suckling calves were conducted in February, March and April of 1984 and 1985. Cows were placed on one of two adjacent tall fescue pastures. Both pastures were fertilized with 39 kg/ha of nitrogen (N). One pasture received no fertilization of K and the other 112 kg/ha. One half the cows on each pasture received an oral dose of KCl on days 0, 2, 4, 7, 9, and 11 of each trial. Plasma, milk, fecal and urine samples were collected over a 160 minute period on the days cows were dosed.

The KCl dose on days 0 and 2 of trial 1 consisted of 280g of KCl administered by stomach tube. On day 4 the KCl dose was changed to 280g of KCl given via gelatin capsule, due to the death of one cow on day 2. The death of the cow appeared to be the result of K toxicity. On day 4 two cows were treated with 30% magnesium chloride enema for K toxicity. As a result of this toxicity the KCl dose was changed to 220g given by gelatin capsule, where it remained throughout the rest of the study. The levels of K given during trial 1 were below the levels of K listed in the literature as necessary to cause K toxicity.

Cows grazing the K fertilized pasture had depressed plasma and urine concentrations of magnesium (Mg) and calcium (Ca), indicating a reduction in absorption of Mg and Ca. Plasma sodium (Na) and K and urine K concentrations were not affected by pasture fertilization. Effects of pasture fertilization on milk minerals were inconsistent. Cows recieving KCl had reduced plasma Mg and increased plasma K and Na

concentrations. Effects of KCl treatment on plasma Ca were variable. During the trials conducted in 1985, KCl dosed cows had reduced concentrations of Mg and Ca in their urine. Administration of KCl resulted in an increase in urine K concentration. KCl dosed animals had elevated concentration of milk Mg. Milk K and Ca concentrations tended to be higher for KCl bolused animals than for their control counterparts. Effects of KCl bolusing on milk Na were inconsistent. These results suggest that dietary K may be more toxic when administered to cattle grazing early spring pasture. It also appears that oral administration of K and K fertilization interfere with the metabolism of Mg and that the incidence of grass tetany may be increased by both.

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CHAPTER I

INTRODUCTION

Sjollema (1932) was the first to associate a metabolic disorder that caused tetanic convulsions and death in livestock with a depression in plasma magnesium (hypomagnesemia). This disorder is commonly known as "grass tetany" but is also referred to as grass staggers, wheat pasture poisoning, lactation tetany and hypomagnesemic tetany (Fontenot et al., 1973). Grass tetany is primarily a problem in mature lactating ruminants grazing lush, rapidly growing spring pastures (Grunes et al., 1970). The symptoms of grass tetany include excitability, muscle incoordination, muscular tremors, excessive salivation, grinding of teeth, tetanic contractions, labored breathing, racing heart beat, coma and death (Sjollema, 1932; Sims and Crookshank, 1956; Aikawa, 1971).

In many parts of the world, grass tetany is one of the major metabolic disorders of cattle (Grunes et al., 1970). Fontenot (1979) reported that hypomagnesemia is not a malady of mismanaged cattle, but is a problem of farms with superior management. It has been estimated that grass tetany causes the death of 1-2% of all mature cows annually (Kemp, 1958 ; Murdock et al., 1975). Greene et al. (1985) reported that death due to grass tetany accounted for 23.2% of all cows leaving a herd of beef cows during a four year period. Although not fully documented, production losses resulting from subclinical hypomagnesemia may far out weigh the losses due to clinical grass tetany (Bell, 1983).

Extensive research involving hypomagnesemia has been conducted, but there still remain many questions related to the origin, etiology and occurrence of the disorder. Potassium has been shown to adversely

affect the metabolism of Mg. Several researchers have reported that the occurrence of grass tetany is increased when animals graze pastures with high K content (Nicholson and Shearer, 1938; Banet, 1983; Hodge and Bell, 1983). The addition of K to the diet of livestock has also been shown to affect Mg metabolism. However results of these studies have been variable. The objectives of this experiment were:

(1) To compare K pasture fertilization and KCl boluses as sources of excess K for lactating beef cows.

(2) To determine the effects of K sources on plasma, milk, fecal and urine minerals.

CHAPTER II

REVIEW OF LITERATURE

I. PHYSIOLOGY OF MAGNESIUM METABOLISM

Physiological Function

Magnesium is the fourth most abundant cation in the body. It is widely distributed among the tissues of the body, but like K is primarily an intracellular ion (Care, 1967). Magnesium has been known to be essential for living cells for many years, but the homeostatic mechanisms by which its distribution within the body are controlled remain obscure.

The animal body contains about 0.05% Mg by weight (Schofield and Morrel, 1960). Approximately 60% of the Mg in the body is found in the skeleton, 40% in soft tissue and only 1% in the extracellular fluids (Rook and Storry, 1962). Care (1967) reported that Mg in bone exists in the hydration shell of hydroxyapatite.

Magnesium ions are necessary for the action of many enzyme systems, particularly those involved in carbohydrate metabolism (Durlach and Rayssiguier, 1983). All enzymes which are required for transferring phosphate groups from adenosine triphosphate (ATP) are activated by Mg (Wacker and Vallee, 1964). Since ATP is required for a wide range of body functions such as muscle contraction, glucose metabolism, protein, fat and enzyme synthesis, the biochemical role of Mg extends to all these functions. Magnesium has also been found to play a major role in nerve transmission and conduction (Wilson, 1964). Walker and Duffus

(1983) reported that Mg may be involved the proliferation of tumor cells.

Plasma and Serum

Rook and Storry (1962) reported the normal range for serum Mg to be 1.2 to 3.8 mg/dl. In a survey conducted in the British Isles over a two year period involving 200,000 cows, the mean serum Mg concentration was found to be 2.1 mg/dl (Ross and Halliday, 1975). The normal lower limit in cattle is believed to be 1.7 mg/dl, animals with values below this are considered hypomagnesemic (Rook and Storry, 1962). In a study where blood samples were taken from 12 cows with clinical hypomagnesemia the mean value of plasma Mg was 0.79 mg/dl (Hall and Reynolds, 1972). Rogers (1979) stated that in clinical cases of grass tetany serum Mg levels are usually below 0.7 mg/dl, however Rook and Storry (1962) stated that serum Mg level of 0.5 mg/dl or less are not always associated with clinical signs of tetany. Forbes (1972) reported average serum Mg concentrations of 0.9 and 1.8 mg/dl for tetanic and normal cows respectively.

A depression in plasma concentration of calcium (Ca) has been associated with hypomagnesemia. Forbes (1972) found that 88% of tetanic animals had some degree of hypocalcemia. Parr (1957) placed the normal range for serum Ca in cattle between 8.5 and 11.5 mg/dl with a mean value of 10 mg/dl. In cases of clinical hypomagnesemia, however plasma Ca generally falls rapidly below 8.0 mg/dl (Rogers, 1979).

Plasma Mg concentrations have been correlated to Mg intake and urinary excretion of Mg. A high correlation has been shown between a drop in urine Mg and the subsequent decline in plasma Mg (Rook and

Balch, 1958; Storry and Rook, 1963; House and Mayland, 1976b; Fisher et al., 1978). Kemp et al. (1961) reported that when plasma Mg concentration dropped below 1.8 mg/dl, urinary excretion became negligible. This is in agreement with Rook and Storry (1962) who stated that the renal threshold for Mg is near 1.7 mg/dl. Intake of Mg has also been correlated to plasma Mg concentration (Kemp et al., 1961; O'Kelly and Fontenot, 1969). Several studies have shown that feeding supplemental Mg increases plasma Mg levels. (Hansard et al., 1975; Boling et al., 1979; McMurray et al., 1981).

Hypomagnesemia is more common in late winter and early spring, when cattle are switched from winter rations to rapidly growing spring pasture. Ramsey et al. (1979) reported a rapid drop in plasma Mg levels of cows switched from winter rations to spring pasture in Tennessee. Hansard et al. (1975) reported that February, when plasma Mg was 29% lower than the experimental average, appeared to be the critical month for grass tetany in Tennessee.

Cerebrospinal Fluid

The average Mg content of the cerebrospinal fluid (CSF) is approximately 2.26 mg/dl in cattle (Aikawa, 1981). A postive correlation has been found between severe hypomagnesemia and lower CSF levels of Mg (Meyer and Scholz, 1972; Pauli and Allsop, 1974; Reynolds et al., 1984). Meyer (1977) reported that when CSF levels fell below 1.6 mg/dl animals were at risk of tetany, and at 1.2 mg/dl tetany occurred on a regular basis. It has been reported that the clinical signs of tetany are more related to a drop in CSF Mg levels than to a fall in plasma Mg concentration (Meyer and Scholtz, 1972). Researchers

have attempted to prove this finding experimentally. Tetany has been produced by infusing synthetic CSF low in Mg content into the lateral ventricles (Allsop and Pauli, 1975). Depressed CSF Mg levels have been raised to normal by intravenous injection of magnesium chloride in sheep (Meyer and Busse, 1975a) and rectal infusion of a 30% $MgCl_2 \cdot 6H_2O$ solution in calves (Reynolds et al., 1984).

Absorption

A knowlege of Mg absorption from the gut, mechanism of absorption and factors effecting Mg availabilty is needed to better understand the etiology of hypomagnesemia. Several factors including binding of Mg to digesta and mineral interrelationships have been reported to influence Mg availabilty. Early work reported that absorption of Mg occurred in the small intestine with little absorption from the forestomach (Care and Van't Klooster, 1965; Phillipson and Storry, 1965). More recent research demonstrated that the forestomach is the primary site of Mg absorption, with the small intestine being more involved with a net excretion of Mg back into the gastrointestinal tract (Rogers and Van't Klooster, 1969; Grace et al., 1974; Ben-Ghedlia et al., 1975). Tomas and Potter (1976b) infused Mg into the abomasum and omasum and found that 100% of the the dose was recovered in the duodenum. These researchers also infused Mg into the rumen and recovered 36-61% of the dose in the duodenum. These findings indicated that absorption of Mg takes place in the forestomach. Field and Munro (1977) infused Mg into the rumen and duodenum and found that urinary excretion of Mg was greatest after ruminal infusion, indicating that the forestomach was the primary site of Mg absorption. Tomas and Potter (1976a) reported total

absorption for Mg of 44.4% when Mg was ruminally infused and 3.75% for postruminal infusion. In this study, the reticulorumen accounted for 70 to 90% of the total Mg absorption. In contrast, Stewart and Moodie (1956) found the principle site of Mg absorption to be the duodenum and small intestine. These researchers dosed sheep with 122g of magnesium sulfate then collected venous blood from different sites of the alimentary tract to determine Mg absorption. However, during the study, sheep were anesthetized which may account for the difference in their findings. Greene et al. (1983b) reported that the primary site of Mg absorption is the preintestinal region with a net excretion into the small intestine of steers. They also reported that preintestinal absorption was decreased 39% when the diet of steers contained 4.8% K.

It has been shown that the large intestine can absorb Mg but to a much lesser extent than the forestomach (Rogers and Van't Klooster, 1969; Grace et al., 1974; Tomas and Potter, 1976a). Rectal infusions of $MgCl_2 \cdot 6H_2O$ have been shown to rapidly increase plasma Mg in cattle and sheep (Meyer and Busse, 1975b; Bell et al., 1977).

Magnesium absorption appears to occur through an active process (Brown et al., 1978; Field and Munro, 1977). Martens et al. (1978) using ^{28}Mg found Mg absorption to be dependent upon the Na-K transport system of the mucosa. Using ouabain an inhibitor of Na-K ATPase, they observed a 90% decrease in Mg absorption. Martens (1983) reported that Mg transport across the rumen wall approached saturation at a concentration of 12.5 mmol/l of Mg in the rumen. The Michaelis-Menten (Km) constant was 11.4 mmole/l and the maximal efflux (Vmax) was 120.3 umole/min. These values for Km and Vmax support the theory that Mg absorption occurs through an active process.

Shockey et al. (1984) showed that Mg absorption is related to the ratio of surface area of ruminal contents to unit weight of ruminal contents. In this study the maximium apparent absorption of Mg for sheep (42%) was 1.75 times that of dairy cows (24%). They also calculated the ratio of digesta surface area : digesta weight to be 1.72 times greater for sheep as compared with dairy cows. These finding compare very well and indicate that Mg absorption may be related to this ratio.

Fecal Excretion

Feces is the primary route of Mg excretion from the body. Shockey et al. (1984) reported that the average fecal excretion of Mg was 76% of the daily intake. These findings agree with those of Kemp et al. (1961) who determined fecal excretion to be 67-93% of the total Mg intake. The daily fecal excretion of Mg for calves 2 to 5 weeks of age has been calculated to be 0.5 mg/kg of body weight (Smith, 1959a). As calves grew older, fecal excretion of Mg increased to 2.2 mg/kg of body weight for 26 to 36 week old calves. Direct estimates of fecal Mg excretion for mature cows fed low Mg diets ranged from 2.2 to 4.0 mg/kg of body weight (Blaxter and Rook, 1954). Simesen et al. (1962) calculated endogenous excretion of Mg to be 1.5 mg/kg of body weight in mature cows.

Rook and Storry (1962) stated that saliva and gastric juices are the two main contributers of endogenous Mg. These researchers determined the Mg content of saliva to be 0.5 to 1.4 mEq/liter and gastric juices to be 0.7 to 2.5 mEq/liter in sheep. Since the contribution of Mg in saliva is relatively important, diets containing

high portions of roughage or fresh grass may be associated with high endogenous fecal Mg loss, thus reducing Mg availability.

The relationship between Mg intake and fecal excretion has been shown to be linear (Field, 1962; Reid et al., 1979; Shockey et al., 1984). Allsop and Rook (1972) speculated that absorption of Mg is regulated by plasma Mg concentrations. They theorized that as plasma Mg increased, absorption decreased and fecal excretion increased. Data to support this was obtained by Frye et al. (1975), who fed sheep diets with Mg levels ranging from 0.5 to 4.1 g/day and found that as Mg intake rose, plasma and fecal Mg levels increased.

Urinary Excretion

Excess absorbed Mg is excreted primarily in the urine. The amount of Mg excreted in the urine is relatively small compared to that excreted in the feces. Urinary excretion of Mg accounts for approximately 5% of the total Mg excreted (Blaxter and Rook, 1954). Jesse et al. (1981) injected 3.0g of Mg intravenously into non-lactating cows and almost 100% was recovered in the urine. Several researchers have found a linear relationship between excess dietary Mg and urinary excretion (Kemp et al., 1961; Field, 1962). It has been suggested that urinary Mg excretion may reflect the nutritional adequacy of the diet (Rook and Storry, 1962). Kemp et al. (1961) showed that urinary Mg levels decreased before plasma Mg concentrations in animals consuming a Mg deficient diet, suggesting that urinary Mg may be a better measure of Mg status than plasma Mg.

The renal threshold for Mg has been estimated to be near 1.7 mg/dl (Rook and Storry, 1962; Meyer, 1976). The control mechanism for renal

excretion of Mg remains obscure. Parathyroid hormone (PTH) is thought to play some role in the excretion of Mg. Littledike and Cox (1979) found that PTH increases tubular reabsorption of Mg. Deetz et al. (1982) showed that older animals had a lower net tubular reabsorption of Mg than younger animals and suggested that this decrease was due to decreased responsiveness to PTH. If these findings are correct, they may help explain why older animals are more susceptible to hypomagnesemia.

Milk Secretion

Magnesium content of cows milk is generally constant regardless of feed or Mg intake. The average Mg content of cows milk is 12 mg/dl (Rook and Storry, 1962) ranging from 7 to 18 mg/dl (Kemp et al., 1961; Grunes et al., 1970; Oluokun and Bell, 1982). Oluokun and Bell (1982) reported that approximately 83% of the Mg in milk is in the bound form. The Mg content of colostrum is high initially, but decreases within a few hours of calving (Rook and Storry, 1962). Small changes in the Mg content of milk have been observed with different types of diets, however, the content remains relatively constant within diet (Rook and Storry, 1962; Oluokun and Bell, 1982). The secretion of Mg in milk contributes to the decrease in the body stores of Mg and is a predisposing factor for hypomagnesemia.

Requirements

O'Kelly and Fontenot (1969) calculated the dietary Mg requirement to maintain plasma Mg of lactating beef cows at 2.0 mg/dl to be 20.9, 22.1 and 18.0 grams per day at 35, 95 and 155 days of lactation, respectively. They also determined that to meet these requirements

forages would need to contain 0.18, 0.19 and 0.16% available Mg on a dry matter basis. Kemp (1960) reported that 2.5 g of Mg plus 0.12 g Mg for each kilogram of milk produced are required by lactating cows.

II. FACTORS AFFECTING MAGNESIUM UTILIZATION

Mineral Interrelationships

Several minerals have been shown to affect the utilization of Mg. Chicco et al. (1973a) fed wethers varing levels of Ca, P and Mg and found that high dietary levels of Ca decreased Mg concentrations in bone and plasma. High dietary levels of Mg reduced plasma and urinary Ca levels, indicating that Mg and Ca are antagonistic. Halse (1984) reported that hypocalcemic cows had an increase in renal conservation of Mg, indicating that Mg and Ca excretion may be controlled by the same mechanism.

Potassium has been shown to have the largest effect of all minerals on Mg metabolism. Several researchers have reported that the occurrence of grass tetany increased when animals grazed pastures with high K content (Nicholson and Shearer, 1938; Banet, 1983; Hodge and Bell, 1983). Butler (1963) stated that tetany prone pastures are generally low in Mg and Na and high in K content. Reduced forage Mg reduces the intake of Mg by grazing ruminants and is especially a problem when the utilization of Mg is affected by other factors. A reduction in forage Mg content can be caused by a number of agronomic factors, such as soil type, species of grass and temperature (Kubota et al., 1980). Fertilization of pastures with K has been shown to increase the forage K content (Smyth et al., 1958; Hodge and Bell, 1983). Hodge and Bell (1983) fertilized two adjacent tall fescue pastures with 0 and 112 kg of

K/ha. The forage content of K was found to be 2.3 and 3.0% for the control and K fertilized pastures respectively. Adams (1975) reported that rapidly growing herbage may contain as much as 4.8% K. Potassium fertilization has also been shown to decrease the forage Mg and Ca content (Blaxter et al., 1960; Kemp, 1960). Lidgate (1976) reported that the depression in forage Mg was greatest after K fertilization in the spring. Cattle grazing K fertilized pastures have been reported to have depressed plasma Mg concentrations (Kemp, 1960; Banet, 1983; Hodge and Bell, 1983). In contrast, some studies have reported that K fertilization had no effect on plasma Mg (Smyth et al., 1958; Hemingway et al., 1963; Ritchie and Hemingway, 1963). Bartlett et al. (1957) reported that K fertilization had no effect on plasma Mg levels of cattle grazing pastures largely composed of clover. However, they did report that plasma Mg levels were lower for cattle grazing pastures containing smaller amounts of clover.

Addition of K to the diet of livestock has also had varying effects on Mg metabolism. The addition of K to the diet has been reported to reduce plasma Mg, increase fecal Mg excretion and decrease urinary excretion of Mg (Kunkel et al., 1953; Suttle and Field, 1967; Newton et al., 1972). Pearson et al. (1949) added 5% K to the diet of mature ewes and found no effect on Mg utilization. Banet (1983) bolused lactating beef cows with 140 g of KCl twice a day and found it to have no effect on Mg utilization 12 hours after treatment. Field and Suttle (1979) fed monozygotic twin cows varing rations high in K and low in Mg. These researchers reported that the effects of K intake on plasma concentrations and urinary excretion of Mg were dependent on Mg intake, whereas the effect on fecal excretion and retention of Mg was not. The

increased fecal Mg excretion seen with high K diets was due to a decrease in Mg absorption rather than to an increase in endogenous excretion of Mg. Greene et al. (1983a) fed sheep four different levels of K and reported that increasing dietary K resulted in linear decreases in Mg absorption and plasma Mg levels. These authors reported that the depression in Mg absorption occurred anterior to the small intestine.

Heavy fertilization of pastures with nitrogen (N) has also been shown to interfere with the utilization of Mg (O'Kelly and Fontenot, 1969; Henry et al., 1977). Bartlett et al. (1957) reported that heavy N fertilization of pastures decreased plasma Mg of cattle grazing those pastures. O'Kelly and Fontenot (1969) and Kemp (1960) have reported similar findings. Stillings et al. (1976) reported that heavy N fertilization decreased urinary and increased fecal excretion of Mg. Nitrogen fertilization has been shown to increase the protein content of forage (Kemp, 1960). This leads to a high protein to carbohydrate ratio which has been implicated in the etiology of grass tetany (Metson et al., 1966). The application of both N and K fertilizer seems to have an additive effect on depressing Mg utilization (Smyth et al., 1958; Fontenot et al., 1960).

Kemp and t'Hart (1957) reported a significant correlation between the incidence of grass tetany and the ratio of K/(Ca+Mg) concentration in milliequivalents in the forage. The incidence of grass tetany was greatly increased when the ratio was greater than 2.2 mEq. Butler (1963) found a similar relationship. However it has been reported that the incidence of grass tetany is more closely related to grasses having less than 0.2% Mg than grasses having K/(Ca+Mg) ratios of 2.2 mEq or more (Kubota et al., 1980).

Recently, it has been suggested that excess aluminium (Al) plays a role in the development of grass tetany. Allen and Robinson (1980) reported that the rumen contents from animals that died from grass tetany contained 2373 ppm Al, while samples from non-tetanic animals contained 405 ppm Al. In this study, the effects of excess Al on the solubility of Mg and Ca were also studied. After a 48 hour incubation of ryegrass in rumen fluid, it was found that excess Al decreased the solubility of Ca and Mg 74% and 56%, respectively. Allen et al. (1984) reported that the addition of Al-citrate to the diet of cattle depressed plasma Mg concentrations. Similiar effects have been reported for Al-sulfate (Kappel et al., 1984). However, some researchers have found Al intake to be unrelated to plasma Mg concentrations (Cherney et al., 1983; Kappel et al., 1983).

Age

Rook and Storry (1962) reported that young animals can mobilize 30% of their skeletal Mg reserves under conditions of Mg deprivation, while in adult animals only 2% of bone Mg can be mobilized. Chicco et al. (1973b) found that when sheep were fed a diet low in Mg, older sheep reduced voluntary intake sooner and suffered from a more rapid decrease in plasma Mg than younger sheep. In a study, conducted over five consecutive years using the same sheep each year, animals had higher plasma and urinary Mg values in the first year (Field, 1962). Smith (1959a) reported that absorption of Mg decreased as calves grew older. It has also been reported that bone concentrations of Mg decrease as calves grow older (Smith, 1959b). The rate of bone depletion is also much more rapid than bone repletion (McAleese et al., 1961; Rook and

Storry, 1962) which may help to explain why older animals are more susceptible to hypomagnesemia.

Climatic Factors

A relationship between grass tetany and climatic conditions has been noted by many researchers. Grass tetany often occurrs on cold wet mornings when animals are on a rapidly growing pasture (Allcroft, 1954). Kemp and t'Hart (1957) correlated the incidence of grass tetany with a rise in temperature and suggested that the incidence of grass tetany increases approximately 5 days after the temperature rises above 14 °C. They postulated that the rise in temperature caused a change in forage composition causing the drop in plasma levels. Sykes et al. (1969) exposed shorn sheep to two levels of acute cold exposure -20 °C and 8 °C. They found that exposure to the 8 °C reduced plasma Mg concentrations by 12%. Exposure to the -20 °C resulted in a drop in both plasma Mg and Ca. Two weeks after the exposure plasma Mg and Ca had not returned to normal.

Fasting has also been associated with decreased serum Mg levels and short term fasts may serve as a trigger for hypomagnesemia (Herd, 1966). Terashima et al. (1982) used a 2x2 factorial arrangement to study the effects of cold exposure and fasting on sheep. Plasma Mg levels fell approximately 1 hour after cold exposure in fasted sheep but cold exposure had no effect on plasma Mg in fed animals. They suggested that cold exposure and fasting act synergistically to trigger hypomagnesemia.

Energy

Rapidly growing spring forage is often low in readily available carbohydrates and high in N which are two conditions related to reduced

utilization of Mg in ruminants (Wilcox and Hoff, 1974). House and Mayland (1976b) fed rations with varied ratios of N to sucrose and found that rations with higher ratios had lower apparent digestibilities of Mg. Reduced levels of carbohydrates favor an increase in the levels of ammonium ions and a higher pH in the rumen, which enhances the formation of magnesium ammonium phosphate an insoluble complex (Van Soest, 1982). Giduck and Fontenot (1984) reported that the addition of glucose, sucrose, lactose or starch to orchardgrass hay increased the apparent absorption of Mg in sheep. Ruminal infusion of glucose increased apparent Mg absorption, but infusion of hydrochloric acid or a mixture of buffered and unbuffered propionate and butyrate did not affect absorption. These findings indicate that rumen pH does not have an effect on the absorption of Mg.

Supplemental carbohydrates can be useful in maintaining plasma Mg levels (Miller et al., 1980). The addition of 2.3 kg of corn/head/day helped reduce the drop in plasma Mg seen after cows calve in the fall (Boling et al., 1979). Addition of glucose to hay diets had no effect on plasma Mg levels, but Mg absorption increased linearly with increasing glucose supplementation (Madsen et al., 1976). It appears that carbohydrates have an effect on Mg utilization, however more work is needed in this area.

Organic Acids

High concentrations of organic acids have been implicated in the etiology of grass tetany (Burau and Stout, 1965; Bohman et al., 1969; DeGregorio et al., 1981). Early spring tetany prone pastures have high levels of the organic acid, trans-aconitate (Stout et al., 1967). However, oral administration of trans-aconitate has not been show to

cause any adverse side effects (Camp et al., 1968; Kennedy, 1968). Citric acid has also been suggested as a potential component in the etiology of grass tetany (Bohman et al., 1969; Deetz et al., 1982). Citric acid increased plasma clearance and urinary excretion of Mg (DeGregorio et al., 1981; Deetz et al., 1982). House and Van Campen (1971) reported however, that the addition of citrate to the diet had no effect on Mg utilization. Trans-aconitate is converted by rumen bacteria to tricarballyate a compound that is slowly fermented in the rumen (Russel and Van Soest, 1984). The slow rate of fermentation and its potential as a Mg chelator suggest that trans-aconitate could be an important factor in hypomagnesemia. Bohman et al. (1969) induced clinical tetany by giving high levels of KCl (150g/100kg of body weight) with either citric acid (157g/100kg of body weight) or trans-aconitate (157g/100kg of body weight). Clinical tetany occurred only when the KCl was given with one of the two organic acids.

III. PREVENTION AND TREATMENT

Forage Treatment

Currently, grass tetany cannot be totally prevented but its incidence can be reduced to near zero with a combination of agronomic and animal husbandry practices (Bell, 1983). An adequate intake of Mg is required to prevent grass tetany from occurring. Two types of forage treatment have been used to try to accomplish this: fertilization of pastures with Mg to increase the forage Mg content, and foliar application of Mg to increase Mg intake.

Pasture fertilization has been used with some success to increase Mg composition of forage. Bartlett et al. (1957) fertilized forage with

2500 lbs of magnesite/acre and found a 64% increase in forage Mg. It has been shown that fertilization with kieserite ($MgSO_4$) increases the Mg content of hay and the apparent retention of Mg in sheep (Reid et al., 1979). Fertilization with 2240 kg of Mg per hectare increased the Mg content of orchardgrass (Thompson and Reid, 1981). When hay from these fields was fed to sheep, the apparent absorption and retention of Mg increased. When orchardgrass was fed to cattle, their plasma Mg levels rose. Parr and Allcroft (1957) showed that both fertilization with limestone (2.5 tons/acre) and magnesite (100 lbs/acre) increased forage Mg, with a greater response to magnesite fertilization. After fertilization, cattle were allowed to graze these pastures and untreated control pastures. During a 6 year period, no animals showed any signs of clincal grass tetany on fertilized pastures, while on the control pastures tetany did occur. West and Reynolds (1984) fertilized pastures with "Sul-Po-Mg" and epsom salts to supply 181 kg of Mg per hectare. It was found that fertilization had no effect on the Mg content of the pastures. A major disadvantage to Mg fertilization of pastures is that large amounts of fertilizer are needed to increase forage Mg content (Grunes et al., 1970).

An alternative to pasture fertilization is foliar application of Mg to forage as a dust or slurry. Two days after the application of 28 lbs/acre of calcined magnesite dust forage Mg increased from 0.16% to 0.31% (Todd and Morrison, 1964). McConaghy et al. (1963) applied a 20% $MgSO_4$ spray to a pasture and increased forage Mg to 0.7% immediately after application. However, plasma Mg levels of cattle grazing the pasture were only slightly increased. Rogers and Poole (1976) applied 33.6 Kg of calcined magnesite per hectare to spring pasture as a dust

and reported an increase in the plasma Mg level of cattle consuming the pasture.

Slurrys of magnesium-bentonite have been used to increase adhesion of Mg to plants (Wilkinson and Stuedemann, 1979; Reynolds, 1980). Adhesion of Mg to the forage is dependent on the wind and amount of rainfall. For the best results it is recommended that the magnesium compounds be applied to dew wet grass (Bell, 1983). Meyer (1976) recovered 40% of a Mg dust that was applied to dew wet grass. Reynolds (1980) reported that a magnesium-bentonite slurry lasted for up to four weeks when rainfall was not excessive.

Supplements

Several supplements have been used successfully in the prevention of grass tetany. However, it can be a problem to get all animals to consume adequate quantities of the supplement. In a review of grass tetany prevention, Bell (1983) identified four causes of inadequate consumption of supplements: lack of palatability of the supplement; unfamiliarity with the supplement; placement of the supplement in the pasture; and cow variation.

Several researchers have used mineral mixes successfully to prevent grass tetany (Allcroft, 1954; Hansard et al., 1975; Bell et al., 1984). Gerken and Fontenot (1967) compared magnesium oxide (MgO) and dolomitic limestone as Mg supplements for steers. Apparent absorption of Mg and plasma Mg were much higher for the steers feed the MgO. The feeding of dolomitic limestone resulted in decreased digestibility of the energy constituients of the ration (Moore et al., 1971). Magnesium phosphate mixed with concentrates increased plasma Mg levels (Fishwick and

Hemingway, 1973; Ritchie and Fishwick, 1977). Ritter et al. (1984) tried to increase the labile Mg reserves of beef cows by supplementing the winter ration with MgO. The three rations used were a control ration of tall fescue hay with a Mg deficient salt-mineral mix, 6.4 Kg of corn silage dry matter plus 114 g MgO/day and tall fescue hay with free access to a 40% MgO salt-mineral mix. Animals were all turned on to the same tetanigenic pasture at the same time. Plasma Mg values for the three treatments were 1.97, 3.58 and 2.06 mg/dl on the day they were turned onto pasture. However, the winter supplementation provided very little long term protection against hypomagnesemia, indicating cows must have a supplemental source of Mg during this critical period.

One problem with high magnesium mixes is their palatablity. Palatablity can be increased by mixing them with corn, silage or other concentrates. Magnesium molasses blocks have also been used in attempts to increase palatabilty. Bell et al., (1984) compared the effectiveness of a "high Mag block" and a "high Mag mix" in preventing hypomagnesemia. Intakes were 11.9 g of Mg/day for the block and 7 g of Mg/day for the mix. The Mg block proved to be more effective in preventing hypomagnesemia.

Supplementation with readily available carbohydrates may prevent hypomagnesemia (Madsen et al., 1976; Rassiguier and Poncet, 1980; Giduck and Fontenot, 1984). Giduck and Fontenot (1984) reported that apparent absorption of Mg was increased by the addition of glucose, sucrose, lactose or starch to orchardgrass hay. Boling et al. (1979) compared cows supplemented with MgO or energy with control cows recieving no supplement. Supplementation with MgO resulted in an increase in plasma Mg concentrations, but the effects of energy supplementation were

variable. McLaren et al., (1975) also reported variable effects from energy supplementation. Thompson et al. (1984) and Hodge et al. (1984) reported finding no effect from energy supplementation.

Addition of Mg to the water supply has met with some success in preventing hypomagnesemia. Rogers and Poole (1976) increased plasma Mg levels by adding magnesium acetate to the water supply. A disadvantage to this is that during periods of rainfall, animals may not need to drink from the supplemented water supply (Wilkinson and Stuedemann, 1979). Oral drenching of animals with Mg compounds has met with limited success but does not appear to be effective or practical (Bell, 1983).

Slow release magnesium bullets have been used in the prevention of hypomagnesemia. Magnesium bullets are composed of a Mg alloy containing 2% Cu, 12% Al and 86% Mg designed to release Mg slowly over long periods. The bullets weigh approximately 100g and are weighted with lead (Davey, 1968). Bullets are administered orally and Mg release occurrs in the rumen (Stuedemann et al., 1984). Some researchers have reported that bullets are effective in preventing hypomagnesemia (Davey, 1968; Smyth, 1969), while others have reported them to be ineffective (Foote et al., 1969; Kemp and Todd, 1970). Stuedemann et al. (1984) reported that the release of Mg from bullets was too slow to provide adequate protection. A wide variation in the decompostion rate of bullets between animals may lead to an inconsistent release of Mg to protect animals (House and Mayland, 1976a).

Treatment

Animals developing grass tetany should recieve medical treatment immediately. Intravenous infusion of a calcium magnesium dextrose

solution will usually give almost immediate results. The quantity to be given depends on the severity of the case. After treatment, animals should be fed a diet adequate to meet their daily Mg and energy requirements (Boling, 1982). Grunes et al. (1970) indicated that after treatment, if animals were not removed from the tetany producing pasture their Mg levels can again become dangerously low within 24 to 37 hours.

Meyer and Busse (1975b) used a rectal infusion of a 30% magnesium chloride solution to raise plasma Mg levels. They reported that plasma Mg levels of normal and hypomagnesemic animals increased drastically after infusion. Bell et al. (1977) used $MgCl_2$ enemas to treat a cow with clinical grass tetany. Intially, the cows plasma Mg concentration was 0.65 mg/dl, twenty minutes after treatment plasma Mg had increased to 2.22 mg/dl. The effect of the enema treatment lasted approximately 24 hours for hypomagnesemic animals and 8 hours for normomagnesemic animals. Reynolds et al. (1984) reported that an enema of $MgCl_2$ was effective in raising plasma Mg levels of both normal and hypomagnesemic calves 5 minutes after administration. In hypomagnesemic calves, CSF Mg levels were elevated 30 minutes after the enema was given.

CHAPTER III

EXPERIMENTAL PROCEDURE

I. GENERAL DESIGN

In February, March and April of 1984 and 1985 two pasture studies were conducted. Each of the studies were composed of two trials two weeks in length. Trials 1 and 2 were conducted in 1984, and trials 3 and 4 were conducted in 1985. Animals ranged in age from three to fourteen years, with an average age of seven years and they averaged 486 kg. All cows had calves at their side ranging in age from one to seven weeks at the start of each trial. Cows were maintained on fescue pasture, supplemented with hay prior to the start of the studies. All cows had access to a Mg deficient salt-mineral mix ad libitum. All cows were bled via jugular puncture begining at the end of January and every three weeks after until the start of the study. Plasma Mg concentrations of these samples were used to allocate cows to treatments.

Twelve lactating Angus and Angus x Hereford cows were alloted to a two by two factoral arrangement in each trial with no cows being used more than once in the same year. Cows were assigned to four groups so that average age, plasma Mg concentration, weight and calf age were similar for each group. Groups were then randomly alloted to treatments.

II. TREATMENTS

Treatments consisted of a control pasture and a high K fertilized pasture. The control pasture was fertilized with 39 kg of N/ha. The high K pasture was fertilized at the same rate plus 112 kg of K/ha. Fertilizer was applied to the tall fescue pastures in the fall preceding each trial. Six animals were placed on each pasture with a stocking rate of 1.25 cows/ha at the start of each trial. These six cows were then subdivided into KCl treatments. KCl treatments consisted of 3 cows recieving an oral dose of KCl with the three remaining cows recieving no KCl.

At the start of trial 1, KCl treatments were 0 or 280 grams of KCl/day administered as a liquid drench beginning on day 0. However, during the course of this trial, KCl dosing level was changed twice. On day 2 of this trial, approximately six minutes after dosing, one cow from the control pasture died after exhibiting tetany like symptoms. As a result of this the dose was changed to 280 grams of KCl administered via gelatin capsule on day 4. On day 4, approximately 20 minutes after dosing, two cows from the K fertilized pasture developed tetany like symptoms. These cows recovered after adminstration of 60g of $MgCl_2 \cdot 6H_2O$ by rectal infusion. On day 7 the dose was again changed to 220g of KCl administered via gelatin capsule on days 7, 9 and 11. During trials 2, 3 and 4, the KCl dose remained at 220g given via gelatin capsule on days 0, 2, 4, 7, 9 and 11.

III. SAMPLE COLLECTION

Cows were stanchioned at approximately 0800 hours on days 0, 2, 4, 7, 9 and 11 of each trial for collection of data. Cows and calves were separated throughout the 3 hour sampling period. Prior to dosing with KCl pre-dose samples were taken from all cows. Plasma samples were obtained via jugular puncture. Fecal samples were obtained by rectal palpation to promote defecation. If necessary, urination was induced by palpating the cow between the udder and vulva. Milk samples were collected into 15 ml plastic bags by hand milking. After collection of the pre-dose sample the KCl dose was administered to half the cows from each pasture group.

Trials 1 and 2

Plasma, urine, fecal and milk samples were collected during trials 1 and 2. On days 0 and 2 the KCl dose was administered as liquid via stomach tube. Plasma samples were collected from all cows at 0, 20, 40, 80 and 160 minutes after KCl administration. Milk, urine and fecal samples were obtained from all KCl dosed cows at the same time. Milk, urine and fecal samples were collected from all non-dosed cows at 0 and 160 minutes after dosing and when time permitted in between. On day 3 of trial 1, a 10 minute sample was added, due to the death of the cow on day 2. The sampling procedure for dosed and non-dosed cows remained the same during the remainder of trial 1 and all of trial 2. Grab forage samples were collected on days 2 and 8 of both trials while walking diagonally across the pastures.

Blood samples were centrifuged and plasma removed for analysis of Mg, Ca and K. The remaining plasma was then frozen at -4 °C for later

analysis. Urine, fecal and milk samples were frozen at -4 °C for later mineral analysis.

Trials 3 and 4

During trials 3 and 4, blood, urine and milk samples were obtained from all dosed and non-dosed cows at 0, 10, 20, 40, 80 and 160 minutes after KCl dosing. Forage samples were collected on days 0, 3, 7 and 10 of trials 3 and 4. After analysis of data from trials 1 and 2 it was determined that little information could be gained from fecal data. As a result feces was not collected in trials 3 and 4. Samples were collected and stored in the same manner as described for trials 1 and 2.

IV. CHEMICAL ANALYSIS

Plasma

Samples were analyzed for Ca, Mg, K, and Na using an Instrumentation Laboratory Model 551 atomic absorption emission spectrophotometer. All samples were diluted to a known volume for analysis. To prevent interference of phosphate, lanthanum trioxide (.1%) was added to all Ca and Mg dilutions and cesium chloride (.1%) was added to all K dilutions.

Milk and Urine

Duplicate 0.5 ml samples were pipetted into 15 ml furnace tubes, dried overnight in a 100 °C oven and ashed at 550 °C for approximately three hours. Ashed samples were dissolved by adding 1ml of 6N HCl, diluted to a known volume and analyzed like the plasma. Urine samples were analyzed for Ca, Mg and K. Milk samples were also analyzed for Ca, Mg and K as well as Na.

Feces

Fecal samples were removed from the freezer and while still frozen duplicate core samples weighing approximately 2 g were taken from each sample using a drill. Samples were transferred to pre-weighed 15 ml furnace tubes, dried, reweighed to deterimine dry matter (Association of Analytical Chemists (A.O.A.C), 1975), and ashed in the same manner as milk and urine samples. Ashed samples were put into solution using 2 ml of 6N HCl and analyzed for Ca, Mg and K using the same procedure as for plasma.

Forage

Forage dry matter was determined using standard A.O.A.C. (1975) procedures. Dried forage samples were ground through a 1 mm screen in a Wiley mill. Approximately 0.5 g of the ground forages were ashed at 550 °C for three hours. Ashed samples were disolved in 1 ml of 6 N HCl, diluted to a known volume and analyzed for total Ca, Mg, and K content.

V. STATISTICAL ANALYSIS

Data were analyzed statistically using the general linear models procedure prepared by the SAS institute (1982). A preliminary analysis was performed separately for each of the different KCl treatments used in trial 1, to determine if the data could be pooled. The following model was used:

$$Y_{ij} = U + P_i + K_j + PK_{ij} + e_{ij}$$

where:

Y_{ij} = dependent variables

U = theoretical population mean

P_i = effect of pasture, $i = 1\text{-}2$

K_j = effect of KCl treatment, $j = 1\text{-}2$

PK_{ij} = pasture treatment interaction, $i = 1\text{-}2, j = 1\text{-}2$

e_{ij} = random error

Mean square errors were then compared and found to be similar, so data were pooled.

For trials 1 and 2, due to the lack of fecal, urine and milk samples for times 10, 20, 40 and 80 minutes after dosing, data from these times were dropped. A preliminary analysis was then conducted on all the data. The model used was:

$$Y_{ijklmn} = U + Y_i + T_j + YT_{ij} + P_k + K_l + PK_{kl} + D_m + PD_{km} + M_n + KM_{ln} + DM_{mn} + e_{ijklmn}$$

where:

Y_{ijklmn} = dependent variables

U = theoretical population mean

Y_i = effect of year, $i = 1\text{-}2$

T_j = effect of trial, $j = 1\text{-}2$

YT_{ij} = year trial interaction, $i = 1\text{-}2, j = 1\text{-}2$

P_k = effect of pasture, $k = 1\text{-}2$

K_l = effect of KCl treatment, $l = 1\text{-}2$

PK_{kl} = pasture KCl treatment interaction, $k = 1\text{-}2$, $l = 1\text{-}2$

D_m = effect of day, $m = 1\text{-}6$

PD_{km} = pastue day interaction, $k = 1\text{-}2$, $m = 1\text{-}6$

M_n = effect of time, $n = 1\text{-}6$

KM_{ln} = KCl treatment time interaction, $l = 1\text{-}2$, $n = 1\text{-}6$

DM_{mn} = day time interaction, $m = 1\text{-}6$, $n = 1\text{-}6$

e_{ijklmn} = random error

Year and trial were found to be significant so a separate analysis of variance was done for each trial. The model used was:

$$Y_{ijkl} = U + P_i + K_j + PK_{ij} + D_k + PD_{ik} + M_l + KM_{jl} + DM_{kl} + e_{ijkl}$$

where:

Y_{ijkl} = dependent variables

U = theoretical population mean

P_i = effect of pasture, $i = 1\text{-}2$

K_j = effect of KCl treatment, $j = 1\text{-}2$

PK_{ij} = pasture KCl treatment interaction, $i = 1\text{-}2$, $j = 1\text{-}2$

D_k = effect of day, $k = 1\text{-}6$

PD_{ik} = pasture day interaction, $i = 1\text{-}2$, $k = 1\text{-}6$

M_l = effect of time, $l = 1\text{-}6$

KM_{jl} = KCl treatment time interaction, $j = 1\text{-}2$, $l = 1\text{-}6$

DM_{kl} = day time interaction, $k = 1\text{-}6$, $l = 1\text{-}6$

e_{ijkl} = random error

If significant differences were found by this procedure, the Student Neuman-Keuls' test (Sokal and Rohlf, 1981) was used to separate means.

CHAPTER IV

RESULTS AND DISCUSSION

I. EFFECTS OF PASTURE FERTILIZATION

Forage Mineral Composition

Forage mineral composition during the four pasture trials is presented in Table 1. The K fertilized pasture tended to have lower total Mg concentrations (Table 1). However, the diffences between the two pasture treatments were only significant in trial 4. Forage Ca concentrations tended to be lower for the K fertilized pasture (Table 1). Differences were only statistically significant during trial 3. In trials 3 and 4, the pasture fertilization with K resulted in higher ($P < .05$) total forage K concentrations. Forage K concentrations tended to be higher for the K fertilized pasture during trials 1 and 2, however differences were not statistically significant. Results similar to these have been reported by Banet (1983) and Hodge and Bell (1983).

Forage concentrations of Mg, Ca, and K were lower for trial 1, than for the three remaining trials. During trial 1, ambient temperatures decreased during the second week. The colder temperatures resulted in slower forage growth during this period. This is a condition which is known to decrease forage mineral composition, by decreasing nutrient uptake by the forage (Kubota et al., 1980).

Plasma Data

Plasma Mg concentrations were an average of 7.5 % lower ($P < .05$) for cows grazing the K fertilizied pasture (Table 2). Similar results have been reported by other researchers (Banet, 1983; Hodge and Bell,

TABLE 1. Total forage mineral composition (mg/g DM basis).

Pasture	Trial 1	Trial 2	Trial 3	Trial 4
			Mg	
Control	1.77	2.72	1.93	2.38[a]
+K	1.64	2.70	1.75	1.85[b]
			Ca	
Control	3.15	4.84	4.63[a]	4.71
+K	2.78	4.74	3.44[b]	3.08
			K	
Control	10.35	34.14	21.78[a]	23.85[a]
+K	12.20	36.89	25.40[b]	27.29[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 2. Effect of pasture on plasma minerals (mg/dl).

Pasture	Trial 1	Trial 2	Trial 3	Trial 4
		Mg		
Control	1.64[a]	2.23[a]	1.86[a]	1.85[a]
+K	1.62[b]	1.91[b]	1.77[b]	1.67[b]
		Ca		
Control	8.96[a]	9.73	9.26[a]	9.35[a]
+K	8.69[b]	9.68	8.92[b]	8.87[b]
		K		
Control	12.80[a]	14.70	13.24	13.19
+K	14.22[b]	14.81	13.05	13.40
		Na		
Control	329[a]	373	297	319[a]
+K	336[b]	374	297	314[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

1983). Plasma Mg concentrations fell sharply on day 7, 9, and 11 of trial 1, for animals grazing both the control and K fertilized pastures (Table 3). During the second week of of the first trial, there was a large drop in ambient temperatures. On the morning of day 7 the temperature fell to -11 °C. On days 7 and 9 the pastures were covered with a light dusting of snow. As a result there was a reduction in forage growth, limiting the amount of forage available to the cows. Cold exposure and fasting have both been associated with a drop in plasma Mg concentrations (Herd, 1966; Sykes et al., 1969). Terashima et al., (1982) suggested that cold exposure and fasting may act synergistically to trigger hypomagnesemia. This may have caused the drop in plasma Mg concentrations during trial 1.

Presented in Table 2 are the effects of pasture fertilization on plasma Ca concentrations. In trials 1, 3 and 4, plasma Ca was lower ($P < .05$) for animals consuming the K fertilized pasture. Plasma Ca levels were also lower for cattle consuming the K fertilized pasture during trial 2, but differences were not significant. Changes in plasma Ca over sampling days were variable but plasma Ca concentrations of cows grazing the K fertilized pasture were lower ($P < .05$) on most sampling days (Table 4).

Potassium fertilization appeared to have little effect on the plasma K concentrations of grazing cattle, except in trial 1, where plasma K was higher ($P < .05$) for cattle grazing the K fertilized pasture (Table 2). Changes in plasma K concentrations over sampling days for animals consuming the control and K fertilized pastures were varible (Table 5).

TABLE 3. Effect of pasture treatment on plasma Mg (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	a 1.83	bf 2.03	a 1.89	cf 1.52	c 1.49	d 1.09
+K	a 1.89	ag 1.87	a 1.92	bg 1.64	c 1.45	d 1.03
	Trial 2					
Control	a 2.00	bf 1.71	cf 2.20	df 2.56	def 2.49	ef 2.42
+K	a 1.94	bg 1.30	ag 1.96	cg 2.22	acg 2.15	ag 1.92
	Trial 3					
Control	a 1.68	a 1.66	bcf 1.97	cf 1.88	c 1.89	b 2.06
+K	ac 1.80	b 1.52	cg 1.74	acg 1.77	ac 1.86	a 1.96
	Trial 4					
Control	a 1.50	bf 1.42	c 2.06	df 1.74	ef 2.17	ef 2.20
+K	a 1.41	bg 1.20	c 1.94	dg 1.58	cg 2.02	cg 1.86

a,b,c,d,e Means within rows in each trial with different superscripts are different ($P < .05$).

f,g Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 4. Effect of pasture treatment on plasma Ca (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	ae 8.33	b 9.04	be 9.07	b 9.00	ce 8.74	de 9.54
+K	af 8.01	bd 8.83	cdf 8.70	bd 8.92	cf 8.48	bf 9.13
	Trial 2					
Control	ae 9.23	ae 8.97	a 9.13	b 10.50	bce 10.34	c 10.20
+K	af 8.88	bf 8.58	a 9.02	c 10.63	cf 10.56	d 10.35
	Trial 3					
Control	abe 9.63	abe 9.59	b 9.45	ce 8.34	d 8.73	ae 9.80
+K	abf 9.36	bf 9.16	a 9.44	cf 7.73	d 8.57	abf 9.22
	Trial 4					
Control	ae 9.26	ae 9.36	b 8.86	ae 9.49	ae 9.31	ce 9.81
+K	af 8.47	abf 9.04	a 8.59	bf 9.27	abf 8.82	abf 9.02

a,b,c,d Means within rows in each trial with different superscripts are different ($P < .05$).

e,f Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 5. Effect of pasture treatment on plasma K (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
	ac	a	ace	be	ce	abce
Control	13.06	13.98	13.79	12.21	11.34	12.47
	ad	bc	cf	bcf	af	bdf
+K	13.20	14.89	15.69	14.95	12.44	14.15
	Trial 2					
	a	ace	ac	be	ce	ac
Control	14.85	14.31	14.69	16.56	13.77	14.21
		f		f	f	
+K	14.08	15.49	14.18	15.50	14.65	14.94
	Trial 3					
	ae	be	bd	c	d	be
Control	14.84	14.02	13.07	10.37	12.40	13.54
	af	bf	c	d	c	af
+K	13.75	15.64	12.54	10.66	12.58	14.23
	Trial 4					
	a	bc	ce	bce	be	be
Control	14.72	12.61	12.23	12.93	13.20	13.46
	a	bcd	bdf	cf	bf	cdf
+K	15.92	12.74	13.54	11.79	13.89	12.55

a,b,c,d
Means within rows in each trial with different superscripts are different ($P < .05$).

e,f
Means within columns in each trial with different superscripts are different ($P < .05$).

The effects of pasture fertilization on plasma Na concentrations were inconsistent (Table 2). Plasma Na concentrations during trials 2 and 3 were not influenced by K fertilization but cows grazing the K fertilized pasture had higher ($P < .05$) plasma Na concentrations during trial 1. However, during trial four, cattle consuming the K fertilized pasture had lower ($P < .05$) plasma Na concentrations. Plasma Na concentrations followed similar patterns over sampling days for cows grazing the control and K fertilized pastures (Table 6).

Urinary Mineral Excretion

Urinary Mg concentrations of cows grazing the K fertilized pasture, in comparison with their control counterparts, tended to be lower in trials 1 and 2 and were significantly lower ($P < .05$) in trials 3 and 4 (Table 7). Banet (1983) and Hodge and Bell (1983) reported similiar findings. Urinary Mg concentrations were lower ($P < .05$) on most sampling days for the cattle consuming the K fertilized pasture (Table 8). Changes in urinary Mg concentrations over sampling days were inconsistent for both pasture groups during trials 1 and 2, but patterns were similiar for cows grazing the control and K fertilizied pastures during trials 3 and 4.

Urine Ca concentrations were lower ($P < .05$) for the cows grazing the K fertilized pasture during trials 2, 3 and 4 (Table 7), which is in agreement with Banet (1983). Changes in urinary Ca concentrations over sampling days were variable (Table 9).

Pasture fertilization appeared to have little effect on urinary K concentrations (Table 7). Urinary K concentrations did not differ significantly between the two groups during trials 1, 2 and 3 but were

TABLE 6. Effect of pasture treatment on plasma Na (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	a 315	a 319	bg 367	b 357	cg 287	a 314
+K	a 313	b 322	ch 381	d 366	ah 309	b 319
	Trial 2					
Control	a 374	bg 340	c 385	a 378	c 388	ag 372
+K	a 374	bh 354	c 385	c 381	c 386	dh 364
	Trial 3					
Control	a 287	ag 292	bg 277	a 288	cg 311	dg 330
+K	a 289	ah 281	ah 272	a 299	ah 306	bh 335
	Trial 4					
Control	a 331	b 367	c 269	d 274	eg 318	fg 327
+K	a 338	b 366	c 268	c 272	dh 330	ah 342

a,b,c,d,e,f Means within rows in each trial with different superscripts are different ($P < .05$).

g,h Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 7. Effect of pasture on urine minerals (mg/dl).

Pasture	Trial 1	2	3	4
	Mg			
Control	4.87	6.16	9.84[a]	5.68[a]
+K	4.00	4.83	3.95[b]	2.71[b]
	Ca			
Control	5.67	7.53[a]	6.08[a]	6.80[a]
+K	10.19	4.58[b]	3.52[b]	3.41[b]
	K			
Control	1402	1460	986	672[a]
+K	1353	1591	946	608[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 8. Effect of pasture treatment on urinary Mg (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	---------- Trial 1 ----------					
Control	3.51	4.55	6.28	7.50	4.13	3.02
	ab	ab	b	ab	a	ab
+K	4.23	3.07	2.05	5.32	7.02	2.82
	---------- Trial 2 ----------					
	ab	ab	be	be	b	ae
Control	8.36	3.07	7.93	2.64	3.02	11.49
	a	b	bf	abf	b	bf
+K	9.96	1.55	4.38	6.20	4.28	2.38
	---------- Trial 3 ----------					
	a	abe	be	ce	de	ce
Control	7.86	5.67	3.71	11.00	16.71	13.37
	a	bcf	cf	bf	af	bf
+K	7.14	1.71	1.17	3.44	7.43	3.32
	---------- Trial 4 ----------					
	ae	ae	be	de	bce	ce
Control	1.44	3.07	5.04	9.95	6.57	7.98
	af	af	af	bf	af	cf
+K	0.56	0.74	1.97	6.44	2.05	4.47

a,b,c,d
Means within rows in each trial with different superscripts are different ($P < .05$).

e,f
Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 9. Effect of pasture treatment on urinary Ca (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	1.84[a]	2.28[a]	3.67[a]	17.40[b]	5.34[a]	3.92[a]
+K	2.16[a]	3.01[a]	2.66[a]	16.06[b]	6.72[a]	5.10[a]
	Trial 2					
Control	4.06[a]	8.17[abd]	5.23[a]	7.72[ab]	7.35[ab]	11.40[bd]
+K	3.55[e]	3.06[e]	3.48[e]	6.76[e]	4.85[e]	5.89[e]
	Trial 3					
Control	5.04[a]	7.95[ab]	2.14[c]	5.24[ad]	8.80[bd]	7.76[abd]
+K	6.97[a]	2.73[c]	1.52[c]	2.56[ce]	4.71[be]	2.80[ce]
	Trial 4					
Control	1.52[a]	4.78[bd]	6.05[bd]	6.63[cd]	10.82[bd]	11.06[cd]
+K	0.78[a]	1.77[abe]	3.15[be]	5.05[ce]	3.24[be]	6.38[ce]

a,b,c Means within rows in each trial with different superscripts are different ($P < .05$).

d,e Means within columns in each trial with different superscripts are different ($P < .05$).

lower ($P < .05$) for cows grazing the K fertilized pasture during trial 4. Changes in urinary K concentrations over sampling days were inconsistent (Table 10).

Milk Mineral Secretion

Effects of K fertilization on milk minerals are shown in Table 11. Relative concentrations of minerals in milk of cows grazing the two pastures were so inconsistent between trials that effects of pasture fertilization cannot be established. For example, no differences were found between pasture fertilization groups for Mg in trials 1 and 2 or for K or Na in trial 2. In contrast, Mg concentrations in the milk of cows grazing K fertilized pasture were lower ($P < .05$) in trial 3 but higher ($P < .05$) in trial 4. Concentrations of Ca, K and Na were lower in milk of cows on the K fertilized pasture in trial 1 but higher in trials 3 and 4. The effects of K fertilization on milk Mg, Ca, K and Na concentrations in the milk over sampling days were also variable and inconsistent (Tables 12, 13, 14 and 15).

II. EFFECTS OF KCl DOSING

KCl Treatment Trial 1

On the second sampling day of trial 1, one cow died approximately 6 minutes after the administration of 280g of KCl by stomach tube. Prior to death, muscle incoordination, muscle tremors, excessive salivation and labored breathing were observed followed by falling and death. These symptoms were very similar to those described in the literature for field cases of grass tetany (Sjollema, 1932; Sims and Crookshank, 1956; Aikawa, 1971; Bell et al., 1977). Blood samples were obtained prior to the administration of KCl and immediately after death. Plasma

TABLE 10. Effect of pasture treatment on urinary K (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	ab 1287	b 430	ab 947	a 1854	ab 1062	c 2981
+K	ac 1376	b 544	ac 1165	a 1521	c 1081	d 2551
	Trial 2					
Control	a 1157	a 1107	a 1130	a 1049	a 1119	b 2934
+K	a 1087	a 1474	a 1191	a 1166	a 1000	b 3532
	Trial 3					
Control	ae 1524	b 745	be 649	ce 1272	b 832	b 806
+K	af 1195	b 803	cf 526	df 1478	b 958	c 649
	Trial 4					
Control	a 1209	bc 534	ce 412	c 486	c 410	be 644
+K	a 1135	b 436	bf 608	b 488	b 508	cf 872

a,b,c,d Means within rows in each trial with different superscripts are different ($P < .05$).

e,f Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 11. Effect of pasture on milk minerals (mg/dl).

Pasture	Trial 1	2	3	4
			Mg	
Control	12.49	9.53	8.38[a]	7.98[a]
+K	12.26	9.70	7.12[b]	8.59[b]
			Ca	
Control	130.4[a]	112.6[a]	98.6[a]	99.5[a]
+K	118.6[b]	108.7[b]	101.1[b]	102.9[b]
			K	
Control	110.9[a]	112.3	84.6[a]	86.8[a]
+K	100.2[b]	115.6	93.7[b]	101.5[b]
			Na	
Control	49.02[a]	92.49	75.20[a]	69.96[a]
+K	57.48[b]	80.85	48.62[b]	65.08[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 12. Effect of pasture treatment on milk Mg (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
			Trial 1			
Control	9.12	8.43	21.45	12.05	9.61	10.23
+K	9.12[a]	8.78[a]	10.10[a]	10.53[a]	8.83[a]	24.76[b]
			Trial 2			
Control	10.03	9.30	9.28	9.31	9.40	9.80
+K	9.89	9.03	10.03	10.00	9.78	9.32
			Trial 3			
Control	9.84[a]	9.55[aef]	6.16[bf]	6.63[cf]	9.20[def]	9.01[df]
+K	9.14[a]	8.21[bg]	4.29[cg]	5.73[dg]	7.38[eg]	7.95[bg]
			Trial 4			
Control	8.86[af]	8.57[a]	8.63[af]	8.27[a]	6.40[bf]	7.11[c]
+K	9.09[abg]	8.78[ab]	9.18[abg]	10.34[a]	6.91[bg]	7.20[b]

[a,b,c,d,e] Means within rows in each trial with different superscripts are different ($P < .05$).

[f,g] Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 13. Effect of pasture treatment on milk Ca (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	ab 115.8	a 97.8	b 160.7	ab 145.3	ab 121.4	abe 126.2
+K	a 112.4	b 100.0	ac 127.0	c 127.0	ac 122.6	acf 115.9
	Trial 2					
Control	118.9	112.2	110.7	110.3	112.8	110.4
+K	ab 110.9	b 102.3	ab 105.9	ab 107.3	a 116.7	ab 107.5
	Trial 3					
Control	ae 107.8	a 112.4	be 77.0	ce 97.6	c 100.9	ce 98.0
+K	af 115.3	a 114.3	bf 64.7	cf 103.0	c 99.5	df 110.0
	Trial 4					
Control	a 101.2	a 102.0	be 107.7	a 102.1	ce 89.9	c 93.8
+K	a 103.1	a 104.3	bf 115.9	b 103.1	bf 100.9	c 89.9

a,b,c,d
Means within rows in each trial with different superscripts are different ($P < .05$).

e,f
Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 14. Effect of pasture treatment on milk K (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	a 119.6	abf 108.7	abf 111.3	b 100.2	b 100.4	a 120.9
+K	ac 110.1	bg 84.9	bcg 99.1	bc 99.5	b 88.5	a 121.9
	Trial 2					
Control	af 134.0	a 132.1	b 113.5	c 89.8	cf 93.3	b 107.9
+K	abg 120.7	a 132.1	ab 114.4	b 100.3	abg 116.7	ab 112.9
	Trial 3					
Control	a 98.1	b 79.6	bf 76.3	bf 76.5	af 94.5	bf 83.1
+K	ac 95.9	b 85.8	bg 86.1	bcg 91.3	ag 102.1	ag 101.4
	Trial 4					
Control	af 100.3	bf 110.0	cf 86.8	df 76.3	cf 91.6	ef 52.8
+K	ag 112.3	ag 115.6	ag 111.6	bg 94.7	bg 99.3	cg 74.4

a,b,c,d,e Means within rows in each trial with different superscripts are different ($P < .05$).

f,g Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 15. Effect of pasture treatment on milk Na (mg/dl) over sampling days

Pasture	Day 0	2	4	7	9	11
	Trial 1					
Control	a 35.71	be 81.06	a 47.13	a 49.68	a 45.81	a 39.86
+K	ac 41.14	bf 102.00	a 55.13	ac 52.80	c 38.78	ac 41.41
	Trial 2					
Control	a 51.17	a 54.40	b 115.79	b 115.82	be 113.60	b 109.63
+K	ab 63.00	b 53.67	ab 82.99	ab 94.88	abf 83.75	a 101.67
	Trial 3					
Control	ae 61.77	be 93.38	ce 71.68	ce 73.09	ce 78.11	ce 75.51
+K	af 50.67	bf 58.56	cf 39.16	af 48.82	df 45.30	af 49.22
	Trial 4					
Control	a 48.36	ae 54.55	ae 51.97	ae 75.83	a 44.06	b 149.15
+K	ab 54.72	abf 59.36	af 68.29	af 67.22	b 47.87	c 93.83

a,b,c,d Means within rows in each trial with different superscripts are different ($P < .05$).

e,f Means within columns in each trial with different superscripts are different ($P < .05$).

K rose from 12.97 mg/dl prior to dosing to 35.42 mg/dl immediately after death. Plasma Mg concentrations were also elevated at the time of death, rising from 1.68 to 3.17 mg/dl. Bohman et al. (1969) reported similar findings in cattle given KCl and organic acids to induce tetany. Rook and Storry (1962) indicated that plasma Mg levels tend to increase with the onset of convulsions; as a result analysis for Mg at this time may be of limited value. The National Research Council (NRC) (1980) reported that severe signs of potassium toxicity occurred when plasma K concentrations reached 31.3 mg/dl so the animals death was most likely the result of K toxicity rather than from grass tetany.

After the loss of one animal on day 2 of trial 1, it was decided to administered the KCl in the crystalline form by gelatin capsule, since KCl is less toxic in this form due to the slower rate of K absorption (NRC, 1980).

On day 4 of trial one after the KCl dose was changed to 280g of KCl administered via gelatin capsule, two more animals experienced episodes of tetany. Symptoms which occurred approximately 30 minutes after the KCl was given to both animals, were similar to those described for the first animal. Animals were treated by administration of two enemas of 30% $MgCl_2$ in 200 ml of water. Blood samples were obtained from these animals at prescribed times and immediately after administration of enemas. Plasma K was again elevated at the time of tetany. Plasma K of cow 897 rose from 12.89 mg/dl prior to dosing to 22.89 mg/dl just before tetany occurred. A similar pattern was observed for cow 185 (14.60 to 27.50 mg/dl). At the time tetany occurred, plasma Mg concentrations were in the normal range for both animals. After administration of the $MgCl_2$ enema, plasma Mg was slightly elevated while plasma Ca, K and Na

concentrations remained unchanged. Both cows were on there feet within 10 minutes of enema administration. Involuntary twitching of the muscles in the hindlegs was observed up to 30 minutes after tetany, otherwise animals appeared normal.

The level of K (147g/cow/day) that was given during first part of trial 1 was well below the toxic levels for K found in the literature. Dennis and Harbaugh (1948) administered 340g of K as KCl by stomach tube to five 300 kg Jersey cows. This resulted in the death of one animal, two animals requiring treatment and two cows showing no signs of toxicity. They also reported that no signs of toxicity were seen when animals were given 182 and 240g of potassium. Ward (1966) reported the death of one 475 kg animal less than 10 minutes after administration of 238g of K as the chloride by drenching. Administration of a combination of 157g of KCl with 157g of trans-aconitic acid or citric acid per 100 kg of body weight, administered by stomach tube to 237 kg heifers, resulted in a high incidence of tetany resembling field cases of grass tetany (Bohman et al., 1969). These researchers also reported that plasma Mg concentrations were unaffected by the treatment.

The level of K given by Bohman et al. (1969) was relatively small but was more than double the amount we used when animal size is considered. Bohman et al. (1969) also reported that tetany only occurred when KCl was given in combination with organic acids. Animals were maintained on hay and concentrate diets during the studies by Dennis and Harbaugh (1948) and Bohman et al. (1969). During the current study, cattle were maintained on lush early spring pasture. High concentrations of organic acids in early spring pasture (Stout et al.,

1967) may enhance the toxic effects of K. These data suggest that K may be more toxic to animals consuming early spring pastures.

Plasma Data

Plasma Mg levels were within the normal range reported by Rook and Storry (1962). Plasma Mg concentrations were lower ($P < .05$) for the cows receiving KCl in all four trials (Table 16). Banet (1983) in a similar study reported that bolusing with 280g of KCl had no effect on plasma Mg. However, in his study, KCl was administered in two 140g doses and plasma samples not obtained until 12 hours after administration of the KCl. These differences in experimental protocol may account for the differences observed in the present study. Greene et al. (1983b.) also reported a decrease in plasma Mg concentrations when steers were fed diets high in K. Presented in Table 17 are the effects of KCl dosing on plasma Mg concentrations over time. Plasma Mg concentrations remained unchanged over time for both KCl treated and control cows during trials 2, 3 and 4 but were lower ($P < .05$) at the 10 minute time sample for both groups during trial 1. This may have been a result of the change in sampling procedure that occurred in the first trial. On days 0 and 2 of trial 1, no plasma samples were obtained at 10 minutes after dosing. Plasma Mg concentrations were highest during the first three days of trial 1 and due to the lack of 10 minute samples on days 0 and 2 of trial 1, plasma Mg concentrations at 10 minutes after dosing may appear lower than they actually were. Data from trials 2, 3 and 4, suggest that KCl bolusing had no effect on plasma Mg concentrations during the 160 minute sampling period.

TABLE 16. Effect of KCl treatment on plasma minerals (mg/dl).

Treatment	Trial 1	2	3	4
	---------- Mg ----------			
Control	1.66[a]	2.16[a]	1.85[a]	1.86[a]
KCl	1.60[b]	1.99[b]	1.78[b]	1.66[b]
	----------Ca----------			
Control	8.89[a]	9.74	9.11	8.97[a]
KCl	8.75[b]	9.66	9.06	9.25[b]
	---------- K ----------			
Control	12.40[a]	14.01[a]	11.69[a]	11.98[a]
KCl	14.77[b]	15.49[b]	14.61[b]	14.61[b]
	---------- Na ----------			
Control	330[a]	372[a]	297	315[a]
KCl	335[b]	375[b]	297	318[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 17. Effect of KCl treatment on plasma Mg (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 1					
Control	a 1.70	b 1.55	ab 1.66	ab 1.66	ab 1.66	a 1.69
KCl	a 1.63	b 1.46	a 1.64	a 1.59	a 1.60	a 1.62
	Trial 2					
Control	c 2.17	2.15	2.16	c 2.16	c 2.15	c 2.20
KCl	d 1.97	1.99	2.00	d 1.99	d 1.97	d 1.99
	Trial 3					
Control	1.91	1.86	1.86	1.84	1.82	1.83
KCl	1.78	1.78	1.78	1.78	1.80	1.76
	Trial 4					
Control	c 1.89	c 1.85	c 1.86	c 1.84	c 1.83	c 1.86
KCl	d 1.65	d 1.64	d 1.65	d 1.69	d 1.67	d 1.66

a,b Means within rows for each trial with different superscripts are different ($P < .05$).

c,d Means within columns for each trial with different superscripts are different ($P < .05$).

Effects of KCl bolusing on plasma Ca concentrations were variable (Table 16). Plasma Ca concentrations of cows receiving KCl were lower ($P < .05$) during trial 1, not significantly different from control cows during trials 2 and 3 and higher ($P < .05$) during trial 4. Potassium chloride bolusing did not affect plasma Ca concentrations during the 160 minute sampling period (Table 18).

Effects of KCl dosing on plasma K concentrations are presented in Table 16. As expected, administration of KCl increased ($P< .05$) plasma K concentrations. Plasma K concentrations were 15.9% greater in dosed animals than non-dosed counterparts. There were no differences in plasma K concentrations of dosed and non-dosed cows at the 0 and 10 minute sample times (Table 19). However, plasma K concentrations of the dosed group were higher ($P <.05$) than the non-dosed group for the remainder of the sampling times. Plasma K concentrations of the dosed cows increased ($P < .05$) at the 20 minute sample and remained elevated throughout the remainder of the sampling period whereas plasma K concentrations of the control cows remained relatively constant. These data indicate that 10 to 20 minutes after KCl administration were required for capsules and KCl to dissolve before K absorption occurred.

In trials 1, 2 and 4, plasma Na concentrations were higher ($P < .05$) for KCl dosed cows than for controls (Table 16) but differences were relatively small and may not be physiologically significant. Plasma Na concentrations changed little during the sampling period for control cows but increased at the 80 and 160 minute sampling times for KCl dosed cows (Table 20). The addition of K to the diets of sheep has been shown to increase the absorption of Na through the activation of the Na-K ATPase pump (Scott, 1967). It appears that this may have

TABLE 18. Effect of KCl treatment on plasma Ca (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	---------- Trial 1 ----------					
Control	8.94	8.93	8.82	8.81	8.88	8.95
KCl	8.80	8.92	8.74	8.68	8.69	8.72
	---------- Trial 2 ----------					
Control	9.81	9.65	9.70	9.77	9.74	9.78
KCl	9.63	9.62	9.55	9.70	9.70	9.76
	---------- Trial 3 ----------					
Control	9.21	9.06	9.07	9.05	9.12	9.16
KCl	9.03	9.12	9.08	8.95	9.03	9.17
	---------- Trial 4 ----------					
Control	9.02	8.91	8.96	8.97[a]	9.00[a]	8.95
KCl	9.25	9.19	9.15	9.34[b]	9.40[b]	9.17

[a,b] Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 19. Effect of KCl treatment on plasma K (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
			Trial 1			
Control	12.75 e	12.40 e	12.33 e	12.00 e	12.06 e	12.85 e
KCl	13.23 a	12.56 a	15.45 bf	15.61 bf	15.74 bf	15.29 bf
			Trial 2			
Control	13.86 a	13.75 a	13.52 ae	13.38 ae	14.63 be	14.97 be
KCl	13.93 a	13.95 a	16.01 bf	16.10 bf	16.56 bf	16.38 bf
			Trial 3			
Control	12.58 a	11.50 b	11.27 be	11.45 be	11.48 be	11.87 be
KCl	12.31 a	12.46 a	15.61 bcf	16.29 cf	16.26 cf	14.71 bf
			Trial 4			
Control	12.48 a	12.12 ab	11.71 abe	11.37 be	11.87 abe	12.35 abe
KCl	12.53 a	12.25 a	15.33 bdf	16.57 cf	16.17 cdf	14.82 bf

a,b,c,d Means within rows in each trial with different superscripts are different ($P < .05$).

e,f Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 20. Effect of KCl treatment on plasma Na (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 1					
Control	334	339	328	324	330 d	330 d
KCl	335 ab	341 a	329 b	332 ab	336 abe	340 ae
	Trial 2					
Control	366 a	369 ab	369 ab	376 b	375 b	376 bd
KCl	369 a	373 ac	372 ac	374 ac	379 bc	382 be
	Trial 3					
Control	294	293	292	293 d	294 d	316
KCl	293 a	295 a	294 a	298 ace	301 bce	304 b
	Trial 4					
Control	317	315	315	316	314 d	316 d
KCl	314 a	313 a	315 a	317 a	323 be	328 be

a,b,c Means within rows in each trial with different superscripts are different ($P < .05$).

d,e Means within columns in each trial with different superscripts are different ($P < .05$).

occurred in the present study. However, the KCl did not stimulate an increase in plasma Na concentrations until 80 minutes after adminstration, indicating that had sampling been carried out over a longer period of time differences in the plasma Na concentrations of control and dosed cows may have been greater. This may also explain why KCl administration had no effect on plasma Mg concentrations during the 160 minute sampling period. Magnesium absorption is believed to be controled by the Na-K ATPase pump (Martens et al., 1978). Since it appears from these data that KCl dosing did not affect the Na-K ATPase pump until 80 minutes after dosing, a change in plasma Mg concentrations would not be expected until sometime after this occurred.

Urinary Mineral Excretion

Effects of KCl treatment on urine Mg are presented in Table 21. Urine Mg concentrations of dosed cows tended to be lower than non-dosed cows during trials 1 and 2, however, differences were not statistically significant. During trials 3 and 4, dosed cows had lower ($P < .05$) urine Mg concentrations than did controls. Urinary Mg concentrations tended to increase over time for non-dosed cattle, and decreased slightly for KCl bolused cows (Table 22). Greene et al. (1983b) reported a decrease in the urinary Mg output of steers fed high K diets which they attributed to a decrease in Mg absorption. This may have been what caused the decrease in urinary Mg concentrations during trials 3 and 4. However this cannot be confirmed due to the lack of data on total urine output. A second possible explaination for the lower urinary Mg concentration of KCl dosed animals is that a change in urine volume occurred. During the 160 minute sampling period cows were not

TABLE 21. Effect of KCl treatment on urine minerals (mg/dl).

Treatment	Trial 1	2	3	4
	---------- Mg ----------			
Control	4.58	5.69	8.82[a]	5.64[a]
KCl	4.24	5.18	4.78[b]	2.65[b]
	----------Ca----------			
Control	9.29	5.46	5.17[a]	6.67[a]
KCl	6.65	6.15	4.34[b]	3.42[b]
	---------- K ----------			
Control	1200[a]	1127[a]	790[a]	538[a]
KCl	1569[b]	1842[b]	1141[b]	740[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 22. Effect of KCl treatment on urinary Mg (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	8.10[ad]	6.83[a]	6.65[a]	7.02[ad]	10.53[bd]	13.61[cd]
KCl	5.88[e]	5.71	4.61	3.64[e]	4.31[e]	4.53[e]
	Trial 4					
Control	3.89[a]	3.54[a]	3.10[a]	4.26[ad]	7.93[bd]	10.34[cd]
KCl	3.67[a]	2.86[ab]	2.69[ab]	2.35[abe]	1.79[be]	2.51[abe]

a,b,c Means within rows in each trial with different superscripts are different ($P < .05$).

d,e Means within columns in each trial with different superscripts are different ($P < .05$).

allowed access to water which could cause a decrease in urine volume and an increase in urinary mineral concentrations. Potassium chloride is known to have diuretic effects which may have offset the effects of water deprivation which may account for the slight decrease in urinary Mg concentrations seen following KCl admimistration.

Urinary Ca concentrations followed a pattern similiar to urinary Mg concentrations (Table 21). Urinary Ca remained relatively constant throughout the 160 minute sampling period for the KCl dosed cows (Table 23). Urinary Ca concentrations remained relatively constant through the 40 minute time sample then increased ($P < .05$) at 80 and 160 minute sampling times for control cows. This increase may again have been due to a decrease in urine volume.

As expected, urinary K concentrations were higher ($P < .05$) for the KCl dosed group (Table 21). Urinary K concentrations were also significantly higher ($P < .05$) at most sampling times for the KCl dosed group (Table 24). Urinary K concentrations of the control cows tended to decrease during the first 40 minutes of the sampling period then increased at the 80 and 160 minute sampling times, again indicating a possible reduction in urine volume. During the first 20 minutes following KCl dosing urinary K concentrations of dosed cows fell then increased at the 40, 80 and 160 minute sampling times. This increase in urine K concentrations corresponds well with the increase in plasma Mg concentration seen 20 minutes folowing KCl administration for dosed cows.

TABLE 23. Effect of KCl treatment on urinary Ca (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	4.21^{a}	2.94^{a}	3.10^{a}	3.33^{a}	6.67^{bd}	10.60^{cd}
KCl	4.47	4.69	4.02	3.77	4.01^{e}	4.87^{e}
	Trial 4					
Control	5.83^{a}	3.37^{a}	3.02^{a}	4.48^{ad}	9.97^{bd}	13.03^{cd}
KCl	4.66	2.78	2.80	3.53^{e}	2.50^{e}	4.19^{e}

^{a,b,c} Means within rows in each trial with different superscripts are different ($P < .05$).

^{d,e} Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 24. Effect of KCl treatment on urinary K (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	d 869	d 776	d 718	d 691	d 771	d 916
KCl	abe 1157	be 977	be 944	ae 1033	ae 1268	ce 1492
	Trial 4					
Control	ad 615	ab 474	bd 386	bd 382	ad 527	cd 812
KCl	ae 822	b 636	be 582	be 608	ae 801	ce 991

a,b,c Means within rows in each trial with different superscripts are different ($P < .05$).

d,e Means within columns in each trial with different superscripts are different ($P < .05$).

Milk Mineral Secretion

Mineral concentrations in milk (Table 25) agree well with reported values of 7 to 28 mg/dl for Mg (Rook and Storry, 1962) and 58 mg/dl for Na (Jenness, 1974) but were slightly lower than the 125 mg/dl for Ca and 138 mg/dl for K reported for bovine milk (Jenness, 1974). Concentrations of K in milk were approximately 7.5 times greater than plasma concentrations. Similar findings have been reported by Oshima (1978).

In comparison with control cows, milk from cows dosed with KCl had higher concentrations of Mg in trials 1, 3 and 4; Ca in trials 1 and 3; and K in trials 3 and 4 but lower Na in trial 3. Differences between groups were not detected for Mg in trial 2, Ca in trials 2 and 4, K in trials 1 and 2 or Na in trials 1, 2 and 4. Milk concentrations of Mg and Na remained relatively constant over the sampling period for both groups but Ca and K concentrations increased over time (Tables 26, 27, 28 and 29).

Milk K concentrations increased ($P < .05$) over time for KCl dosed cows and were higher ($P < .05$) at 80 and 160 after dosing than all other times. Milk K concentration of KCl dosed animals were higher than controls at 80 and 160 minutes in trials 3 and 4 but not different in trials 1 and 2 where samples from the 10, 20, 40 and 80 minute sampling times were not included in the analysis. Milk K concentrations are controlled by the Na-K ATPase pump (Oshima, 1978) which could account for the increase in milk K and Mg concentrations of KCl dosed cows. When activated by the increase in K from the KCl dose, the Na-K ATPase pump could increase the amount of K taken up by the mammary gland. The

TABLE 25. Effect of KCl treatment on milk minerals (mg/dl).

Treatment	Trial 1	2	3	4
			Mg	
Control	9.34[a]	9.48	7.35[a]	7.85[a]
KCl	15.24[b]	9.74	8.07[b]	8.73[b]
			Ca	
Control	117.0[a]	110.3	98.0[a]	100.9
KCl	131.0[b]	110.9	101.9[b]	101.6
			K	
Control	106.0	111.7	85.7[a]	89.6[a]
KCl	104.5	116.2	93.1[b]	98.9[b]
			Na	
Control	57.69	91.93	67.27[a]	67.01
KCl	49.54	81.56	55.41[b]	68.01

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

TABLE 26. Effect of KCl treatment on milk Mg (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	6.98 ac	7.24 abc	7.35 abc	7.42 bc	7.58 bc	7.58 bc
KCl	7.77 ad	8.09 abd	8.15 abd	7.99 abd	8.14 abd	8.30 bd
	Trial 4					
Control	7.73	7.93 c	8.03	7.93	7.89 c	7.61 c
KCl	8.18	8.49 d	8.41	10.19	8.61 d	8.44 d

a,b Means within rows in each trial with different superscripts are different ($P < .05$).

c,d Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 27. Effect of KCl treatment on milk Ca (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	91.7[a]	95.4[ac]	98.7[bc]	99.5[bc]	102.2[b]	101.0[b]
KCl	95.9[a]	98.9[abd]	102.5[bd]	102.7[bd]	105.7[b]	105.5[b]
	Trial 4					
Control	97.6	100.8	102.4	101.6	102.6	100.1
KCl	98.8	100.7	100.4	101.9	104.4	103.2

[a,b] Means within rows in each trial with different superscripts are different ($P < .05$).

[c,d] Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 28. Effect of KCl treatment on milk K (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	82.6	85.1	86.7	84.2[d]	87.8[d]	88.1[d]
KCl	86.1[a]	88.3[a]	91.7[ac]	93.2[ace]	98.1[bce]	100.9[be]
	Trial 4					
Control	90.2	87.6	87.1[d]	89.5	90.9[d]	92.4[d]
KCl	91.8[a]	95.1[a]	95.9[ae]	96.6[a]	106.1[be]	107.3[be]

a,b,c Means within rows in each trial with different superscripts are different ($P < .05$).

d,e Means within columns in each trial with different superscripts are different ($P < .05$).

TABLE 29. Effect of KCl treatment on milk Na (mg/dl) over sampling period

Treatment	Time (minutes) 0	10	20	40	80	160
	Trial 3					
Control	65.78	67.13[c]	68.52[c]	67.05[c]	66.88[c]	68.33[c]
KCl	59.65[a]	58.28[ad]	55.63[abd]	56.14[abd]	51.62[bd]	51.28[bd]
	Trial 4					
Control	62.84	67.26	71.74	69.74	67.35	63.02
KCl	116.46	60.99	60.53	62.17	56.27	53.71

[a,b] Means within rows in each trial with different superscripts are different ($P < .05$).

[c,d] Means within columns in each trial with different superscripts are different ($P < .05$).

pump requires Mg to operate (Martens et al., 1978) which could increase the amount of Mg taken up by the mammary gland.

III. GENERAL DISCUSSION

Environmental factors have been implicated as a major cause of grass tetany. The large differences in data between years, trials and sampling days suggest that environmental factors played a major role in this study. Cold exposure and fasting have been implicated in the etiology of grass tetany (Herd, 1966; Sykes et al., 1969; Terashima et al., 1982). These factors appeared to have a large effect during trial 1. Several other environmental factors have been suggested to play a role in grass tetany, including precipitation, soil type, species of grass and forage Mg content (Kubota et al., 1980). One or a combination of these factors may have caused the variability of data over time, between sampling days, years and trials.

Forage content of Mg, Ca and K were similar to those reported by Banet (1983) and Hodge and Bell (1983) for the same pastures. Effects of K fertilization were similar to those reported by Kemp (1960), who reported that K fertilization decreased forage Ca and Mg content and increased forage K. Banet (1983) and Hodge and Bell (1983) also reported that forage Ca decreased and K increased when pastures were fertilized with K. These researchers however reported that K fertilization had no effect on forage Mg content.

Cows grazing the K fertilized pasture had lower plasma and urine concentrations of Mg and Ca. This decrease in plasma and urinary Mg and Ca appeared to be the result of cows consuming young, rapidly growing pasture. Ramsey et al. (1979) reported a decline in plasma Mg after cows

were switched from a wintering ration to lush spring pasture. A reduction in the plasma and urinary Mg concentrations of cattle grazing pastures fertilized with K has also been reported by Kemp (1960), Banet (1983) and Hodge and Bell (1983). However these researchers reported that K fertilization had no effect on plasma Ca concentrations. Banet (1983) and Hodge and Bell (1983) also reported that K fertilization had no affect on plasma K. Milk concentrations were unaffected by K fertilization of pastures. Rook and Storry (1962) stated that milk mineral content remains relatively constant regardless of diet. Hodge and Bell (1983) also reported that K fertilization did not effect milk mineral content. Several researchers have examined the effects of excess dietary K on mineral metabolism. Greene et al (1983b) reported that excess dietary K resulted in a reduction in the absorption of Mg. In our study excess K supplied as KCl resulted in a reduction in plasma Mg. Bohman et al. (1969) also reported a reduction in plasma Mg when cattle were given KCl. Banet (1983) reported that plasma Mg was unaffected by KCl administration. However the amount of KCl given was 140g given twice daily with samples being taken 12 hours after KCl administration. Greene et al, (1983b) also reported that dietary K decreased urinary Ca concentrations but had no effect on plasma Ca or Ca absorption. Banet, (1983) reported that KCl bolusing had no effect on milk mineral content. However in our current study KCl administration tended to have elevated concentrations of milk Mg, Ca and K which would further stress the reduced plasma Mg. It appears from these data that K fertilization and excess dietary K may increase the incidence of hypomagnesemia, however they do not appear to be the main causative agents. It appears that factors such as temperature, soil type,

precipitation and forage type or some other unidentified factor may control the triggering of grass tetany.

CHAPTER V

SUMMARY

Four pasture trials using lactating beef cows with young calves were conducted in 1984 and 1985. The objectives of this study were to compare K fertilization with KCl boluses as sources of excess K for lactating beef cows and to determine the effects of K sources on plasma, milk, fecal and urine minerals. Cattle were placed on one of two adjacent pastures. One pasture was fertilized with 112 kg/ha of K while the second received no K fertilization. Half the cows on each pasture recieved an oral dose of KCl while the remaining half recieved no KCl.

Administration of 280g of KCl by liquid drench resulted in the death of one cow from K toxicity. When 280g of KCl was given by gelatin capsule two more animals experienced episodes of potassium toxicity. These animals were treated with a 200 ml solution of 30% $MgCl_2$ given as an enema. The literature indicated that 280g of KCl was well below the amount required to produce potassium toxicity. It appears that K may be more toxic when given to cattle grazing early spring pastures. When 220 g of KCl was given to the cows by gelatin capsule no signs of K toxicity were observed.

Cattle grazing the K fertilized pasture and KCl dosed animals had reduced plasma and urine concentrations of Mg, indicating a reduction in the absorption of Mg. Cattle consuming the K fertilized pasture had depressed plasma and urine Ca concentrations. KCl dosing had inconsistent effects on plasma Ca levels. In 1985 administration of KCl resulted in a depression of urine Ca concentrations. Effects of K fertilization on plasma Na were inconsistent. Plasma and urine

concentrations of K were not affected by K fertilization. Dosing with KCl resulted in elevated urine and plasma levels of K. Plasma Na levels were elevated for KCl bolused cows. The effects of K fertilization on milk minerals were variable and inconsistent. Concentrations of Mg, Ca and K in the milk tended to be higher for KCl dosed cows than their control counterparts. Effects of KCl administration on milk Na levels were inconsistent. Results indicated that the incidence of grass tetany may be increased by both K fertilization and oral administration of KCl.

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APPENDIX

TABLE A1. Effect of KCl treatment on fecal minerals (mg/g).

Treatment	Trial 1	Trial 2
	Mg	
Control	3.10	7.80
KCl	2.98	6.22
	Ca	
Control	8.62	27.97
KCl	8.17	322.26
	K	
Control	6.68[a]	13.25[a]
KCl	8.47[b]	10.13[b]

[a,b] Means within columns for ech mineral with different superscripts are different ($P < .05$).

TABLE A2. Effect of pasture on fecal minerals (mg/g).

Pasture	Trial 1	Trial 2
	Mg	
Control	2.76[a]	8.15[a]
+K	3.28[b]	6.17[b]
	Ca	
Control	8.19	458.94
+K	8.52	22.11
	K	
Control	7.49	8.33[a]
+K	7.91	13.59[b]

[a,b] Means within columns for each mineral with different superscripts are different ($P < .05$).

VITA

Francis John Mueller, son of Francis and Trudy Mueller was born in Syracuse, New York on October 21, 1960. He attended elementary and secondary schools in Jamesville and Dewitt, New York, and graduated from Jamesville Dewitt High School in June of 1978. In January of 1979 he enrolled in the State University of New York, Morrisville and received the Associate of Science degree in Agriculture in January of 1981. He immediately enrolled at Cornell University receiving the Bachelor of Science degree in Animal Science in January of 1983. His graduate studies were initated in September of 1983 at The University of Tennessee, Knoxville, where he worked as a graduate research assistant. He received the Master of Science degree in Animal Science, specializing in ruminant nutrition, in December of 1985.