THE EFFECT OF DIETARY CATION-ANION BALANCE ON ACID-BASE STATUS AND MINERAL BALANCE IN HORSES

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CHAPTER I

INTRODUCTION

In today's competitive horse industry, proper nutrition is imperative to gaining the full genetic potential of the horse, regardless of the events taken part in, or the production state of the animal. Many recent studies dealing with basic nutrition and the physiological impact of nutrion have demonstrated that proper nutrition can improve the performance of the equine. The issue of the effect of Dietary Cation-Anion Balance (DCAB) on performance variables and acid-base status has been studied extensively in other domestic species. The DCAB exerts its influence on the physiological state of the animal via the dietary elements sodium, potassium and chloride, and consequently may have a major impact on the acid-base status of the animal, as well as on various production variables. At the present time, the NRC (1989) has no specific reccomendations on DCAB for any class of horses. It is hoped that by quantifying an optimum DCAB for various classes of horses that performance may be improved in the areas of exercise performance, digestibility efficiency, nutrient utilization and growth. However, this subject of interest has not gained much effort from equine researchers until recently.

The purpose of this project was to determine the effects of DCAB on mature, sedentary horses. The objectives of this study were: 1) To

determine the effect of vaying cation-anion balances on the acid-base status of the horse by measuring urine pH, blood pH and blood gases; 2) To determine the effect of varying cation-anion balances on mineral balances, particularly calcium.

CHAPTER II

LITERATURE REVIEW

History Of Dietary Cation-Anion Balance

The electrolytes sodium, potassium and chloride have not received the primary attention of animal nutritionists until recently. This may be explained by the fact that deficiencies of any one of these minerals is rare in today's common animal rations. Sodium and chloride are supplied in the diet in the form of common salt (NaCl) which is commonly added to rations. Furthermore, most livestock rations today contain an excessive amount of potassium. More importantly, though, is the amount of these minerals in relation to the amount of the others in ration ingredients and supplements. Recently, it has become apparent to researchers, particularly those involved with dairy cattle, poultry and swine, that the ratio between these minerals has a major impact on animal nutrition.

One of the earliest researchers to propose an equation which would define a balance containing the electrolytes sodium, potassium and chloride was Mongin (1980). This equation took into account these monovalent elements as they seemed to have the most metabolic impact on acid-base physiology, and they appear to be the ones most readily available via absorption from the gut (Austic, 1988). This equation is defined as follows: meq $(Na^+ + K^+) - Cl^-$/100g diet dry matter. This equation uses the units milliequivalents (meq), as opposed to

milligrams, as these elements produce their physiological effects on the body according to their valence rather than their weight.

The phrases dietary cation-anion balance (DCAB), and acid-base balance have both been accepted to descibe the relationship of these elements to one another, however, since the DCAB exerts its influence on acid-base physiology via the diet and the acid-base mechanisms of the body, it has become the most popular and accepted term.

DCAB Effect on Other Species

Rabbit

The first report of the cation-anion balance of the diet having an effect on physiological factors was Morgen and Berger (1915). These researchers demonstrated that sodium carbonate was more effective than sodium chloride in attempting to increase the mineral content in rabbit bones. These authors suggested that the carbonate salt acted to increase the alkaline reserve. Thacker (1959) inferred from this hypothesis that calcium, potassium, sodium and magnesium deficiencies could be caused by the manipulation of their level in the ration and the cation-anion balance of the ration. Thacker (1959) demonstrated that rabbits fed a ration based on timothy hay grown in heavily fertilized soil which previously had not supported proper growth, hemoglobin or bone ash levels (Keener and Thacker, 1958) was rendered adequate by the addition of a salt of sodium, potassium, magnesium or calcium carrying an anion capable of being oxidized to CO_2 and H_2O in the animal. It was also suggested that the mineral imbalance suffered by the animals in this study induced a calcium and potassium

deficiency in the animals when the diet contained adequate levels of these elements.

Rat

The influence of DCAB in the rat has been concentrated on the subsequent effect on bone physiology. In 1969, Barzel and Jowsey demonstrated that rats consuming ammonium chloride for a long period of time had increased bone resorption. However, this loss of bone tissue was prevented by the ingestion of sodium and potassium carbonate, apparently by the stimulation of bone formation. This physiological response of the bone was attributed to changes in systemic acid-base balance, and it was therefore suggested that the intracellular mechanism controlling calcium deposition and resorption in the bone was sensitive to systemic pH.

Newell and Beauchene (1975) investigated the effects of acid stress and age on renal, serum and bone responses in 13 and 25 month old rats fed ammonium chloride at 2% of the diet for nine months. The acid stressed animals showed significant decreases in urinary pH, and also significant increases in urinary calcium and phosphorus excretions, as well as total acid excretion in the urine. However, analysis of bone showed no effect on calcium content of the bone due to the diet. Petito and Evans (1984) evaluated the effects of acid ingestion, phosphates and protein on calcium status in growing rats. Ammonium chloride was fed to the treatment group of animals at 1% of the diet. Treated rats had decreased blood pH and as well had increased urinary cAMP and calcium concentrations. Furthermore, these rats had a two-fold increase in fecal calcium, and had lower specific gravity of the femur. Beck and Webster (1976) suggested that metabolic acidosis inhibits the tubular reabsorption of calcium in the nephron,

and that this inhibition, coupled with the ingestion of ammonium chloride may explain how cAMP and calcium could both be excreted in the urine at higher levels. Goulding and Campbell (1984) demonstrated that rats given salt supplements excreted more calcium in the urine and had less calcium in the bone than control rats.

Poultry

Poultry nutritionists were the first livestock nutritionists to recognize and study the effects of dietary cation-anion balance on production parameters. Early research in this area was concentrated on the effects that the elements sodium, potassium and chloride had on growth and food consumption through their roles in the maintenance of osmotic pressure and acid-base status of the animal.

Neishiem and coworkers (1964) demonstrated that chicks suffered dramatic decreases in growth rate when fed excesses of dietary chloride or sulfate supplied as glutamic acid hydrochloride, calcium chloride or calcium sulfate. However, this decreased growth rate was alleviated by supplying equimolar amounts of potassium or sodium supplied in glutamate or carbonate. These researchers also demonstrated that chicks suffered decreased growth rate when fed excess sodium supplied as sodium glutamate. This depression in growth was alleviated when equivalent amounts of chloride were added. Melliere and Forbes (1966) performed a similar study and demonstrated that food consumption and growth were maximized when chicks were fed a cation-anion ratio of 1.2 to 1.8. A ratio of 0.6 cation to anion ratio almost completely inhibited growth. These authors also reported that sodium and potassium chlorides did not reduce food consumption or weight gain when added to the diet at levels equal to the highest amount of hydrochloride. Feeding excess calcium did not

alleviate the depression in growth demonstrated by excess chloride, however, excess magnesium intake partially alleviated the depression.

During the 1960's, a group of researchers began to study the effect of acid-base balance on egg shell calcification in the hen (Frank and Beger, 1965; Howes, 1967; Anderson, 1967; Mongin, 1968). All of these studies demonstrated that the calcification process of the egg shell could be altered by manipulating the acid-base status of the laying hen.

In 1972, Cohen, Hurwitz and Bar studied the effects of dietary sodium and chloride on blood pH, pCO_2, HCO_3, Cl and Na on laying hens during egg shell formation. These authors hypothesized that dietary sodium and chloride were the alkalogenic and acidogenic agents, respectively, and that the acid-base response would depend on the ratio between these two components. It was demonstrated that excess dietary sodium fed with a constant level of dietary chloride produced an alkalosis, and excess dietary chloride fed with a constant level of sodium produced an acidosis. When sodium and chloride were added to the total diet in equal amounts, no differences in the acid-base balance of the animal were detected. Therefore, these researchers determined that the acid-base balance of the body, as measured by blood pH, pCO_2 and HCO_3, was a function of the ratio of sodium to chloride, and not by the absolute amount of each. These researchers also stated that the actual pH of the diet was irrelevant in producing a metabolic alkalosis or acidosis. Feeding calcium chloride with a pH near neutral caused an acidosis, whereas feeding an acid salt such as sodium monophosphate caused an alkalosis.

In 1974, Cohen and Hurwitz studied the response of blood parameters to dietary sodium, potassium and chloride in laying hens. These authors demonstrated that supplemental sodium or potassium in the diet resulted in an increase in blood pH and HCO_3, while the supplementation of chloride resulted in

a decrease in these same parameters. These findings suggested that sodium and potassium are additive in their response to offset the metabolic acidosis caused by excess dietary chloride. These findings agree with that of Neshiem et al. (1964) who demonstrated that growth retardation caused by excess dietary chloride could be alleviated with the addition of sodium and potassium salts void of chloride to the diet.

In 1980, Mongin was the first to suggest a cation-anion balance equation using the elements sodium, potassium and chloride. This equation reads as follows; meq (Na + K) - Cl/100g diet dry matter. This equation could be used to quantify the acid-base balance of the ration. The author's defense of this equation was based on the results of two experiments. The first was performed by Mongin and Saveur (1973) who demonstrated that animals fed diets with a range of -20 to +40 meq/100g had plasma bicarbonate levels linearly related to that sum. The second experiment was performed by Hurwitz et al. (1973) who demonstrated that animals fed diets containing equivalent amounts of sodium and potassium had a blood pH markedly dependent on dietary chloride.

Also in 1980, Hamilton and Thompson demonstrated a decrease in blood pH, bicarbonate level and eggshell strength in hens when the chloride level in the diet was increased from .11 to 2.13%. These findings agreed with those of Hall and Helbacka (1959), Hunt and Aitken (1962) and Saveur and Mongin (1971) who reported that eggshell calcification was depressed in hens fed excessive levels of acid chlorides. Furthermore, it has been demonstrated that egg shell strength was increased when hens were fed a diet that increased the alkaline reserve (Frank and Burger, 1965; Howes, 1967; Mongin, 1968). In 1984, Austic also reported a decrease in eggshell strength and thickness in hens consuming diets with excess dietary chloride.

The effect of cation-anion balance has also been associated with bone abnormalities in fowl, particularly tibial dyschondroplasia (TD). In 1965, Leach and Neshium described this disorder in young chicks, and later discovered that this condition could be affected by the cation-anion balance of the diet (Leach and Neshium, 1972). In 1978, Saveur and Mongin reported an increase in the incidence of TD resulting from metabolic acidosis caused by excessive dietary chloride. Halley et al. (1987) studied the effect of dietary mineral balance on growth, leg abnormalities and blood base excess in chicks. It was demonstrated that base excess was negatively correlated with the incidence of TD and with 3-week body weights. These findings agree with later work that demonstrated the relationship between the anionic content of the diet and a subsequent alteration in acid base status and a higher incidence of TD (Edwards, 1984; Hamilton and Thompson, 1980; Hurwitz et al., 1973; Mongin, 1981).

In 1983, Riley and Austic studied the effects of dietary electrolytes on digestive tract pH and acid-base status of chicks. The cation-anion balance of the diet was altered by the addition of potassium bicarbonate or calcium chloride. It was reported that chicks consuming a diet with excess chloride had decreased plasma bicarbonate, base excess of the blood and pCO_2. The pH of the crop was also depressed by dietary chloride, however, the pH of the proventriculus, duodenum, or middle and distal portions of the small intestine were not affected.

Swine

In the early 1980's, swine researchers took note of the effect of cation-anion balance in the diet. In 1981, Yen and others studied the effect of calcium chloride as a regulator of feed intake and weight gain in pigs. It was demonstrated that crossbred barrows fed a basal diet with 4% CaCl had lower

daily feed intake, weight gain and gain/feed ratios as compared to those fed a basal diet. Those pigs fed the CaCl also had lower blood pH, HCO_3, tCO_2 and base excess. These parameters were restored to normal levels in pigs fed a diet containing calcium chloride and sodium bicarbonate. These authors also made note of the fact that even though pigs fed the CaCl and $NaHCO_3$ diet had persistently high Cl levels in the plasma, the chloride to bicarbonate ratio was restored to that observed in pigs fed a basal diet.

Patience et al. (1987) fed 8 - 12 week old pigs five rations with electrolyte balances (defined in this study as meq (Na + K) - Cl/kg) ranging from -85 to +341. It was demonstrated that growth and feed intake were maximized in those pigs fed diets with a balance between 0 and 341, while these parameters were decreased in those pigs consuming the -85 diet. Furthermore, as the electrolyte balance in the diet dropped below a base level of +175 meq/kg, blood pH and bicarbonate levels dropped indicative of a metabolic acidosis. Golz and Crenshaw (1984) studied the importance of sodium, potassium and chloride on growth in young swine. These authors suggested that dietary potassium and chloride levels have an interactive effect on gain when the sodium level is held constant. Optimum growth occurred with a K to Cl ratio of approximately 2:1 (.57% K and .27% Cl) when the sodium level in the diet was held between .03 and .60%.

In 1990, Haydon and West examined the effects of electrolyte balance on nutrient digestibility in growing pigs. Apparent nutrient digestibilities were determined by fitting the animals with ileal T-cannulas. Experimental diets consisted of a corn-soybean meal base, and electrolyte balance was altered by substituting $CaCl_2$ for $CaCO_3$, or $NaHCO_3$ for corn and soybean meal, resulting in four experimental diets with cation-anion balances of -50, +100, +250 and +400 meq/kg diet dry matter. Apparent ileal digestibility was increased linearly

for N, energy, dry matter and all amino acids, except alanine and methionine, as the electrolyte balance of the diet was increased. Furthermore, blood pH, tCO_2, HCO_3 and base excess concentrations increased with increasing dietary electrolyte balance.

Dairy Cattle

Coppock (1986) reviewed the current literature on the effect of DCAB on production parameters in livestock. At that time, there was very little interest in this area by dairy cattle researchers. Coppock evaluated and calculated the DCAB in various beef and dairy experiments that had been conducted. It was suggested that the ruminant could more easily withstand a higher DCAB than could poultry. In 1984, Escobosa and coworkers demonstrated that cows consuming a diet with a negative cation-anion balance suffered a decreased feed intake. Since then, an enormous amount of progress has been made in studying the effects of DCAB on production traits by dairy researchers.

In 1984, Block studied the effects of DCAB on reducing the incidence of milk fever in dairy cows. Previous research had indicated a relationship between dietary anions and an increased calcium availability (Dishington, 1975; Ender et al., 1971; Lomba et al., 1978). Block (1984) demonstrated that cows fed a highly anionic diet (-128 meq/kg) during the dry period had decreased incidence of parturient pariesis during subsequent lactation. Tucker et al. (1988) studied the effects of DCAB on milk, blood, urine and rumen fluid in lactating dairy cattle. It was demonstrated that increasing the DCAB from -100 to +200 meq/kg resulted in a linear increase in blood pH and bicarbonate, while actual milk yield was increased 8.6%. These researchers also noted that the responses observed, except for blood bicarbonate, could be attributed to the DCAB itself, and not the

effects of a single ion. Because maintaining the blood pH at a constant level is critical for normal body function, several mechanisms in the body exist to maintain the concentration of blood bicarbonate to pCO_2 at a constant ratio. According to Tucker et al. (1988), this control is accomplished by a respiratory response by adjusting the respiration rate to control the blood levels of pCO_2, and a renal response by adjusting the excretion of bicarbonate to control blood bicarbonate concentrations. It has been proven that altering the DCAB has a marked effect on blood acid-base balance (Tucker et al., 1988).

In 1990, Beighle and others reported that dairy calves fed diets with a low cation-anion ratio had higher concentrations of phosphorus in the blood and feces versus those calves fed diets with a higher cation-anion balance. Those calves fed the low DCAB also showed lower concentrations of phosphorus in the bone. These researchers noted that when a low phosphorus diet was fed along with the low DCAB, these effects were amplified, indicating an interaction between DCAB and dietary phosphorus on the changes seen in blood, bone and fecal phosphorus concentrations.

In 1991, Tucker and others studied the influence of dietary sodium bicarbonate on potassium metabolism in young calves. According to this study, feed intake was not affected by supplemental potassium chloride or sodium bicarbonate, however, average daily gain increased with increased potassium and decreased with increased sodium bicarbonate. Urinary calcium excretion also declined with increased sodium bicarbonate while urine pH showed an increase. Tucker and others (1991) also studied the influence of calcium chloride on systemic acid-base balance and calcium metabolism in dairy heifers. These researchers demonstrated that urinary calcium excretion and blood free proton concentration (H^+) increased with increasing dietary $CaCl_2$, while blood bicarbonate and urine pH decreased. These authors suggest that the increased

Ca excretion in the urine was due to either an increased bone mobilization or increased intestinal absorption of calcium. It was also noted that increasing the dietary level of chloride caused a subsequent increase in both plasma chloride and urinary chloride excretion.

In 1991, Goff and others studied the effects of the addition of chloride to a prepartal diet fed to dairy cows which was also high in cations. These researchers demonstrated that in cows fed highly anionic diets, parathyroid hormone (PTH) had a more dramatic effect on renal production of 1,25 dihydroxyvitamin D, thus increasing intestinal absorption of calcium. Furthermore, the response of the bone to parathyroid hormone, which is osteoclastic bone resorption, was more responsive to PTH as plasma hydroxyproline concentration was higher in those cows fed the highly anionic diet. These researchers stated that the addition of anions to the diet is thought to induce a metabolic acidosis in the cow, which facilitates bone calcium resorption (Block, 1984). This is in agreement with Beck and Webster (1970) who indicated that bone, and perhaps renal tissue, in rats is refractory to the effects of PTH in the alkaline state and the stimulatory effects of PTH are enhanced during metabolic acidosis. It has been shown in dairy cattle and poultry (Tucker, 1988; Austic, 1984) that this increased PTH activity is a possible cause for increased levels of ionized or free calcium in the blood and, subsequently, increased levels of calcium in the urine.

Equine

Mineral Requirements

Sodium. The National Research Council (NRC), (1989) states that in many cases, the sodium concentration of natural feedstuffs for horses is lower than 0.1%. Sodium is therefore commonly added to the total diet in the form of sodium chloride, or common salt, between a range of 0.5 to 1.0%, or as trace mineralized salt. Sodium is often described as the major extracellular cation for its role in acid-base status and the osmotic regulation of body fluids. The optimal sodium concentration of the diet has been reported to be between 1.6 and 1.8g/kg diet dry matter for growth, maintenance and late gestation and 3.6 g/kg diet dry matter for moderate to heavy work (Jarrige and Martin-Rosset, 1981). Since there is limited data on specific requirements for sodium and the effect of physical activity and environment on the animals requirements, the NRC (1989) does not make specific recommendations, however it is stated that the sodium concentration in the maintenance diet be no lower than 0.1%.

Potassium. Potassium is the major intracellular ion involved with acid-base balance and the osmotic regulation of body fluid. The NRC (1989) lists the potassium concentration of forages and oilseed meals as 1 to 2% dry matter, and that of common cereal grains (corn, wheat and oats) to be 0.3 to 0.4%. Hintz and Schryver (1976) estimated that mature horses required .06 g/kg of body weight/day, or approximately 0.4% of the diet. Therefore, if forage constitutes a significant portion of the diet, than potassium requirements should easliy be met. Drepper and others (1982) estimated the potassium requirements for a 600 kg horse to be 22 g/day for maintenance. Based on this research, the NRC (1989) estimates the potassium requirement for maintenance to be 0.05 g/kg of body weight or 1.52 g/Mcal of DE.

Chloride. Chloride is an important extracellular anion involved in acid-base balance, osmotic regulation, as a minor component of bile, and in the formation of hydrochloric acid which is an important component of gastric secretions necessary for proper digestion. However, chloride requirements of horses have not been established, and requirements are thought to be met when sodium requirements are met with salt (NaCl).

Magnesium. Magnesium is important as an activator of many enzymes, and the magnesium concentration of common feedstuffs has been listed at 0.1 to 0.3% (NRC, 1989). Researchers have estimated that the true absorption of magnesium from feeds is between 40 and 60% (Hintz and Schryver, 1972;, Meyer, 1979) and between 42 and 45% (McKenzie, 1981). In 1982, Drepper and others proposed a daily magnesium requirement of 12 g for maintenance in a 600 kg horse. Using the conservative value of 40% absorption efficiency, the NRC (1989) suggests a magnesium requirement of approximately 15 mg/kg body weight/day, or .46 g/Mcal DE.

Sulfur. The requirements of sulfur by the equine has received very little attention by researchers. Feeding adequate, high quality dietary protein will usually provide a minimum of 0.15% organic sulfur. According to Jarrige and Martin-Rosset (1981) and the NRC (1978), this is adequate to meet the horses needs.

Calcium. The 1989 NRC estimates the true absorptive efficiency of calcium is approximately 70% in young horses and approximately 50% in mature horses. For the purpose of estimating calcium requirements for all classes of horses, however, the NRC (1989) suggests a value of 50% absorptive efficiency be used,

due to the possibility of calcium being bound to phytates in feed, rendering it unavailable to the animal. Using this value, the calcium requirement for maintenance is stated to be .04 g/kg of body weight/day or 1.22 g/Mcal of DE/day.

Phosphorus. The efficiency of true phosphorus absorption in the horse is variable due to the age of the horse and the source and concentration of phosphorus in the diet. The NRC (1989) estimates that the efficiency of true phosphorus absorption ranges between 30 and 55%. However, the NRC (1989) uses the more conservative figure of 35% for horses at maintenance, gestating mares and horses at work as they all consume mainly plant sources of phosphorus. Using the above values, the NRC (1989) lists the phosphorus requirements for maintenance at 28.6 mg/kg of body weight/day or 0.87 g/Mcal of DE/day.

Equine Studies

In 1970 Schryver and others studied the effect of calcium intake on skeletal metabolism and the calcium homeostatic mechanisms of young, growing horses. Three dietary levels of calcium were fed ranging from below, equal to, and above that which the NRC (1966) recommended. These diets contained 0.15%, .80% and 1.5% of the recommended amount of calcium, which was supplied in the form of calcium carbonate at the expense of hay and corn in the diet. These researchers also used a kinetic analysis with a radioactive isotope of calcium so that more accurate determinations of the rate of exchange of calcium between body fluids and bone, and the rate of deposition and removal of calcium from the bone could be measured. These researchers demonstrated a large variation

between intake groups in excretion and retention of calcium in order to maintain calcium homeostasis but there was no difference in the concentration of calcium in the plasma or on the size of the exchangeable pool. Ponies fed the lower calcium diet had increased fractional absorption of calcium and had a decreased renal excretion rate. Furthermore, bone resorption was increased above the deposition rate resulting in a net transfer of calcium from the bone into the exchangeable pool. Despite these homeostatic mechanisms, these ponies experienced a net negative calcium balance. These researchers also observed opposite responses in ponies fed the high calcium diet. Unlike the rate of removal, however, the deposition rate of calcium was insensitive to the dietary level of calcium.

In a later study, Schryver and coworkers (1971a) studied the effect of high dietary phosphorus levels on calcium utilization and skeletal metabolism in growing Shetland ponies fed .4% calcium and either .2% or 1.2% phosphorus in the diet. Ponies fed the high phosphorus diet were observed to have increased phosphorus retention and plasma levels. However, absorption of calcium, renal excretion and retention of calcium were all decreased while total and endogenous fecal calcium excretion were increased. Furthermore, Schryver et al. (1971b) showed that renal phosphorus excretion, total phosphorus absorption from the gut and phosphorus retention were all dependent on phosphorus intake. The efficiency of phosphorus absorption averaged 45% across all diets, however.

In 1987, Schryver and others studied the effects of voluntary salt intake in mature, sedentary horses and its effect on mineral metabolism. Diets containing 1, 3 and 5% NaCl were fed, with a mean daily salt consumption ranging from 19 to 143 g and a mean of 53 g. These researchers demonstrated that fecal excretion of calcium was higher in those horses consuming the 1% NaCl diet, and determined that calcium absorption and retention were greater at the higher levels

of consumption. Furthermore they noted that phosphorus absorption and retention were greater at both the 3 and 5% levels of intake. These researchers also determined that urinary sodium excretion was directly related to intake and that urinary excretion was the primary excretory path for sodium, as fecal excretion, intestinal absorption and retention of sodium were not affected by intake.

In 1989, Young and others evaluated the extent of mineral losses in feces, urine and sweat in miniature horses at rest and during exercise. During the exercise period, daily sodium intake increased and there was trend for daily fecal excretion of sodium to increase. Also, urinary excretion of sodium decreased, possibly due to large amounts of sodium being lost in the sweat. In this same trial, both daily chloride intake and fecal concentration of chloride increased, contradictory to Schryver et al. (1987). During the exercise period, daily intake of potassium and excretion of potassium in the feces both increased, while daily intake of calcium increased as did daily fecal excretion of calcium, resulting in an increase in daily calcium retention. Furthermore, both the daily intake and the daily fecal excretion of phosphorus were increased, resulting in an increase in daily retention.

In 1974, Milne studied the effects of exercise on blood parameters, acid-base balance and electrolyte levels. He proposed a linear relationship between the changes in arterial and venous blood pH, pCO_2 and HCO_3 in response to exercise, and suggested that arterial blood parameters could be predicted from venous blood values, with the exception of pO_2.

DCAB Studies

In 1989, Topliff and coworkers studied the effect of a low (+6.5 meq/kg) vs. a high (+150 meq/kg) DCAB on calcium and chloride metabolism in

exercising mares. No change in serum calcium or chloride concentrations were observed. Horses consuming the low cation-anion balance excreted more calcium in the urine (84.7 mg/dl) than those consuming the high cation-anion diet (9.2 mg/dl). Those horses consuming the low DCAB also excreted more total calcium per day, as total urine output was not different. Furthermore, those horses consuming the low DCAB had higher amounts of chloride in the urine (176.1 meq/l) as opposed to those consuming the high DCAB (124.8 meq/l). This response of urinary excretion of calcium and chloride was attributed to the acid producing power of the diet.

In 1992, Stutz and coworkers studied the effects of DCAB on blood variables in exercising horses. Four diets were fed with DCABs of +5 (L), +107 (ML), +201 (MH) and +327 (H) meq/kg dry matter. Treatments were formed by the addition of calcium chloride and ammonium chloride to diet L, calcium chloride to diet ML and sodium bicarbonate and potassium citrate to diet H. While at rest, those horses consuming diet L had lower venous blood pH, pCO_2 and HCO_3 concentrations as compared to those consuming the MH and H diets. However, no differences were observed in blood pH or acid-base parameters between treatments after anaerobic exercise.

During the same trial, Wall et al. (1992) evaluated the effects of DCAB on urine pH and urinary mineral excretion in exercising horses. The diets fed were the same as in the trial performed by Stutz et al. (1992). These researchers observed a significant decrease in urine pH as the DCAB was decreased. Furthermore, they observed that horses consuming the low diet excreted more calcium and chloride in the urine than those consuming the medium high and high diets. Also, horses excreted more sodium in the urine when consuming diet H versus those consuming the other diets. These researchers stated that,

depending on the calcium intake, exercising horses consuming a low DCAB could have a negative calcium balance.

It is evident from experiments in swine, poultry, dairy cattle and other species that the cation-anion balance of the diet can have a significant effect on the acid-base status in the animal, as well as on various production parameters in these species. It is also evident from mineral studies in other species and from limited studies in the horse that the DCAB can have a major effect not only on the acid-base status in the animal but also an effect on mineral metabolism. It was therefore the purpose of this trial to study the effects of feeding varying levels of DCAB and the subsequent effects on acid-base status and mineral balance in the sedentary horse.

CHAPTER III

MATERIALS AND METHODS

Experimental Design

Four mature stock type geldings, two Quarter Horses, one Appaloosa and one 1/2 Arabian 1/2 Quarter Horse were used in a 4x4 Latin square design experiment to study the effects of varing Dietary Cation-Anion Balances on acid-base status and mineral metabolism in the non-exercised horse. The 16 week trial consisted of a 3 week dietary adjustment period followed by a 72 hour sample collection period.

Horses were individually stalled and were exercised for 30 minutes daily on a mechanical walker. Horses were fed at 10 AM and 10 PM daily. All horses were immunized and dewormed prior to, and received routine health care throughout the trial.

Experimental Treatments

Diets consisted of a pelleted base concentrate of corn, soybean meal and cottonseed hulls, and was produced at the Oklahoma State University Feedmill. The concentrate was fed in a 60:40 ratio with native prairiegrass hay grown by the Oklahoma State University Purebred Beef Research Center. The complete diet was fed in amounts to maintain a constant body weight throughout the

experimental trial. The four diets were formed by the addition of .50% calcium chloride and .50% ammonium chloride to the low diet (L) (Table I). The medium low diet (ML) was supplemented with .50% calcium chloride. The high diet (H) was formed by the addition of .40% sodium bicarbonate and 1.0% potassium citrate. The medium high diet (MH) served as the control diet and received no additional supplementation. Diets were calculated to contain 2.5 Mcal/kg DM and 9.6% crude protein across all treatments (Table II). Diets were analyzed and determined to contain approximately equivalent amounts of calcium, phosphorus, magnesium and sulphur. This analysis also determined that after supplementation, the high diet contained 1.25% potassium and .40% sodium, while the medium low diet contained .73% chloride and the low diet contained 1.04% chloride. The varying concentration of these minerals gave treatment dietary cation-anion balances of +21, +125, +231 and +350, respectively.

Blood Collection

Arterial and venous blood samples were taken on the first day of each collection period. Approximately 4 weeks prior to the start of the trial, all horses had the carotid artery surgically raised to the subcutaneous level to allow catheterization and the subsequent collection of arterial blood. Arterial blood samples were drawn, using an 18 gauge catheter, for 12 hours beginning at feeding, and hourly thereafter. Venous blood samples were drawn using a 14 gauge catheter, and were drawn at the same time as arterial samples. All blood samples were analyzed immediately for pH, pCO_2, tCO_2, HCO_3, standard bicarbonate, base excess and base excess extracellular fluid, using a blood gas analyzer (Insrumentation Laboratory Model 1304, Lexington, Ma.).

Table I.

COMPOSITION OF DIETS, AS FED BASIS

Ingredient (%)	L	ML	MH	H
Ground Corn	36.80	37.30	37.30	35.90
Soybean Meal	6.00	6.00	6.00	6.00
Cottonseed Hulls	15.00	15.00	15.00	15.00
Dical	.50	.50	.50	.50
Trace Mineral Salt	.50	.50	.50	.50
Limestone	---	---	.50	.50
Chromic Oxide	.20	.20	.20	.20
Calcium Chloride	.50	.50	---	---
Ammonium Chloride	.50	---	---	---
Sodium Bicarbonate	---	---	---	.40
Potassium Citrate	---	---	---	1.00
Prairie Grass Hay	40.00	40.00	40.00	40.00
Total	100	100	100	100
DCAB	**+21**	**+125**	**+231**	**+350**

TABLE II.

DIET ANALYSIS (DRY MATTER BASIS)

	Treatment			
	L	**ML**	**MH**	**H**
DE, Mcal/kg	2.34	2.56	2.56	2.50
Crude Protein	9.60	9.70	9.70	9.50
Calcium, %	.52	.54	.50	.58
Phosphorus, %	.29	.34	.28	.33
Magnesium, %	.15	.16	.15	.15
Potassium, %	.86	.86	.86	1.25
Sodium, %	.22	.28	.32	.40
Sulfur, %	.13	.13	.11	.14
Chloride, %	1.04	.73	.40	.38

Urine Collection

Total urine production was collected, beginning on the first day of each collection period, via urine harnesses, every 4 hours for 72 hours. The volume of urine produced was recorded for every four hour period. A representative sample of 10% of total volume was composited over time for each horse and time period. An additional sample of 100 ml was analyzed for pH using a Fischer Accumet Model 950 pHc meter with a standard glass body combination electrode which accounts for sample temperature. This pH meter was standardized prior to each four hour collection. After analysis of pH, these samples were then acidified with concentrated HCl at 3% of total volume. A separate 20 ml sample was taken at each interval, non-acidified, and frozen for later analysis of chloride.

Fecal Collection

Fecal samples were obtained 6 times randomly over 72 hours of each collection period so that every 2 hours post feeding was represented. Chromium oxide was added at 2% of the total diet as an indigestible marker for the determination of fecal volume. Each sample was identified by horse number, treatment number and time and all samples were immediately frozen in freezer bags for later mineral analysis.

Laboratory Analyses

Urinary Mineral

Calcium, Sodium, Potassium and Magnesium Analysis

For analysis of calcium content of the urine, the composite samples were diluted with a .5% La + .1% K solution for a dilution rate of 1:937.10 and analyzed on a Perkins-Elmer Model 4000 Atomic Absorption Spectrophotometer using a 4 ppm standard, and read at 422.7 nm. For analysis of sodium, composite samples were diluted with distilled, deionized water for a final dilution rate of 1:6503.64. Samples were analyzed using an Atomic Absorption Spectrophotometer using a 1 ppm standard and read at 589.0 nm. For the analysis of potassium, composite samples were diluted with a .1% La solution for a final dilution rate of 1:7431.63. Samples were analyzed using an Atomic Absorption Spectrophotometer using a 2 ppm standard and read at 766.5 nm. For the analysis of magnesium, composite samples were diluted with a .1% La + .1% K solution for a final dilution rate of 1:6503.64. Magnesium concentration was determined using an Atomic Absorption Spectrophotometer, using a .40 ppm standard and samples were read at 285.2 nm.

Phosphorus

For the analysis of phosphorus, composite samples were analyzed using the procedure by Sigma chemical #360-UV using a Gilford Spectrophotometer and read at 340 nm.

Chloride

Urine chloride concentration was determined via potentiometric titration using an HBI Digital Chloridometer (Haake Buchler Instruments, Inc.).

Fecal and Feed Analysis

For the analysis of fecal and Feed Na, Ca, K, Mg and P 1 gram of composited dried sample was dried at 55 degrees C for 24 hours, weighed, and dried again for two hours until a final dry weight was confirmed. Samples were then ashed at 500 degrees C for four hours. Two ml of 1:1 HCl was then added and samples were then boiled on a hot plate at a temperature between 150 and 200 degrees F until evaporated to dryness. Twenty five ml of a blank solution containing 1.5 n HNO3 and 0.5 n HCL was added and samples were then analyzed using Inductively Coupled Plasma Spectroscopy (Model ICAP61 Therno Jarrell-Ash).

Chloride Analysis

For the analysis of fecal and feed chloride, 1 g of dried composited sample was ashed for 4 hours at 500 degrees C. Before ashing, 20 ml of sodium carbonate was added to the dried sample to prevent the loss of chloride during ashing. After cooling, 20 ml of 20% nitric acid was added to the sample. Samples were then boiled on a hot plate on a setting of Low for 30 minutes. Samples were then transferred to 50 ml volumetric flasks, and the flasks were filled to volume with distilled, deionized water. Chloride concentrations were then determined via potentiometric titration using an HBI Digital Chloridometer (Haake Buchler Instruments, Inc.).

Chromium Analysis

Approximately .4 g of fecal and .5 g of feed sample was placed in oven-dried 100 ml beakers, and the air dried sample weight was recorded. Samples were then placed in drying ovens for 24 hours at 60 degrees C. After cooling in dessicators, the beaker and sample were reweighed to determine oven dried sample weight. Samples were then ashed at 500 degrees C for 4 hours. Six ml of an acid mixture (1000 ml DDH, 500 ml H2SO4 and 500 ml H3PO4) was then added to the ashed sample. Samples were then placed on a hot plate and brought to a boil at a setting of 6. Three ml of KBrO3 was added, and the sample was boiled for .5 to 1 minute after SO_3 fumes appeared. The beakers were then allowed to cool to room temperature for 10 minutes. Twenty ml of dilute Bromate was then added and the mixture was brought to a boil at a setting of 4. When the sample changed from clear to milky, the beaker was removed from the hot plate and allowed to cool. The sample was then transferred to 100 ml volumetric flasks and filled to volume with DDH. The flasks were then capped and inverted 3 times. Five ml was transferred to centrifuge tubes and 7.5 ml of 5% NaOH was added. After 15 minutes, the tubes were vortexed and allowed to settle for 15 minutes. The sample tubes were then centrifuged at 2000 rpm for 15 minutes. Samples and standards were then analyzed for chromium concentration on a spectrophotometer (Gilford Response Series UV-VIS Spectrophotometer, Ciba Corning Diagnostics Corporation.), and read at 400 nm.

Statistical Analysis

Data for urine pH and blood gasses were analyzed using a repeated measures model, with horse, period and treatment as the main effects and time as the repeated variable. Least squared means over time were then calculated and tested for significance using the pdiff procedure. Significance was declared at $p < .05$ (SAS, 1985). Data for urine minerals was analyzed using the general linear models procedure with horse, period and treatment as the main effects. Least squared means were then calculated and significance was declared at $p < .05$ using the pdiff procedure (SAS, 1985). Data for fecal mineral concentrations, fecal chromium concentrations, dry matter digestibilities and mineral balances were analyzed using the general linear models procedure with horse, period and treatment as the main effects. Least squared means were then calculated and significance declared at $p < .05$ using the pdiff procedure (SAS, 1985). Mineral balances for the Low treatment do not equal intakes less excretions due to the removal of one horse from the Low treatment. Standard errors for urine and fecal excretions and mineral balances were then averaged over all treatments.

CHAPTER IV

RESULTS AND DISCUSSION

Urine pH

The effect of treatment over time on urine pH is shown graphically in Table III and Figure 1. Least square mean urine pH levels tended to decrease linearly as the cation-anion balance of the diet decreased. Mean urine pH values increased significantly ($p < .05$) between diets L, ML and MH at all measured intervals, and between diets MH and H at 6 and 10pm and again at 6am. Least squares means ranged from 5.40 to 5.86 on diet L, 6.79 to 7.30 on diet ML, 7.35 to 7.63 for diet MH and 7.52 to 8.14 on diet H. This agrees with Wall et al. (1992) who reported that exercising horses consuming a lower DCAB had lower urine pH values than those consuming the higher diets. This decrease in urinary pH may be attributed to the increase in urinary chloride excretion, which, when accompanied by a hydrogen ion, will cause a decrease in pH. When excess chloride is excreted in the urine, it is accompanied by either a hydrogen, sodium or potassium ion. When it combines with a hydrogen ion, urinary pH will decrease, as one of the routes of excretion for excess hydrogen is to combine with chloride. As this HCl would be extremely damaging to the tubule lumen, it subsequently combines with ammonia and is excreted as NH_4Cl. Another route by which hydrogen ions may be excreted to maintain the proper pH is in the form

Table III.

EFFECT OF DCAB ON URINE pH POST FEEDING

	Treatment				
Time	L	ML	MH	H	S.E.
10 AM[a]	5.60[b]	6.82[c]	7.63[d]	7.72[d]	.125
2 PM	5.40[b]	7.05[c]	7.35[d]	7.52[d]	.091
6 PM	5.56[b]	7.30[c]	7.62[d]	8.01[e]	.095
10 PM[a]	5.67[b]	6.90[c]	7.58[d]	8.14[e]	.142
2 AM	5.46[b]	6.96[c]	7.43[d]	7.63[d]	.081
6 AM	5.86[b]	6.79[c]	7.51[d]	7.90[e]	.129

[a] Indicates Feeding Time

[b,c,d,e] Values in rows with different superscripts differ ($p < .05$)

of disodium phosphate. Poultry researchers (Nesheim et al., 1984) have stated that since the pH of the urine is rarely below 4 and the pH of HCl is extremely low, excess Cl is also excreted in the form of a salt of sodium chloride or potassium chloride.

Blood Acid-Base Status

Blood pH

The effect of DCAB on arterial blood pH is shown graphically in Table IV and Figure 2. The effect of DCAB on venous blood pH is shown graphically in Table V and Figure 3. Arterial and venous blood pH values were significantly lower in those horses consuming diet L as compared to values in those consuming diets MH and H at all measured intervals, except at feeding and at 3 hours post feeding. There was a trend for both arterial and venous blood values to decrease across treatments at 3 hours post feeding. This decrease in horses consuming the ML and L diets may be attributed to a time of peak chloride absorption, while this response in those horses consuming the MH and H diets may be explained by the exchange of potassium for hydrogen into the intracellular fluid at the cellular level. In the plasma, there is a balance of ions known as the anion gap, which is defined as ([Na] + [K]) - ([Cl] + [HCO_3]). The body will attempt to maintain this ratio of ions within a specified range. As sodium and potassium concentrations in the plasma are controlled by antidiuretic hormone and aldosterone, respectively, the concentrations of chloride and bicarbonate are controlled mainly by the kidney. As the amount of chloride in the plasma increases, the body will reduce the amount of bicarbonate to maintain the anion

TABLE IV.

EFFECT OF DCAB ON ARTERIAL BLOOD pH POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	7.35^{a}	7.31^{a}	7.37^{a}	7.37^{a}	.022
1	7.36^{a}	7.37^{ab}	7.40^{b}	7.40^{b}	.009
2	7.34^{a}	7.38^{b}	7.40^{b}	7.40^{b}	.007
3	7.33^{a}	7.35^{a}	7.34^{a}	7.37^{a}	.015
4	7.34^{a}	7.38^{b}	7.40^{c}	7.41^{c}	.006
5	7.32^{a}	7.39^{b}	7.40^{b}	7.40^{b}	.005
6	7.33^{a}	7.39^{bc}	7.40^{cd}	7.42^{d}	.007
7	7.34^{a}	7.40^{b}	7.40^{b}	7.41^{b}	.004
8	7.37^{a}	7.39^{ab}	7.40^{b}	7.41^{b}	.010
9	7.35^{a}	7.40^{b}	7.42^{b}	7.41^{b}	.006
10	7.35^{a}	7.41^{b}	7.41^{b}	7.40^{b}	.006
11	7.36^{a}	7.41^{b}	7.42^{b}	7.42^{b}	.007

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE V.

EFFECT OF DCAB ON VENOUS BLOOD pH POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	7.31^{a}	7.34^{a}	7.33^{a}	7.35^{a}	.022
1	7.31^{a}	7.32^{ab}	7.35^{bc}	7.36^{c}	.009
2	7.33^{a}	7.35^{b}	7.37^{bc}	7.38^{c}	.007
3	7.32^{a}	7.33^{a}	7.34^{a}	7.35^{a}	.015
4	7.32^{a}	7.37^{bc}	7.38^{c}	7.41^{d}	.006
5	7.32^{a}	7.37^{b}	7.38^{bc}	7.40^{c}	.005
6	7.32^{a}	7.36^{b}	7.39^{c}	7.41^{c}	.007
7	7.32^{a}	7.38^{bc}	7.38^{c}	7.40^{d}	.004
8	7.33^{a}	7.37^{b}	7.37^{b}	7.39^{b}	.010
9	7.33^{a}	7.38^{b}	7.38^{b}	7.40^{b}	.006
10	7.34^{a}	7.39^{b}	7.39^{b}	7.39^{b}	.006
11	7.34^{a}	7.39^{b}	7.39^{b}	7.39^{b}	.007

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

gap. This decrease in HCO_3, and consequently $NaHCO_3$, results in a decrease in blood pH, and a metabolic acidosis. Another possible cause of the decrease in blood pH is a direct exchange for Cl and HCO_3 at the luminal epithelium in the small intestine. Many researchers believe that, as Na and Cl are absorbed together, there are actually two simultaneous exchanges occurring; one in which the absorption of Na is in exchange for a H ion, and absorption of Cl in exchange for a HCO_3 ion. Although it is not known which mechanism is responsible for the decrease in blood pH when excess Cl is consumed, it is known that both blood and urine pH decrease.

Blood pCO_2

The effect of DCAB on arterial blood pCO_2 is shown graphically in Table VI and Figure 4. The effect of DCAB on venous blood pCO_2 is shown graphically in Table VII and Figure 5. Horses tended ($p < .10$) to have lower mean arterial pCO_2 levels when fed diet L than when fed diet MH at 6 of the 12 intervals measured. When fed diet L, horses also tended ($p < .10$) to have lower mean venous pCO_2 levels than when fed diet MH at 10 of the 12 intervals measured. This decrease in arterial and venous blood pCO_2 is due to the decrease in $NaHCO_3$ in the plasma, which causes an acidemia. The body responds to this acidosis by increasing ventilation which results in reducing the amount of CO_2 in the blood, as alveolar ventilation is inversely related to pCO_2 concentrations.

Blood HCO_3

The effect of treatment on arterial blood HCO_3 is shown graphically in Table VIII and Figure 6. The effect of DCAB on venous blood HCO_3 is shown

TABLE VI.

EFFECT OF DCAB ON ARTERIAL BLOOD
pCO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	43.22^{a}	50.97^{b}	48.17^{ab}	48.37^{ab}	2.06
1	38.92^{a}	44.57^{a}	44.17^{a}	43.75^{a}	1.86
2	42.77^{a}	43.62^{a}	44.37^{a}	44.25^{a}	1.06
3	43.13^{a}	45.70^{a}	48.95^{a}	46.62^{a}	2.59
4	41.57^{a}	44.25^{a}	44.95^{a}	43.97^{a}	4.40
5	42.63^{a}	42.52^{a}	45.25^{a}	44.50^{a}	.96
6	41.28^{a}	45.12^{b}	45.60^{b}	44.25^{ab}	1.05
7	41.86^{a}	44.42^{a}	45.45^{a}	44.52^{a}	1.27
8	39.11^{a}	43.85^{b}	45.60^{b}	44.02^{b}	1.33
9	41.57^{a}	43.55^{a}	44.25^{a}	44.42^{a}	1.28
10	42.58^{a}	43.57^{a}	45.40^{a}	44.85^{a}	1.08
11	41.76^{a}	43.05^{a}	43.80^{a}	43.72^{a}	.95

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE VII.

EFFECT OF DCAB ON VENOUS BLOOD
pCO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	49.89[a]	53.87[a]	55.52[a]	53.37[a]	2.06
1	49.99[a]	55.75[a]	55.85[a]	53.70[a]	1.86
2	47.21[a]	50.67[b]	50.57[ab]	49.42[ab]	1.06
3	47.66[a]	52.07[a]	53.30[a]	52.20[a]	2.59
4	46.87[a]	48.70[a]	50.47[a]	46.45[a]	4.40
5	45.36[a]	47.55[a]	50.97[a]	48.00[a]	.96
6	46.08[a]	49.47[b]	49.32[ab]	48.12[ab]	1.05
7	46.93[a]	49.32[a]	50.05[a]	49.12[a]	1.27
8	47.25[a]	49.87[ab]	52.17[ab]	49.27[ab]	1.33
9	47.87[a]	49.00[a]	51.35[a]	48.87[a]	1.28
10	47.18[a]	49.02[a]	50.50[a]	49.50[a]	1.08
11	47.03[a]	49.10[ab]	50.57[a]	47.80[a]	.95

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE VIII.

EFFECT OF DCAB ON ARTERIAL BLOOD HCO_3 (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	24.74^{a}	26.42^{ab}	28.45^{b}	28.57^{b}	1.03
1	22.54^{a}	26.80^{b}	28.02^{b}	27.60^{b}	.57
2	23.56^{a}	26.87^{b}	27.87^{bc}	28.17^{c}	.41
3	23.54^{a}	25.70^{ab}	27.32^{b}	27.32^{b}	.69
4	22.88^{a}	26.95^{b}	28.77^{b}	28.52^{b}	.77
5	22.60^{a}	26.65^{b}	28.90^{c}	28.40^{bc}	.66
6	22.53^{a}	27.50^{b}	29.12^{b}	29.22^{b}	.65
7	23.24^{a}	28.17^{b}	28.85^{b}	29.02^{b}	.77
8	23.01^{a}	27.62^{b}	29.35^{b}	28.87^{b}	.79
9	23.55^{a}	28.02^{b}	29.40^{b}	29.30^{b}	.78
10	24.26^{a}	28.17^{b}	29.75^{b}	28.72^{b}	.86
11	24.02^{a}	28.27^{b}	28.97^{b}	28.87^{b}	.69

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE IX.

EFFECT OF DCAB ON VENOUS BLOOD HCO_3 (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	25.97^{a}	29.75^{b}	30.05^{b}	30.52^{b}	1.03
1	26.07^{a}	29.75^{b}	31.35^{b}	31.20^{b}	.57
2	25.53^{a}	29.07^{b}	30.25^{c}	30.05^{bc}	.41
3	25.34^{a}	27.82^{b}	29.50^{b}	29.45^{b}	.69
4	24.92^{a}	28.90^{b}	30.70^{b}	29.97^{b}	.77
5	23.93^{a}	28.32^{b}	31.00^{c}	30.22^{c}	.66
6	24.30^{a}	29.20^{b}	30.85^{bc}	31.20^{c}	.65
7	25.10^{a}	29.97^{b}	30.60^{b}	31.22^{b}	.77
8	25.51^{a}	29.62^{b}	31.40^{b}	30.97^{b}	.79
9	25.89^{a}	30.10^{b}	31.32^{b}	30.75^{b}	.78
10	26.16^{a}	30.40^{b}	31.65^{b}	30.70^{b}	.86
11	26.05^{a}	30.45^{b}	31.45^{b}	30.10^{b}	.69

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

graphically in Table IX and Figure 7. Horses consuming diet L had significantly lower ($p < .05$) mean arterial blood HCO_3 levels as compared to those horses consuming diets ML, MH and H at all measured intervals, except at feeding and 3 hours post feeding. Furthermore, horses consuming diet L had significantly lower mean venous HCO_3 levels as compared to those fed diets ML, MH and H at all measured intervals. This decrease in blood HCO_3 is again due to the decrease in $NaHCO_3$ in the plasma, which is the cause of a metabolic acidosis.
When excess chloride is absorbed from the gastrointestinal tract, the body responds by decreasing the amount of HCO_3 ions in the plasma to maintain the anion gap. Thus a decrease in the amount of HCO_3 occurs in the blood along with a decrease in blood pH.

Blood tCO_2

The effect of DCAB on arterial blood tCO_2 is shown graphically in Table X. The effect of DCAB on venous blood tCO_2 is shown graphically in Table XI. Total carbon dioxide, tCO_2, is the total concentration (both free and bound) of CO_2 in the blood, and is expressed in mmol/l. Arterial blood tCO_2 concentrations were lower ($p < .05$) in those horses consuming diet L as compared to those horses consuming all other diets at all measured intervals, except at feeding and three hours post feeding. Venous blood tCO_2 concentrations were lower ($p < .05$) in those horses consuming diet L as compared to those horses consuming all other diets at all measured intervals. This decrease in the total concentration of carbon dioxide in those horses consuming diet L is an indicator of an acidotic state, which is mostly due, as in pCO_2, to an increase in alveolar ventilation in response to an acidemia and the increased amounts of CO_2 in the blood.

Base Excess (BEB), Base Excess, Extracellular Fluid (BEecf), and Standard Bicarbonate (SBC)

The effect of DCAB on base excess of the arterial blood, (BEB), is shown graphically in Table XII. The effect of DCAB on base excess in the venous blood is shown graphically in Table XIII. Base excess is an indicator of the overall buffering capacity of the blood, most commonly HCO_3. Those horses consuming diet L had lower ($p < .05$) arterial BEB concentrations as compared to those consuming diets ML, MH and H at all measured intervals, except at the time of feeding. Those horses consuming diet L had lower ($p < .05$) venous BEB concentrations as compared to those consuming diets ML, MH and H at all measured intervals. This decrease in base excess indicates that the buffering capacity of the blood has been decreased, and is due to the decrease in both arterial and venous HCO_3.

Base excess, extracellular fluid, (BEecf), is also an indicator of the buffering capacity of the blood. The effect of DCAB on arterial blood BEecf is shown graphically in Table XIV. The effect of DCAB on venous blood BEecf is shown in Table XV. Those horses consuming diet L had lower ($p < .05$) arterial BEecf concentrations as compared to those consuming all other diets at all measured intervals, except at feeding. Those horses consuming diet L also had lower ($p < .05$) venous BEecf concentrations as compared to those consuming all other diets at all measured intervals. This decreased Beecf in those horses consuming the Low diet is further indication of a metabolic acidosis.

The effect of DCAB on arterial and venous standard bicarbonate,(SBC), an additional indicator of the amount of bicarbonate in the blood, is shown graphically in Tables XVI and XVII, respectively. Those horses consuming diet L had lower ($p < .05$) arterial SBC concentrations as compared to those horses

consuming diets ML, MH and H at all measured intervals, except at feeding and four hours post feeding. Those horses consuming diet L had lower ($p < .05$) venous SBC concentrations as compared to those horses consuming diets ML, MH and H at all measured intervals, except at feeding. Once again, this decrease in the buffering capacity of the blood in those horses consuming the Low diet is an indication of a metabolic acidosis.

Partial Pressure of Oxygen (pO_2) and Percent Oxygen Saturation (sO_2C)

The effect of DCAB on arterial and venous blood sO2C is shown graphically in Tables XVIII and XIX, respectively. No differences in arterial heme saturation were detected between any treatments at any of the measured intervals. Furthermore, no differences in venous blood sO_2C concentration were detected at any of the measured intervals, except at four and five hours post feeding. Treatment least squared means for arterial blood sO_2C ranged from a low of 94.37 mmol/l to 100.66 mmol/l, both values coming from those horses consuming the Low diet. The effect of DCAB on arterial and venous pO_2 is shown graphically in Tables XX and XXI. No differences in arterial blood pO_2 concentrations were detected between any treatments at any interval measured, except at five and eight hours post feeding. No differences in venous blood pO_2 concentrations were detected between any treatments at any interval. Although pO_2 and sO_2C measurements are not usually considered in the discussion of acid-base balance, these data confirm that the acid-base status of sedentary horses has little effect on the measured variables of oxygen in the arterial or venous blood.

TABLE X.

EFFECT OF DCAB ON ARTERIAL BLOOD tCO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	26.06^{a}	28.02^{ab}	29.95^{b}	30.02^{b}	1.02
1	23.72^{a}	28.15^{b}	29.37^{b}	28.95^{b}	.625
2	24.87^{a}	28.17^{b}	29.25^{bc}	29.52^{c}	.428
3	24.85^{a}	27.12^{ab}	28.80^{b}	28.77^{b}	.747
4	24.17^{a}	28.30^{b}	30.17^{b}	30.02^{b}	.783
5	23.92^{a}	27.97^{b}	30.30^{c}	29.75^{bc}	.673
6	23.76^{a}	28.92^{b}	30.52^{b}	30.55^{b}	.676
7	24.30^{a}	29.50^{b}	30.22^{b}	31.65^{b}	1.06
8	24.20^{a}	28.97^{b}	30.75^{b}	30.25^{b}	.827
9	24.86^{a}	29.35^{b}	30.77^{b}	30.67^{b}	.819
10	25.57^{a}	29.50^{b}	31.20^{b}	30.12^{b}	.897
11	25.32^{a}	29.65^{b}	30.35^{b}	30.22^{b}	.722

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XI.

EFFECT OF DCAB ON VENOUS BLOOD tCO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	27.53[a]	31.35[b]	31.75[b]	32.15[b]	1.02
1	27.58[a]	31.45[b]	33.07[b]	32.82[b]	.625
2	27.01[a]	30.62[b]	31.80[b]	31.55[b]	.428
3	26.79[a]	29.45[b]	31.12[b]	31.02[b]	.747
4	26.37[a]	30.37[b]	32.25[b]	31.40[b]	.783
5	25.28[a]	29.80[b]	32.52[c]	31.67[bc]	.673
6	25.69[a]	30.70[b]	32.35[bc]	32.67[c]	.676
7	26.13[a]	31.52[b]	32.15[b]	32.70[b]	1.06
8	26.97[a]	31.15[b]	33.00[b]	32.47[b]	.827
9	27.36[a]	31.65[b]	32.87[b]	32.22[b]	.819
10	27.60[a]	31.90[b]	33.20[b]	32.22[b]	.897
11	27.49[a]	31.97[b]	33.00[b]	31.55[b]	.722

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE XII.

EFFECT OF DCAB ON ARTERIAL BLOOD BASE EXCESS (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	-0.188^{a}	0.200^{a}	3.075^{a}	3.125^{a}	1.220
1	-1.908^{a}	1.800^{b}	3.350^{c}	3.000^{c}	.367
2	-1.575^{a}	2.100^{b}	3.175^{c}	3.550^{c}	.377
3	-1.745^{a}	0.400^{b}	1.600^{bc}	2.150^{c}	.509
4	-2.104^{a}	2.100^{b}	4.100^{c}	3.975^{bc}	.685
5	-2.679^{a}	2.200^{b}	4.100^{c}	3.700^{bc}	.586
6	-2.458^{a}	2.550^{b}	4.300^{c}	4.750^{c}	.609
7	-1.770^{a}	3.475^{b}	4.050^{b}	4.425^{b}	.647
8	-1.317^{a}	2.950^{b}	4.525^{b}	4.425^{b}	.762
9	-1.229^{a}	3.475^{b}	4.925^{b}	4.775^{b}	.690
10	-0.683^{a}	3.600^{b}	5.075^{b}	4.025^{b}	.795
11	-0.767^{a}	3.925^{b}	4.550^{b}	4.425^{b}	.653

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XIII.

EFFECT OF DCAB ON VENOUS BLOOD BASE EXCESS (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	-0.154^{a}	3.325^{ab}	3.425^{ab}	4.325^{b}	1.220
1	-0.108^{a}	3.025^{b}	4.825^{c}	5.050^{c}	.367
2	-0.175^{a}	3.200^{b}	4.525^{c}	4.550^{c}	.377
3	-0.513^{a}	1.625^{b}	3.250^{c}	3.350^{c}	.509
4	-0.804^{a}	3.375^{b}	5.075^{b}	5.100^{b}	.685
5	-1.679^{a}	3.025^{b}	5.300^{c}	5.050^{c}	.586
6	-1.392^{a}	3.575^{b}	5.425^{c}	6.100^{c}	.609
7	-0.638^{a}	4.500^{b}	5.025^{b}	5.900^{b}	.647
8	-0.183^{a}	3.975^{b}	5.525^{b}	5.600^{b}	.762
9	0.138^{a}	4.700^{b}	5.600^{b}	5.450^{b}	.690
10	0.550^{a}	5.025^{b}	6.150^{b}	5.275^{b}	.795
11	0.467^{a}	5.075^{b}	5.875^{b}	4.900^{b}	.653

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XIV.

EFFECT OF DCAB ON ARTERIAL BLOOD BASE EXCESS EXTRACELLULAR FLUID (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	-0.829^{a}	0.200^{a}	3.150^{a}	3.175^{a}	1.300
1	-2.971^{a}	1.525^{b}	3.125^{c}	2.675^{bc}	.493
2	-2.288^{a}	1.750^{b}	2.950^{bc}	3.325^{c}	.438
3	-2.438^{a}	0.025^{b}	1.575^{bc}	2.000^{c}	.634
4	-2.958^{a}	1.800^{b}	4.000^{b}	3.775^{b}	.819
5	-3.550^{a}	1.725^{b}	4.050^{c}	3.550^{bc}	.692
6	-3.396^{a}	2.350^{b}	4.250^{bc}	4.650^{c}	.710
7	-2.613^{a}	3.275^{b}	4.000^{b}	4.375^{b}	.795
8	-2.404^{a}	2.700^{b}	4.550^{b}	4.275^{b}	.872
9	-2.092^{a}	3.200^{b}	4.825^{b}	4.700^{b}	.833
10	-1.371^{a}	3.400^{b}	5.125^{b}	3.925^{b}	.936
11	-1.525^{a}	3.650^{b}	4.425^{b}	4.250^{b}	.761

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XV.

EFFECT OF DCAB ON VENOUS BLOOD BASE EXCESS EXTRACELLULAR FLUID (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	-0.296[a]	3.850[b]	4.025[b]	4.875[b]	1.300
1	-0.204[a]	3.625[b]	5.600[c]	5.675[c]	.493
2	-0.454[a]	3.500[b]	4.925[c]	4.875[c]	.438
3	-0.804[a]	1.800[b]	3.675[c]	3.800[c]	.634
4	-1.158[a]	3.500[b]	5.525[b]	5.250[b]	.819
5	-2.183[a]	3.025[b]	5.775[b]	5.275[b]	.692
6	-1.863[a]	3.800[b]	5.875[c]	6.450[c]	.710
7	-1.013[a]	4.800[b]	5.425[b]	6.350[b]	.795
8	-0.471[a]	4.250[b]	6.150[b]	6.000[b]	.872
9	-0.058[a]	5.025[b]	6.175[b]	5.825[b]	.833
10	0.329[a]	5.325[b]	6.675[b]	5.675[b]	.936
11	0.242[a]	5.425[b]	6.425[b]	5.125[b]	.761

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE XVI.

EFFECT OF DCAB ON ARTERIAL BLOOD STANDARD BICARBONATE (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	24.75^{a}	25.10^{a}	27.35^{a}	27.37^{a}	.985
1	23.55^{a}	26.35^{b}	27.57^{c}	27.25^{c}	.272
2	23.56^{a}	26.60^{b}	27.42^{bc}	27.67^{c}	.353
3	23.52^{a}	25.27^{b}	26.17^{bc}	26.62^{c}	.390
4	24.92^{a}	26.57^{ab}	28.12^{b}	28.05^{b}	.711
5	22.76^{a}	26.65^{b}	28.12^{c}	27.85^{bc}	.459
6	22.97^{a}	26.92^{b}	28.30^{bc}	28.62^{c}	.498
7	23.49^{a}	27.62^{b}	28.05^{b}	28.40^{b}	.540
8	23.91^{a}	27.27^{b}	28.47^{b}	28.37^{b}	.622
9	23.92^{a}	27.65^{b}	28.77^{b}	28.67^{b}	.543
10	24.36^{a}	27.80^{b}	28.92^{b}	28.10^{b}	.615
11	24.30^{a}	28.02^{b}	28.50^{b}	28.42^{b}	.529

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XVII.

EFFECT OF DCAB ON VENOUS BLOOD STANDARD BICARBONATE (mmol/l) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	24.15^{a}	26.75^{ab}	27.35^{a}	27.52^{b}	.985
1	23.78^{a}	26.07^{b}	27.57^{c}	27.80^{c}	.272
2	23.96^{a}	26.60^{b}	27.42^{bc}	27.80^{c}	.353
3	23.85^{a}	24.45^{b}	26.17^{bc}	26.75^{c}	.390
4	23.52^{a}	26.90^{b}	28.12^{b}	28.45^{b}	.711
5	22.96^{a}	26.67^{b}	28.12^{c}	28.20^{c}	.459
6	23.14^{a}	26.97^{b}	28.30^{bc}	28.95^{c}	.498
7	23.89^{a}	27.72^{b}	28.05^{b}	28.90^{b}	.540
8	23.98^{a}	27.35^{b}	28.47^{b}	28.60^{b}	.622
9	24.32^{a}	27.95^{b}	28.77^{b}	28.55^{b}	.543
10	24.69^{a}	28.07^{b}	28.92^{b}	28.35^{b}	.615
11	24.57^{a}	28.22^{b}	28.50^{b}	28.15^{b}	.529

^{a,b,c,d} Means in rows with different superscripts differ ($p < .05$)

TABLE XVIII.

EFFECT OF DCAB ON ARTERIAL BLOOD PERCENT SATURATED OXYGEN POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	96.32[a]	97.87[a]	98.70[a]	98.35[a]	2.59
1	100.66[a]	98.25[a]	98.15[a]	97.88[a]	5.36
2	94.37[a]	97.90[a]	98.50[a]	98.00[a]	5.34
3	97.15[a]	98.47[a]	98.15[a]	98.17[a]	2.69
4	96.28[a]	98.07[a]	98.47[a]	98.17[a]	2.75
5	96.09[a]	98.37[a]	97.97[a]	98.17[a]	2.18
6	96.50[a]	98.05[a]	98.30[a]	98.37[a]	2.14
7	97.02[a]	98.20[a]	98.15[a]	98.32[a]	2.95
8	97.67[a]	98.22[a]	97.92[a]	98.25[a]	1.94
9	97.35[a]	98.52[a]	98.62[a]	98.62[a]	2.07
10	96.66[a]	98.32[a]	98.20[a]	98.45[a]	2.01
11	97.14[a]	98.50[a]	98.20[a]	98.60[a]	2.15

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE XIX.

EFFECT OF DCAB ON VENOUS BLOOD PERCENT SATURATED OXYGEN POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	76.05[a]	71.65[a]	69.70[a]	71.52[a]	2.59
1	61.13[a]	55.07[a]	57.95[a]	61.20[a]	5.36
2	68.37[a]	69.20[a]	72.02[a]	74.52[a]	5.34
3	75.02[a]	72.80[a]	72.90[a]	69.17[a]	2.69
4	69.58[a]	74.97[ab]	70.97[a]	82.57[b]	2.75
5	76.09[ab]	76.42[a]	70.15[b]	75.40[ab]	2.18
6	74.70[a]	71.32[a]	75.77[a]	73.22[a]	2.14
7	79.65[a]	73.35[a]	77.15[a]	75.70[a]	2.95
8	69.41[a]	75.27[a]	72.65[a]	73.60[a]	1.94
9	73.15[a]	74.77[a]	74.45[a]	76.25[a]	2.07
10	73.69[a]	72.10[a]	74.02[a]	73.42[a]	2.01
11	73.34[a]	74.80[a]	73.95[a]	77.05[a]	2.15

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE XX.

EFFECT OF DCAB ON ARTERIAL BLOOD
pO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	116.66[a]	114.00[a]	148.75[a]	144.00[a]	14.34
1	115.45[a]	113.00[a]	108.75[a]	104.75[a]	4.39
2	108.91[a]	104.50[a]	120.50[a]	105.50[a]	7.19
3	108.25[a]	121.50[a]	116.00[a]	112.75[a]	5.23
4	118.70[a]	108.25[a]	115.00[a]	108.00[a]	4.21
5	119.87[a]	114.25[a]	104.00[b]	109.25[ab]	3.37
6	118.95[a]	108.25[a]	111.00[a]	110.75[a]	3.50
7	115.00[a]	109.25[a]	108.50[a]	109.75[a]	5.76
8	118.25[a]	111.00[ab]	104.00[b]	109.50[ab]	4.00
9	114.08[a]	120.75[a]	117.75[a]	121.00[a]	6.39
10	115.45[a]	111.00[a]	107.50[a]	119.50[a]	5.44
11	114.00[a]	117.25[a]	108.50[a]	120.50[a]	6.03

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

TABLE XXI.

EFFECT OF DCAB ON VENOUS BLOOD
pO_2 (mmHg) POST FEEDING.

	Treatment				
Hour	**L**	**ML**	**MH**	**H**	**S.E.**
0	37.00[a]	40.75[a]	40.00[a]	38.00[a]	14.34
1	38.12[a]	33.25[a]	32.75[a]	33.75[a]	4.39
2	39.25[a]	40.25[a]	41.50[a]	51.75[a]	7.19
3	42.91[a]	42.00[a]	42.50[a]	38.25[a]	5.23
4	39.37[a]	42.00[a]	38.75[a]	50.75[a]	4.21
5	44.20[a]	42.75[a]	38.50[a]	41.50[a]	3.37
6	43.62[a]	39.25[a]	42.25[a]	39.25[a]	3.50
7	44.00[a]	40.00[a]	43.00[a]	41.50[a]	5.76
8	37.91[a]	42.50[a]	39.75[a]	39.75[a]	4.00
9	43.08[a]	40.75[a]	41.25[a]	42.00[a]	6.39
10	42.45[a]	39.00[a]	40.75[a]	40.00[a]	5.44
11	38.33[a]	41.25[a]	40.25[a]	53.25[a]	6.03

[a,b,c,d] Means in rows with different superscripts differ ($p < .05$)

Dry Matter Digestibility

The effect of DCAB on dry matter digestibility is shown graphically in Table XXII. Dry matter digestibility was calculated by dividing the grams of DM fecal output by the grams of DM intake per day. Dry matter digestibility percentages ranged from a low of 57.91% for treatment L to a high of 62.09% for treatment MH. No significant differences were observed for dry matter digestibility among treatments. This disagrees with Nelson and coworkers (1981) who reported a decrease in dry matter digestibility in chicks fed a higher cation-anion ratio. However, these researchers manipulated the cation-anion ratio with the addition of calcium and phosphorus. These data also disagree with that of Yen et al. (1981) who showed a decreased feed intake, feed efficiency and weight gain in barrows fed a diet with 4% cacium chloride added, and also with Haydon and West (1990) who reported a linear relationship between DCAB and apparent ileal digestibility of energy, dry matter, N, and amino acids (with the exception of alanine and methionine) in diets with DCAB's of -50 to 400 meq/kg of diet dry matter. However, these researchers noted that nutrient and amino acid digestibilities were similar when measured over the entire tract.

Sodium Balance

After supplementation of the High diet with potassium citrate and sodium bicarbonate, diet H was determined to contain 1.25% potassium and 0.40% sodium, increasing sodium intake to 33.13 g/d for the High diet as compared to 19.21, 22.94 and 26.93 g/d for the L, ML and MH diets, respectively. The effect of DCAB on sodium balance is shown in Table XXIII and Figure 8. Horses consuming diets L and H excreted more sodium in the urine than those horses

TABLE XXII

THE EFFECT OF DIETARY CATION-ANION BALANCE ON DRY MATTER DIGESTIBILITY IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
DM Digestibility %	57.91[a]	58.42[a]	62.09[a]	59.95[a]	1.99

[a,b] Means in rows with different superscripts differ ($p < .05$)

consuming diets ML and MH ($p < .05$), with least square means of 13.51 and 11.53 g/d for the L and H diets, respectively, and 6.20 and 5.32 g/d for the ML and MH diets, respectively. In addition, those horses consuming diet L excreted less sodium in the feces ($p < .05$) than those consuming diets ML, MH and H, with those horses consuming the Low diet excreting 5.66 g/d in the feces versus 13.03, 13.71 and 16.56 g/d for treatments ML, MH and H. The increased daily intake of sodium in those horses consuming the High diet resulted in an increased urinary excretion of sodium as compared to those horses consuming the MH and ML diets, but was not statistically different than the urinary excretion of sodium in those horses consuming the Low diet. One explanation for the increased urinary sodium excretion in those horses consuming the highly anionic diet (diet L) is that those horses consuming the Low diet also excreted a significantly higher amount of chloride ions in the urine, (Table 15, Figure 10). These chloride ions must combine with another ion to be excreted, and since combining with hydrogen ions results in an acidity that would be very destructive to the tubule lumen, these chloride ions most often are excreted in the form of Na or K salts (Nesheim et al., 1984).

These findings are in partial agreement with that of Schryver and coworkers (1987) who demonstrated that urinary excretion was the primary pathway for sodium loss in sedentary horses consuming 1, 3 and 5% sodium chloride. The author noted that sodium intake was directly related to urinary sodium excretion but had no effect on fecal excretion, intestinal absorption or retention of sodium. Sodium balance of those horses consuming diets ML (3.71 g/d), MH (7.90 g/d) and H (5.04 g/d) were not statistically different, however those horses consuming the Low diet had a balance of -0.76 g/d, and this was significantly lower ($p < .05$) than the balance for those horses consuming diet MH (7.90 g/d).

TABLE XXIII

THE EFFECT OF DIETARY CATION-ANION BALANCE ON SODIUM BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	19.21	22.94	26.93	33.13	
Urine, g/d	13.51[b]	6.20[a]	5.32[a]	11.53[b]	.91
Fecal, g/d	5.66[a]	13.03[b]	13.71[b]	16.56[b]	1.33
Balance, g/d	-0.76[a]	3.71[ab]	7.90[b]	5.04[ab]	1.96

[a,b] Means in rows with different superscripts differ ($p < .05$)

These findings are in partial agreement with Wall et al. (1992) who observed a higher urinary sodium excretion in exercising horses consuming the highest DCAB as compared to the other treatments. However, those researchers did not observe higher sodium excretions in those horses consuming the lowest DCAB diet. These findings suggest that horses consuming a highly anionic diet may be in a negative sodium balance due to the increased amount of sodium lost in the urine.

Potassium Balance

Potassium was one of the cations used to manipulate the DCAB, and was supplemented in the form of potassium citrate at 1.00% of the diet along with .40% sodium bicarbonate (Table I). This supplementation resulted in a total dietary concentration of potassium in the High diet of 1.25%, as compared to a concentration of 0.86% in the Low, Medium Low and Medium High diets. Therefore, daily potassium intake for those horses consuming the High diet was increased to 104.14 g/d as compared to 73.38, 71.17 and 71.17 g/d for diets L, ML and MH, respectively. The effect of DCAB on potassium balance is shown in Table XXIV and Figure 9. Those horses consuming the High diet had higher ($p < .05$) concentrations of potassium in the urine (68.47 g/d) as compared to all other treatments (L = 34.85, ML = 46.55 and MH = 41.52 g/d). Furthermore, those horses consuming diet L had higher ($p < .05$) concentrations of potassium in the feces (24.05 g/d) as compared to all other diets (ML = 18.19, MH = 16.98 and H = 15.28 g/d). Potassium balances were similar among treatments L (12.27 g/d), ML (6.42 g/d) and MH (12.66 g/d). However, those horses consuming diet H had a higher ($p < .05$) potassium balance (20.47 g/d) than those horses consuming the ML diet (6.42 g/d). The NRC (1989) lists the potassium requirement for horses at maintenence at 1.52 g/Mcal of DE, therefore a 500 kg

TABLE XXIV

THE EFFECT OF DIETARY CATION-ANION BALANCE ON POTASSIUM BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	73.38	71.17	71.17	104.14	
Urine, g/d	34.85[a]	46.55[a]	41.52[a]	68.48[b]	3.18
Fecal, g/d	24.05[b]	18.19[a]	16.98[a]	15.28[a]	.948
Balance, g/d	12.27[ab]	6.42[a]	12.66[ab]	20.47[b]	2.36

[a,b] Means in rows with different superscripts differ ($p < .05$)

(1100 lb) horse would need 25 g/d of dietary potassium. The horses in this trial received dietary potassium well above the minimum requirements, which is common in most rations fed today.

These findings are in agreement with Wall and coworkers (1992) who reported that exercising horses consuming the highest DCAB had higher urinary excretion of potassium than those horses consuming the other diets.

Chloride Balance

Chloride was the only anion used in this experiment to manipulate the DCAB. The ML diet was supplemented with .50% calcium chloride, while the Low diet was supplemented with .50% calcium chloride and .50% ammonium chloride (Table I). This supplementation resulted in a daily intake of 89.37 and 60.76 g/d for diet L and ML, respectively, as compared to 33.29 and 31.80 g/d for diets MH and H. The effect of DCAB on chloride balance is shown in Table XXV and Figure 10. No differences in fecal excretion of chloride were observed across treatments. Decreasing the DCAB resulted in higher ($p < .05$) urinary excretions of chloride in both diet L (70.59 g/d) and diet ML (57.54 g/d) as compared to both diet MH (31.34 g/d) and H (31.44 g/d). Apparently, the increase in urinary chloride excretion in those horses consuming diets ML and L was enough to offset the increased intake of chloride, as daily chloride balance was similar across all treatments. However, in this trial only those horses consuming diet L had a positive daily chloride balance (13.16 g/d).

These results agree with other data demonsrating an increased daily urinary excretion of chloride in horses consuming a lower DCAB (Topliff et al., 1989; Wall et al., 1992). However, these results disagree with those of Schryver and others (1987) who reported that dietary chloride was completely absorbed in

TABLE XXV

THE EFFECT OF DIETARY CATION-ANION BALANCE ON CHLORIDE BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	89.37	60.76	33.29	31.79	
Urine, g/d	70.60[b]	57.54[b]	31.34[a]	31.44[a]	5.79
Fecal, g/d	4.60[a]	5.58[a]	5.78[a]	3.52[a]	.686
Balance, g/d	13.16[a]	-2.35[a]	-3.82[a]	-3.16[a]	4.70

[a,b] Means in rows with different superscripts differ ($p < .05$)

sedentary horses consuming diets with 1, 3 and 5% sodium chloride, and that urinary excretion was the sole pathway for elimination of chloride from the body.

The NRC (1989) states that chloride requirements are presumed to be met when the sodium requirements are met with sodium chloride. In 1989, Young and others fed approximately 1.5 times more chloride than sodium to exercised miniature horses and still experienced a chloride deficiency. In the present study, those horses consuming diets ML, MH and H all had a negative daily chloride balance (-2.35, -3.82 and -3.16 g/d, respectively). From the data in this study, we may suggest that diets with a low DCAB manipulated with the addition of chloride results in an increased daily chloride balance. Furthermore, this increased amount of chloride retained in the body has a significant effect on blood and urine pH, along with a possible deleterious role in calcium, phosphorus and sodium balance in sedentary horses.

Magnesium Balance

Magnesium has been previously implicated as having a possible role in the DCAB equation in dairy cattle. Therefore, in the present study dietary magnesium intakes were held constant (13.03, 13.14, 12.14 and 12.64 g/d for diets L, ML, MH and H, respectively) across treatments. The effect of DCAB on magnesium balance is shown in Table XXVI and Figure 11. No differences in daily urinary magnesium excretions were observed, with excretions ranging from 6.02 to 6.74 g/d. Daily fecal magnesium excretion increased as the DCAB decreased. Those horses consuming diet L excreted more ($p < .05$) magnesium in the feces (7.95 g/d) than those consuming diets MH (6.76 g/d) and H (6.68 g/d), while those consuming diet ML (7.42 g/d) excreted more ($p < .05$) magnesium in the feces than those consuming diet H (6.68 g/d). However, these urinary and fecal

TABLE XXVI

THE EFFECT OF DIETARY CATION-ANION BALANCE ON MAGNESIUM BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	13.03	13.14	12.14	12.64	
Urine, g/d	6.02[a]	6.74[a]	6.47[a]	6.44[a]	.926
Fecal, g/d	7.95[c]	7.42[bc]	6.76[ab]	6.68[a]	.212
Balance, g/d	-1.31[a]	-1.01[a]	-1.08[a]	-0.48[a]	0.78

[a,b] Means in rows with different superscripts differ ($p < .05$)

excretions resulted in no differences in daily magnesium balance across treatments, although magnesium balance was slightly negative for all treatments.

These data are in agreement with Wall and others (1992) who reported no differences in urinary magnesium excretion in exercising horses consuming diets varying in DCAB. The NRC (1989) suggests a magnesium intake of .46 g/Mcal DE to meet the horse's requirement. Therefore, the horses in this trial would require approximately 10 g/d (due to varying intakes between horses). All diets used in the present trial should have been sufficient in meeting the magnesium requirement, however, these data suggest that this value may be inadequate independent of DCAB. Further research is needed to accurately quantify the magnesium requirements of sedentary horses.

Phosphorus Balance

It was attempted by these researchers to hold the intake of phosphorus constant across all treatments. However, due to possible variation concentration of phosphorus in the feedstuffs used, or due to sampling error, the actual phosphorus intakes in g/d were 25.22 for diet L, 27.96 for diet ML, 22.97 for diet MH and 27.46 for diet H. The effect of DCAB on phosphorus balance is shown in Table XXVII and Figure 12. No differences were observed in daily urinary excretion of phosphorus, with urinary excretions ranging from 0.128 g/d for diet ML to 0.156 g/d for diet H. Daily fecal excretion of phosphorus was similar among treatments L (19.05 g/d), ML (19.10 g/d) and MH (18.97 g/d), however those horses consuming diet H had lower ($p < .05$) daily fecal excretion of phosphorus as compared to horses consuming the L, ML and MH diets. The low renal excretion values may be due to the possibility that even in the frozen state, phosphorus may be changed from the organic to the inorganic form.

TABLE XXVII

THE EFFECT OF DIETARY CATION-ANION BALANCE ON PHOSPHORUS BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	25.22	27.96	22.97	27.46	
Urine, g/d	0.148[a]	0.128[a]	0.135[a]	0.156[a]	.016
Fecal, g/d	19.05[b]	19.10[b]	18.97[b]	17.32[a]	.281
Balance, g/d	5.31[b]	8.73[c]	3.86[a]	9.98[d]	.270

[a,b] Means in rows with different superscripts differ ($p < .05$)

Although there was a significant difference in phosphorus balance between all treatments ($p < .05$), phosphorus balance tended to reflect phosphorus intake across all treatments. These data agree with that of Wall and others (1992) who observed no differences in daily urinary phosphorus excretions in horses consuming a varying DCAB. Furthermore, the urinary excretions observed in the present study were similar to those observed by Wall and others (1992). The NRC (1989) suggests a phosphorus requirement of .87 g/Mcal DE. Therefore, the horses in this study required approximately 18.5 g/d. Each of the diets used in this study appear to have supplied adequate phosphorus, as all daily balances across treatments were positive.

Calcium Balance

The effect of DCAB on calcium balance is shown in Table XXVIII and Figure 13. Diets were formulated to have equivalent amounts of calcium in each treatment. However, due to possible variation in feedstuffs or possible sampling error, calcium intake in grams/day was 44.24 for diet L, 44.91 for diet ML, 41.42 for diet MH and 48.41 for diet H. Some differences were observed regarding calcium absorption and excretion as compared to the other minerals studied. Daily fecal excretion of calcium was similar in those horses consuming diets L, ML and H (15.35, 15.76 and 15.92 g/d, respectively), however, those horses consuming diet MH excreted more ($p < .05$) calcium in the feces (19.11 g/d) as compared to all other diets. Daily urinary excretion of calcium increased significantly ($p < .05$) between all treatments as the DCAB was decreased. Daily urinary calcium excretions ranged from 3.99 g/d for diet H to 39.81 g/d for diet L. These urinary and fecal excretions of calcium resulted in a marked difference between daily calcium balances between treatments. Daily calcium balance

TABLE XXVIII

THE EFFECT OF DIETARY CATION-ANION BALANCE ON CALCIUM BALANCE IN SEDENTARY HORSES

	Treatment				
	L	ML	MH	H	S.E.
Intake, g/d	44.24	44.91	41.42	48.41	
Urine, g/d	39.82^{d}	31.80^{c}	13.99^{b}	3.99^{a}	1.27
Fecal, g/d	15.35^{a}	15.76^{a}	19.11^{b}	15.92^{a}	.503
Balance, g/d	-12.20^{a}	-2.65^{b}	8.31^{c}	28.51^{d}	1.02

^{a,b} Means in rows with different superscripts differ ($p < .05$)

decreased significantly ($p < .05$) between all treatments as the DCAB was decreased, with balances of -12.20, -2.65, 8.31 and 28.51 g/d for diets L, ML, MH and H, respectively.

These data on urinary excretion of calcium agree with other data in horses (Wall et al., 1992; Topliff et al., 1989), rats (Barzel and Jowsey, 1989; Newell and Beauchene, 1975; Petito and Evans, 1984; Goulding and Campbell, 1984), rabbits (Thacker, 1959), and dairy cattle (Tucker et al., 1988) that consume diets with a lower DCAB.

In 1991, Goff and others demonstrated that parathyroid hormone has a more dramatic effect on renal production of 1,25 dihydroxyvitamin D_3 in dairy cows fed highly anionic diets, resulting in increased intestinal calcium absorption. Furthermore, osteoclastic bone resorption was more responsive to parathyroid hormone as plasma hydroxyproline concentration was higher in those cows fed the low DCAB diet. It has also been suggested that renal tubular reabsorption of calcium may be inhibited by the acidotic state and low pH induced by the lower DCAB diets (Beck and Webster, 1976).

The NRC (1989) suggests that the calcium requirement is 1.22 g/Mcal DE/d. The horses in the present trial would therefore have required approximately 26 g/d of dietary calcium. The calcium intake in this trial was purposely exceeded so that these horses would not be predisposed to a daily negative calcium balance. However, these data demonstrate that as the DCAB is lowered, daily calcium balance decreases, and horses consuming the ML and L diets are in a negative calcium balance. If this condition were prolonged, these animals could be predisposed to an osteoporotic weakening of the skeletal system that has been demonstrated in poultry (Leach and Neshium, 1965, 1972; Hamilton and Thompson, 1980; Mongin, 1981; Halley et al., 1987; Sauveur and

Mongin, 1978; Hurwitz et al., 1973), rabbits (Thacker, 1959), and dairy cattle (Tucker et al., 1988) that consume diets with a lower DCAB.

CHAPTER V

SUMMARY AND CONCLUSIONS

In summary, these results indicate a direct correlation between dietary cation-anion balance and acid-base status in horses, as evidenced by the decrease in arterial and venous blood pH and bicarbonate concentrations, as well as the urine pH as the cation-anion balance of the diet decreases. Furthermore, these results provide critical information regarding the relationship between DCAB and mineral balance in sedentary horses. These results suggest that the NRC (1989) recommendations for magnesium may be marginal. We may conclude that the horses consuming diets with a lower (less than 230 meq/kg diet DM) DCAB were in a net negative calcium balance, as well as a lowered sodium balance.

It is clear that both the absolute amounts as well as the ratios of minerals in the diets of horses should be under strict control. It is commonplace in the horse industry today, however, to see completely balanced rations supplemented with feed additives and vitamin and mineral mixtures that may alter these critical ratios of the diet. The feeding of diets with a lowered DCAB may have far reaching effects on many classes of horses as well as on different production stages. It may be possible to increase milk yield by feeding a higher DCAB to lactating mares, as has been shown in dairy cattle. It may also be possible to decrease the incidence of many developmental orthopedic disease in the growing horse by improving the calcium status and balance of these young horses. Furthermore,

the feeding of higher cation-anion balances to heavily exercised horses, such as those on the track, could result in a decrease in the amount of injuries and breakdowns which are possibly due to lowered calcium retention and decreased bone mineralization.

Further research is needed to determine the effects of DCAB in the young growing horse, as they would be particularly susceptible to these alterations in mineral balance, particularly a decreased calcium balance, which may affect proper bone and skeletal formation.

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APPENDIX

FIGURES

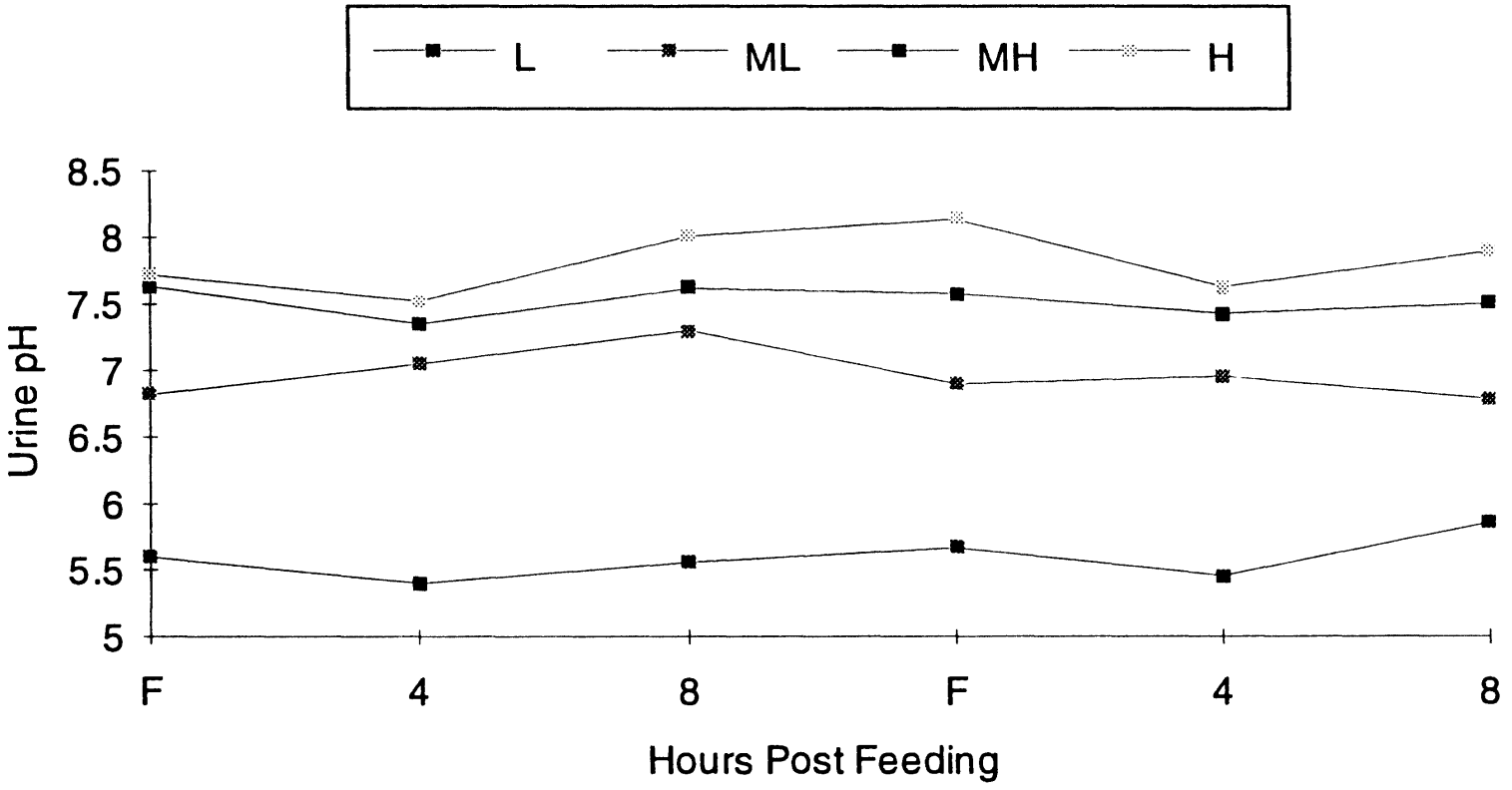

Figure 1. EFFECT OF DIETARY CATION-ANION BALANCE ON URINE pH POST FEEDING

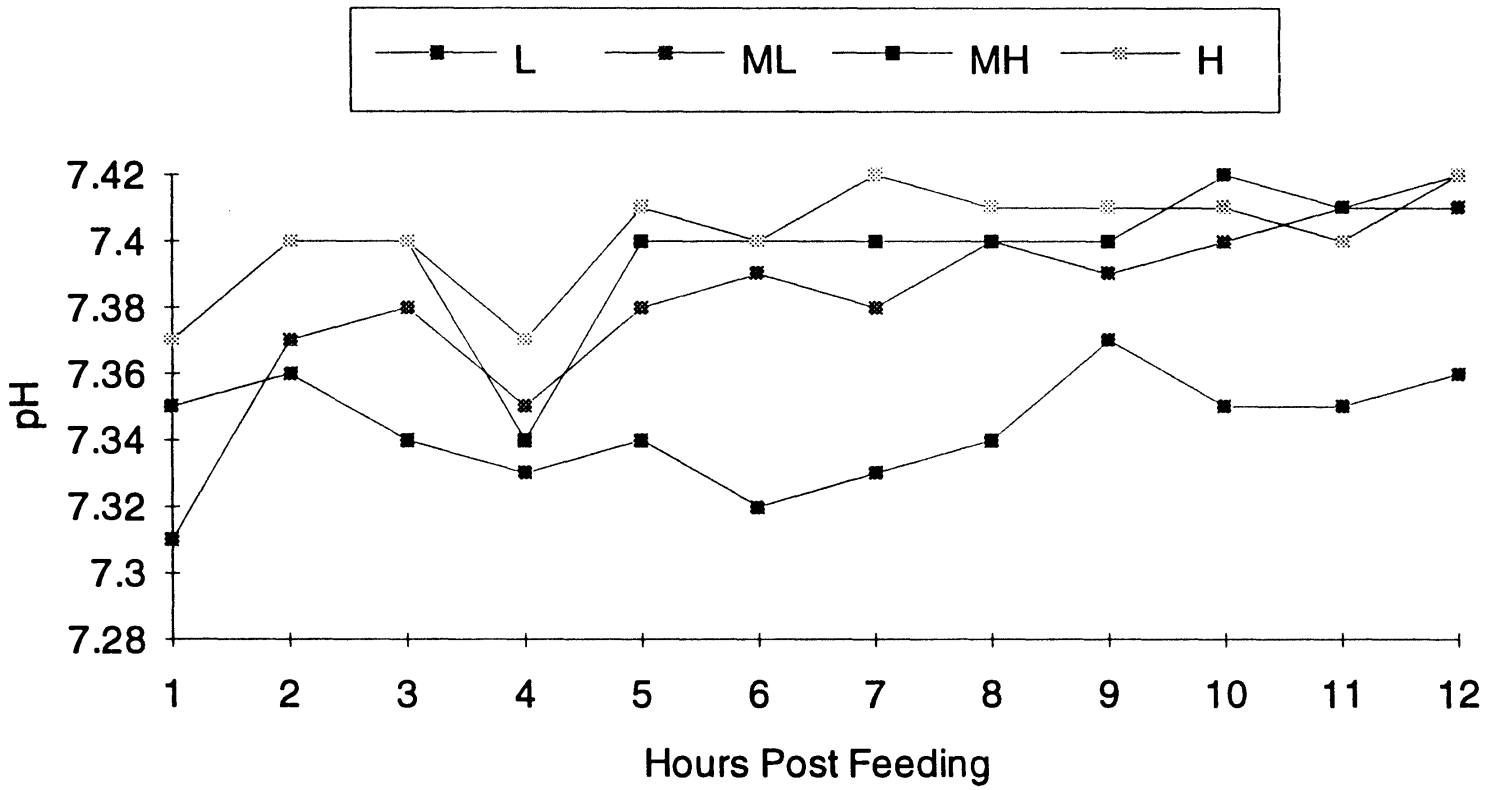

Figure 2. EFFECT OF DIETARY CATION-ANION BALANCE ON ARTERIAL BLOOD pH POST FEEDING

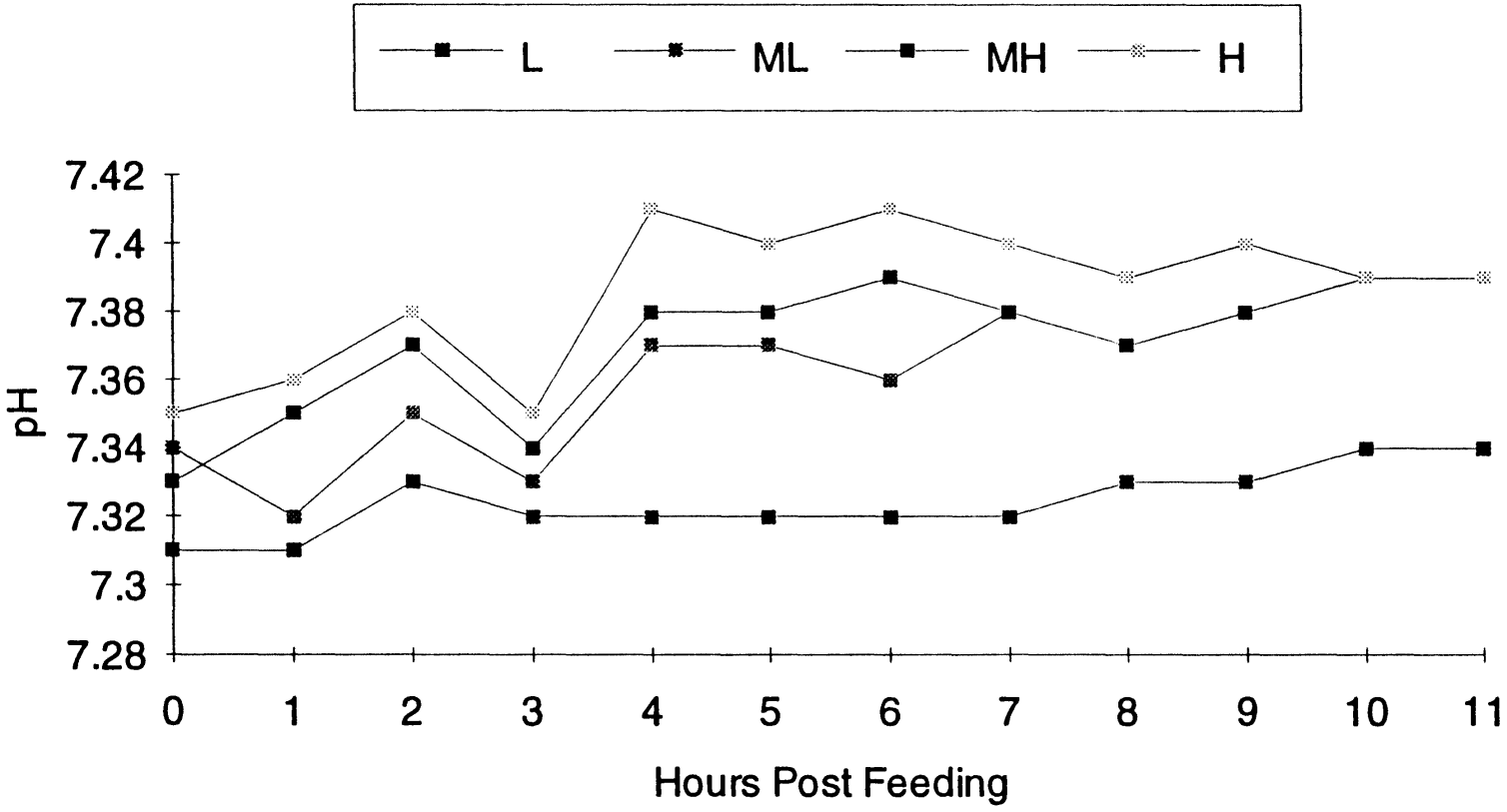

Figure 3. EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pH POST FEEDING

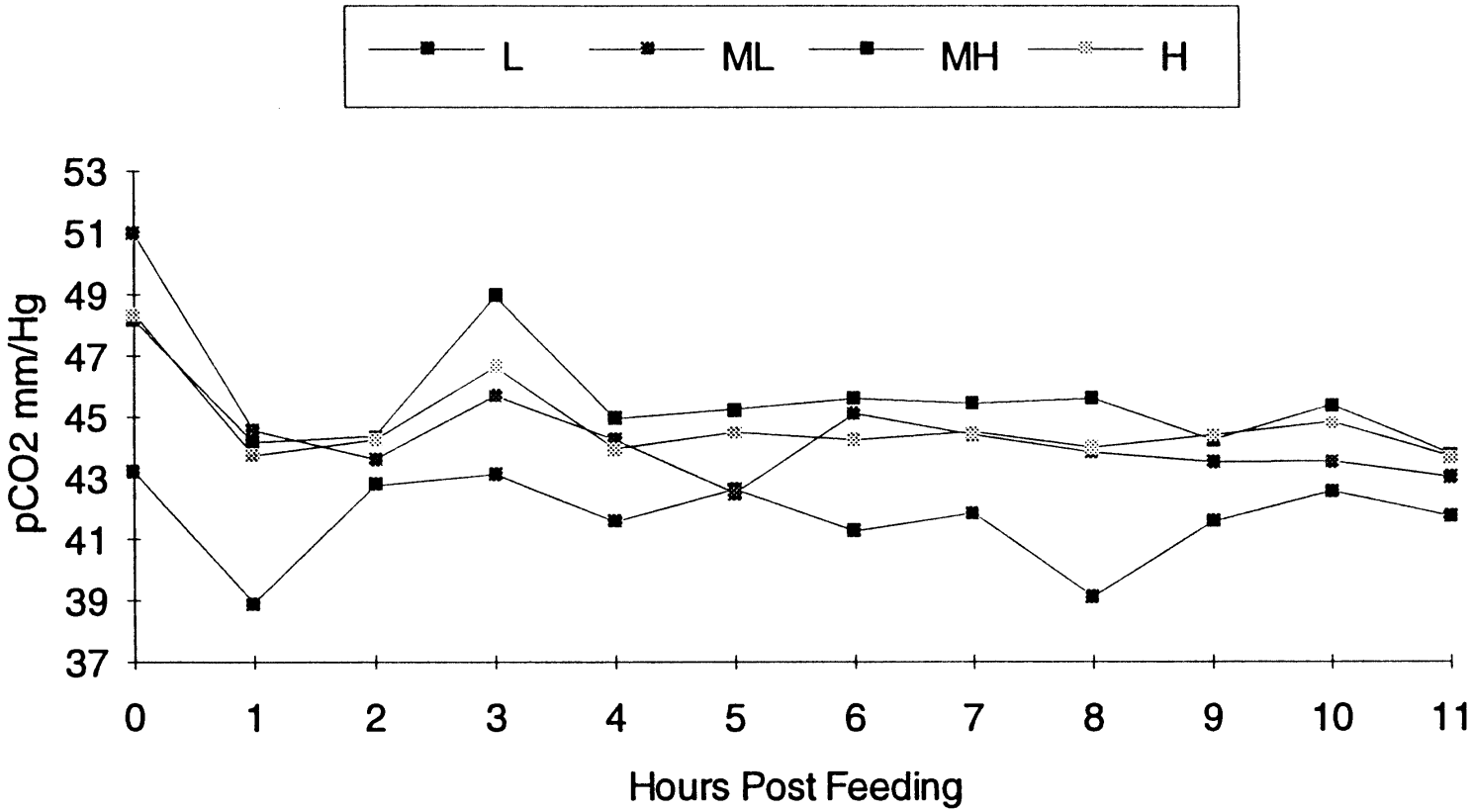

Figure 4. EFFECT OF DIETARY CATION-ANION BALANCE ON ARTERIAL BLOOD pCO_2 POST FEEDING

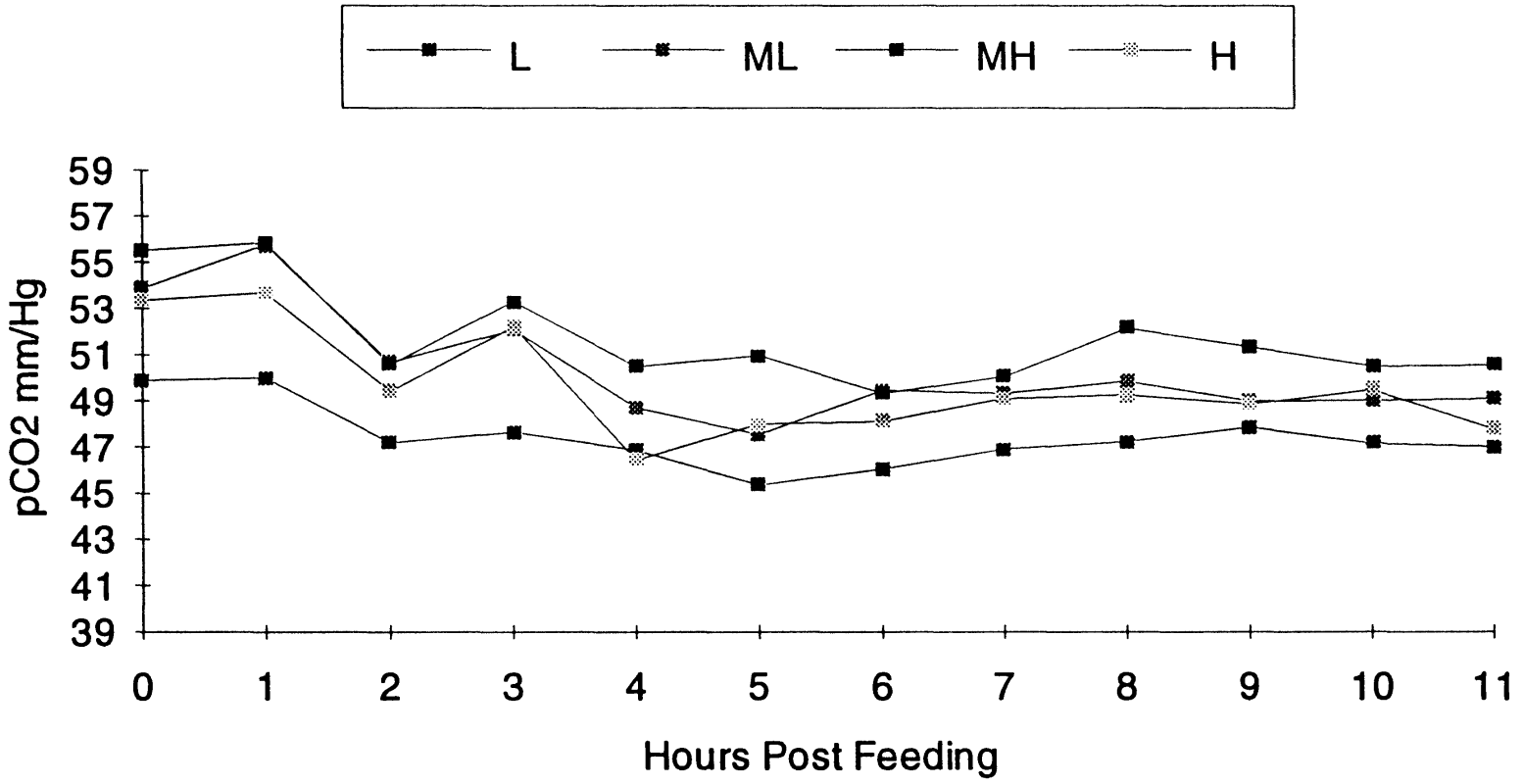

Figure 5. EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pCO_2 POST FEEDING

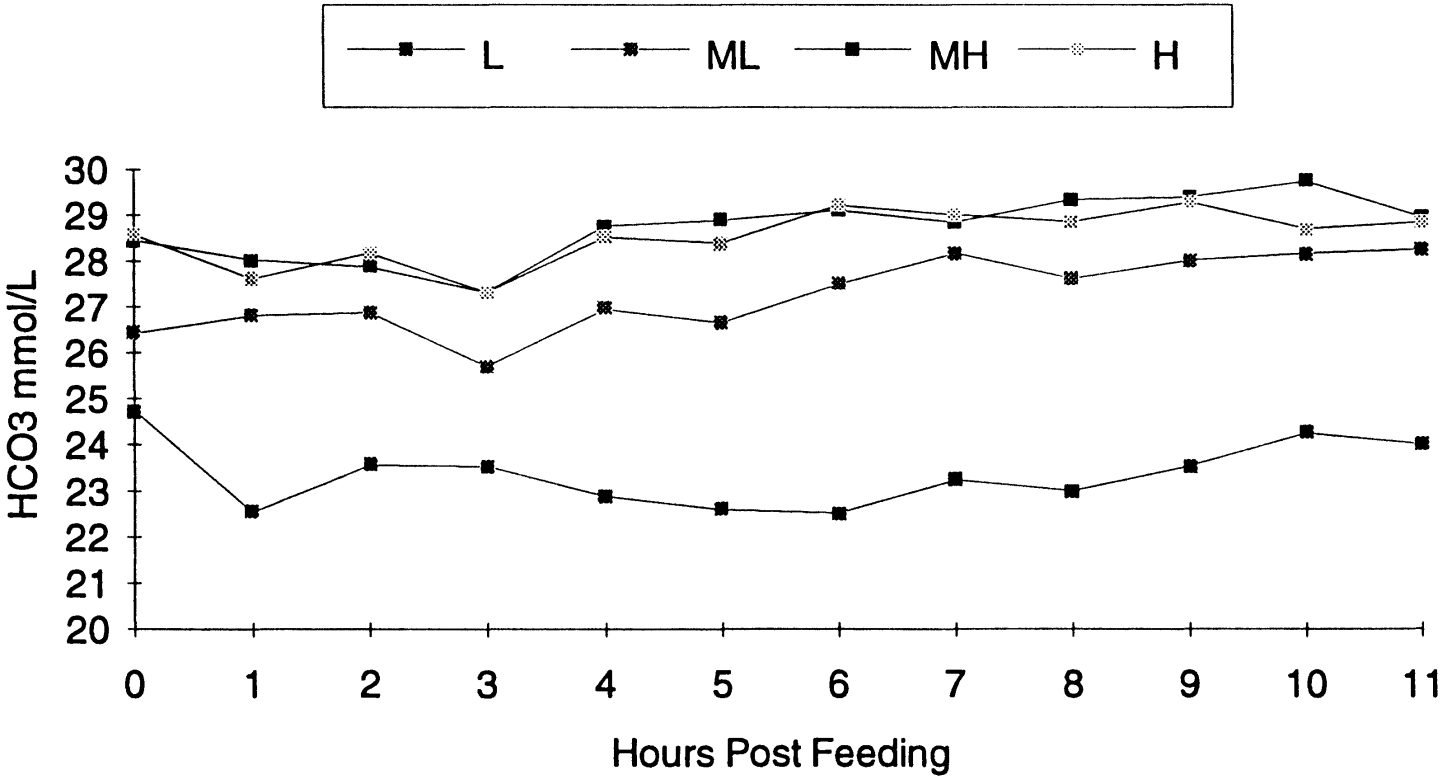

Figure 6. EFFECT OF DIETARY CATION-ANION BALANCE ON ARTERIAL BLOOD HCO_3 POST FEEDING

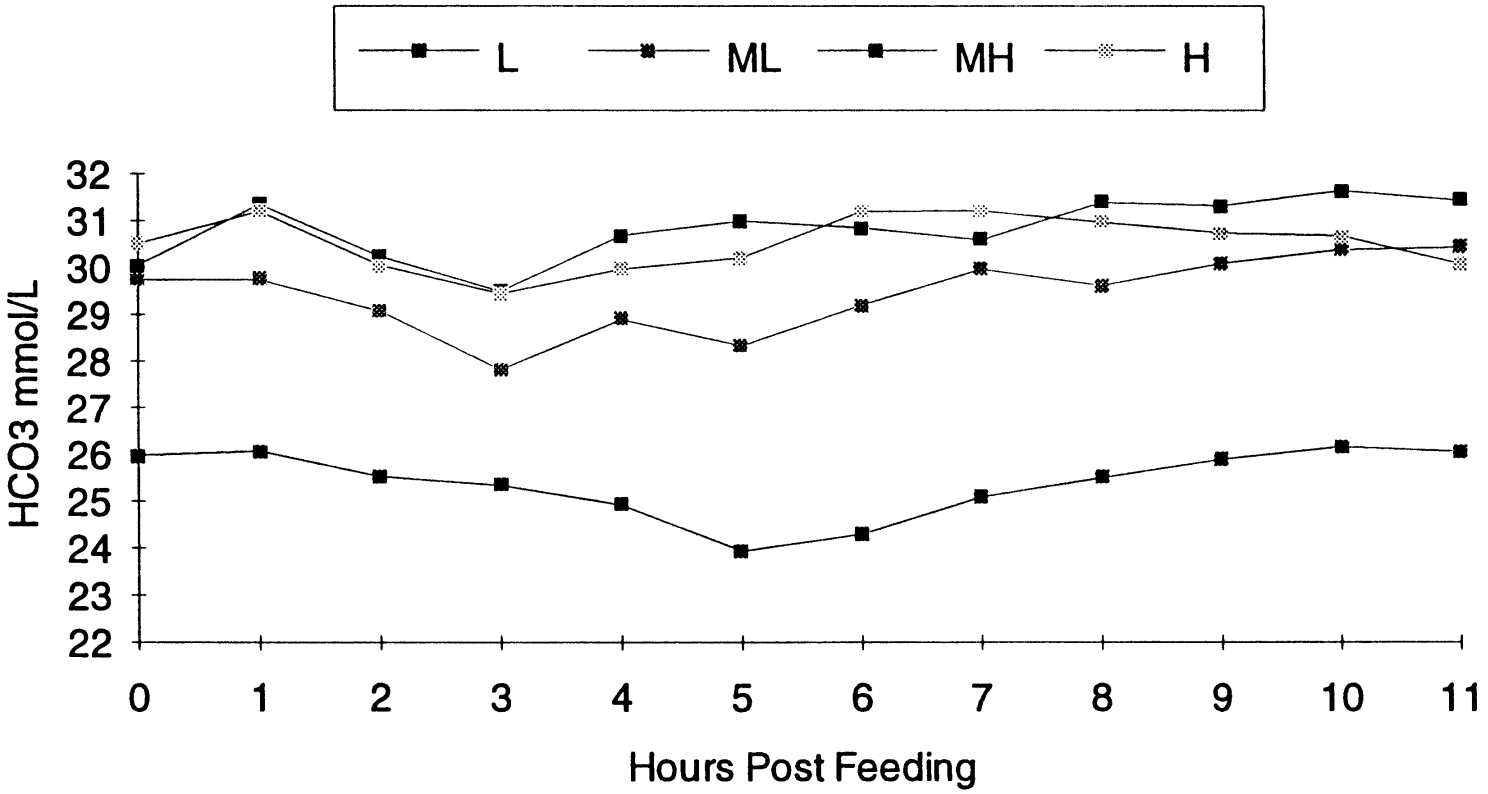

Figure 7. EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD HCO_3 POST FEEDING

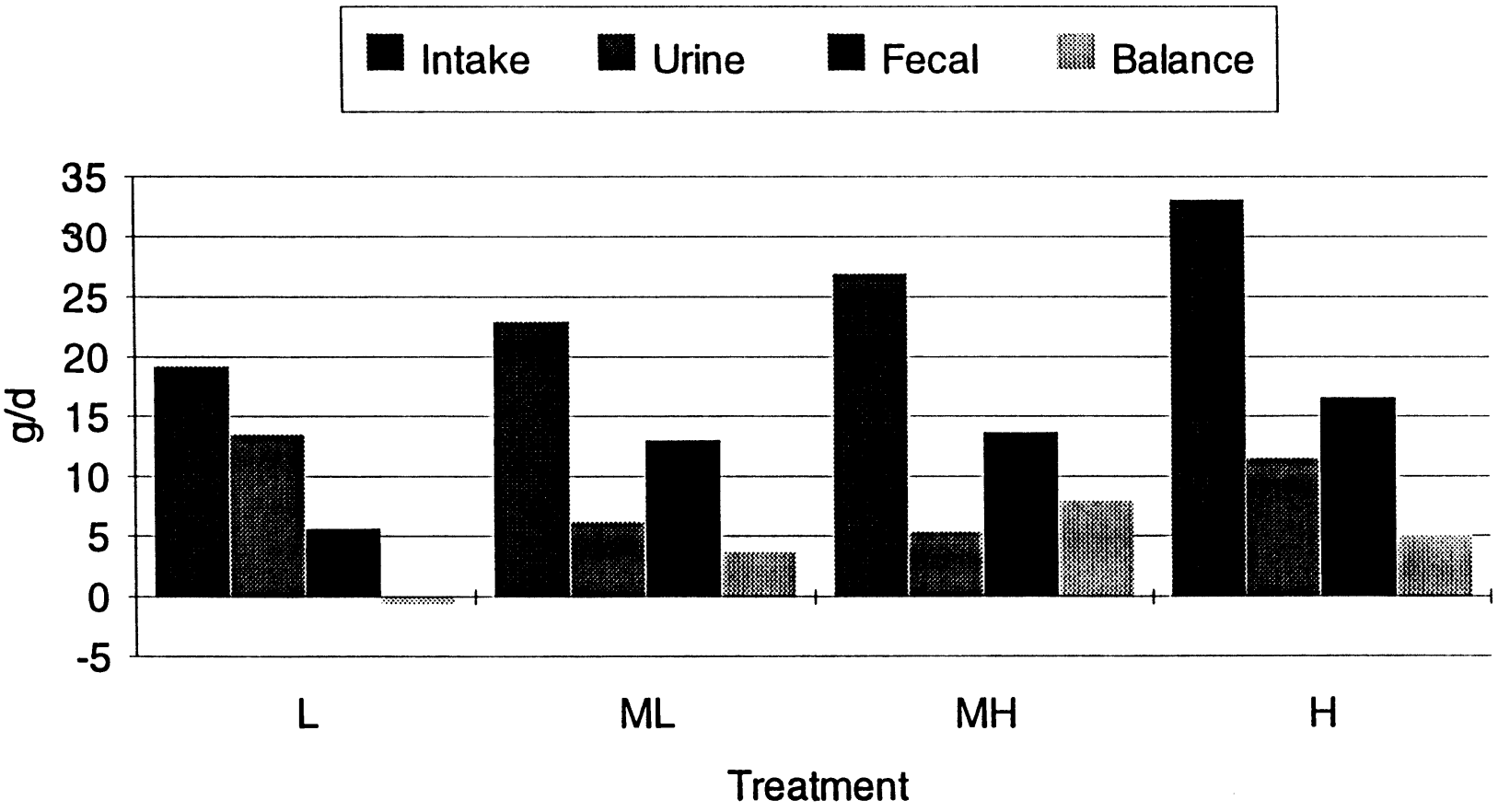

FIGURE 8. THE EFFECT OF DCAB ON SODIUM BALANCE IN SEDENTARY HORSES.

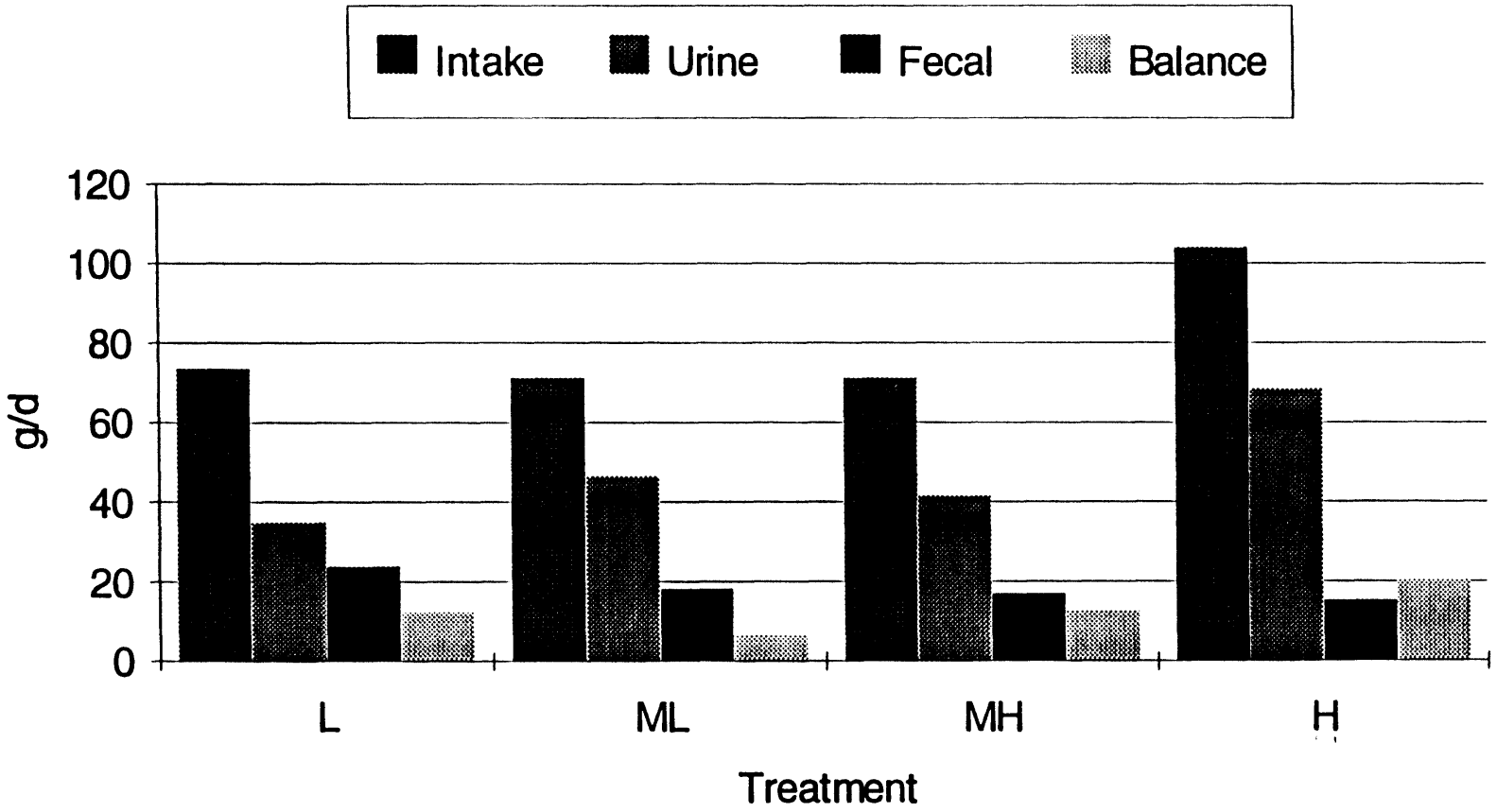

FIGURE 9. THE EFFECT OF DCAB ON POTASSIUM BALANCE IN SEDENTARY HORSES.

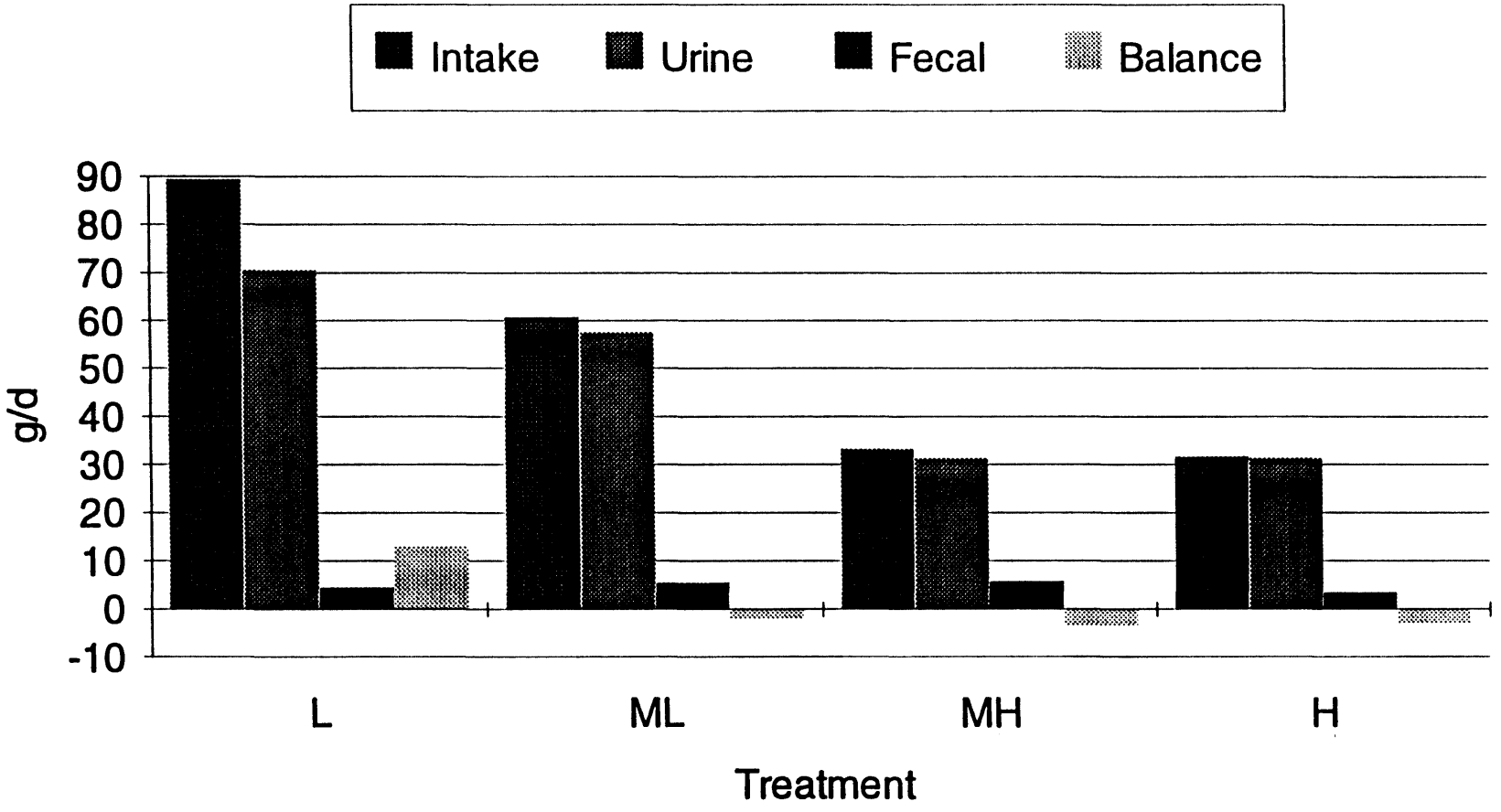

FIGURE 10. THE EFFECT OF DCAB ON CHLORIDE BALANCE SEDENTARY HORSES.

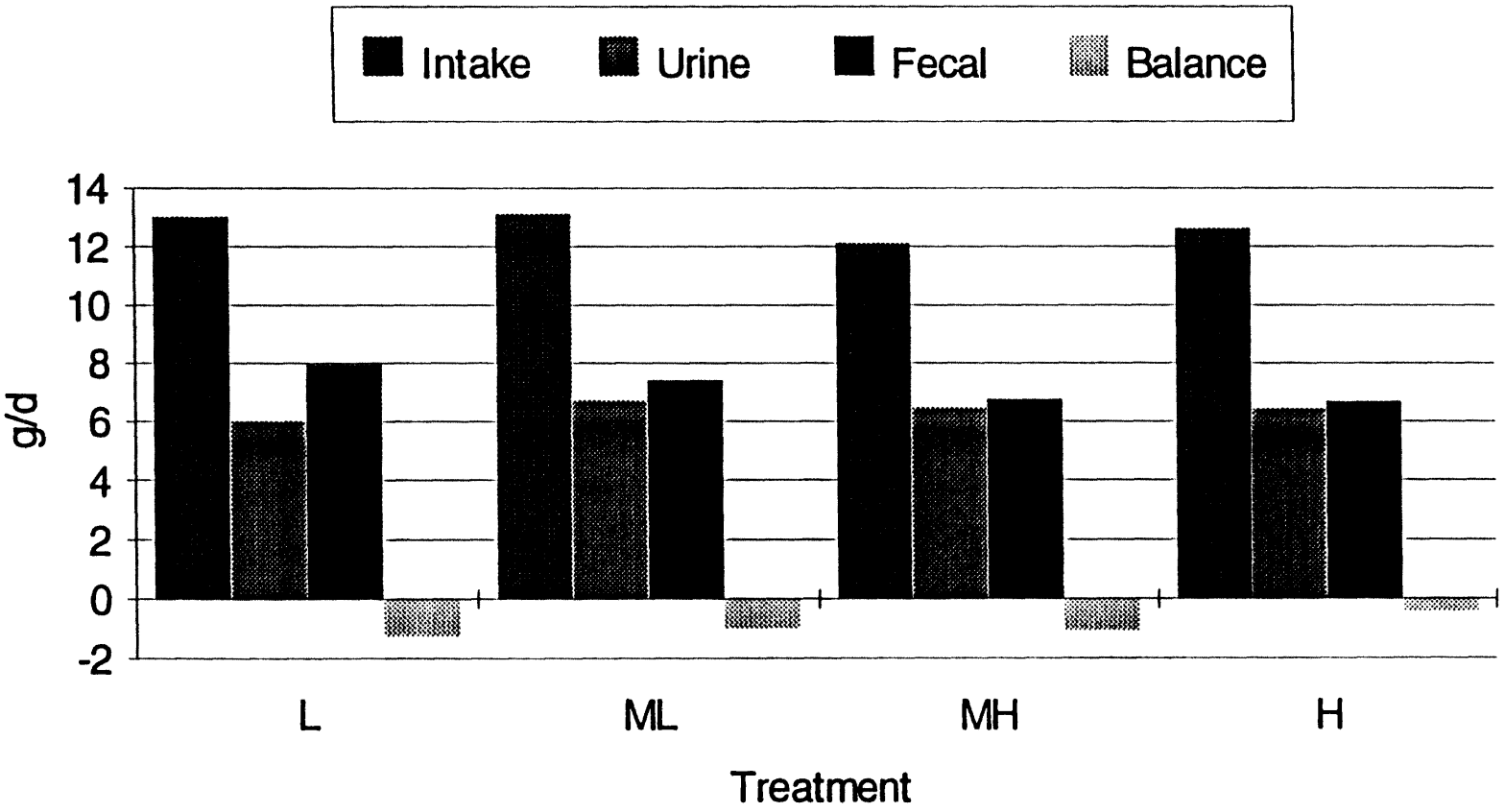

FIGURE 11. THE EFFECT OF DCAB ON MAGNESIUM BALANCE IN SEDENTARY HORSES.

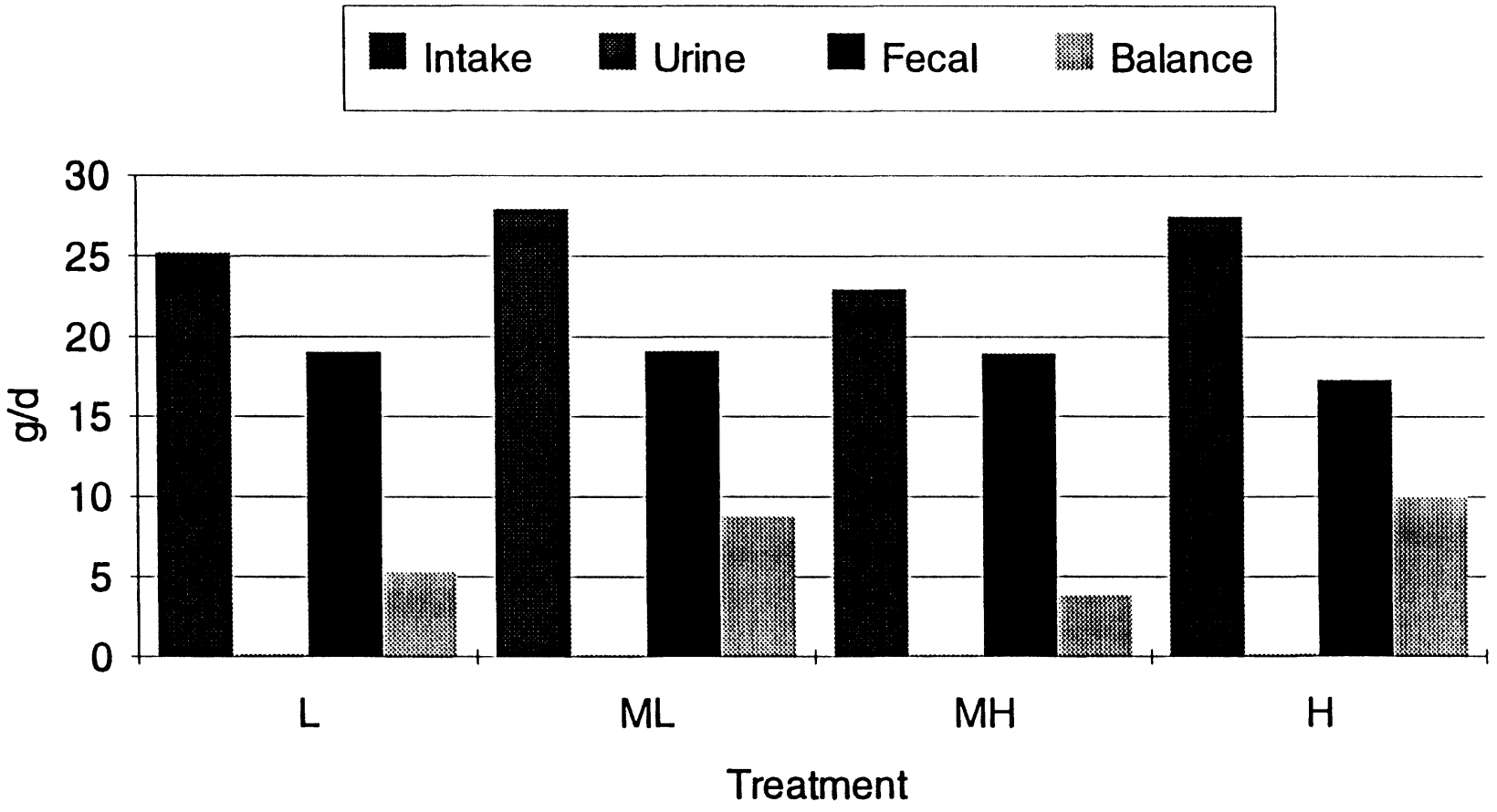

FIGURE 12. THE EFFECT OF DCAB ON PHOSPORUS BALANCE IN SEDENTARY HORSES.

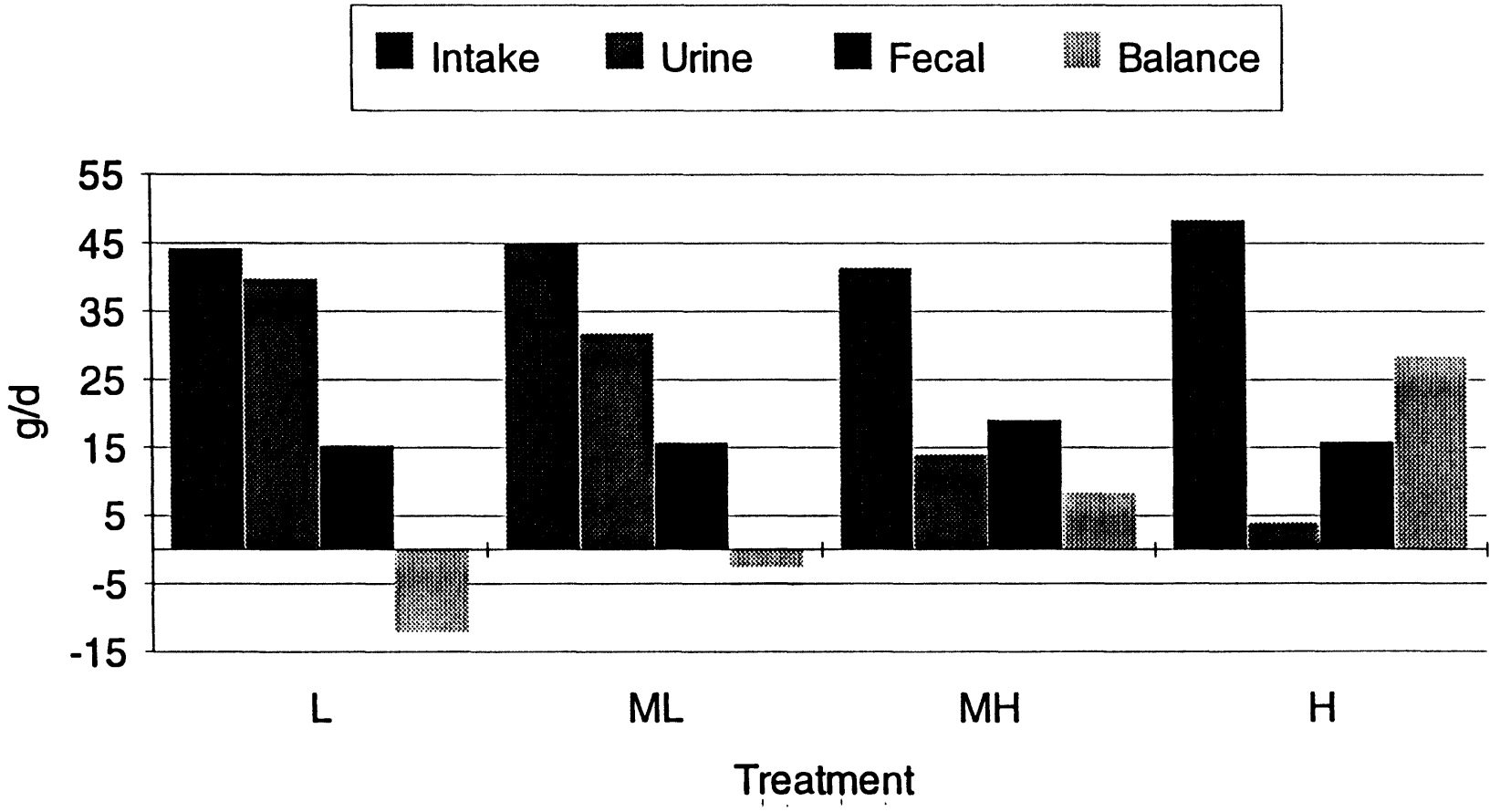

FIGURE 13. THE EFFECT OF DCAB ON CALCIUM BALANCE IN SEDENTARY HORSES.

Vita

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