EFFECTS OF DIETARY CATION-ANION BALANCE ON BLOOD PARAMETERS IN THE EXERCISING HORSE

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CHAPTER I

INTRODUCTION

Today's horse industry is more competitive than ever. Some horsemen are beginning to use scientific methods for answers to assist them in conditioning their horses for competitive events. Studies have been conducted in exercise physiology, biomechanics, and nutrition that are proven to improve performance in the equine athlete. Little research effort has been made to determine the effects of dietary cation-anion balance (DCAB) on exercising horses. Presently it is not known how varying DCAB might affect acid-base measures of blood and urine, mineral metabolism, bone strength, soundness, or performance in exercising horses.

The effects of DCAB have been well documented in many other species. The dairy industry has discovered several uses for varying the levels of DCAB in dairy cows. Block (1984) manipulated dietary cations and anions to formulate a ration with a low DCAB that when fed to prepartum dairy cows completely eliminated parturient paresis during lactation. Tucker (1988) found that feeding a diet with a high DCAB will increase milk production. At the present time the Equine NRC (1989) has no recommendation on the optimum DCAB.Nutrition of the exercising horse has always been a highly debated topic. Determining the optimum DCAB may improve performance and remove some of the myth of feeding horses.

It is necessary to continue research in the area of DCAB in order to see if it can positively affect performance of the equine athlete. The purpose of this project was to determine the effects of DCAB on exercising horses. Therefore the objectives of this study were: 1. To determine the effect of varying DCAB on blood pH and blood gases; 2. To determine the effect of varying DCAB on blood glucose and lactate; 3. To determine if performance may be enhanced or hindered by varying DCAB.

CHAPTER II

REVIEW OF LITERATURE

Definition of Dietary Cation-Anion Balance

The Mongin Sum equation, mEq((Na+K)-Cl)/100g diet dry matter, determines the Dietary Cation-Anion Balance, (DCAB), (Mongin 1980). Research conducted by poultry nutritionists demonstrated that dietary sodium (Na^{+}), potassium (K^{+}), and chloride (Cl^{-}) are the major inorganic ions affecting the acid-base status of the animal. The result of the equation is dependent on the percentage of sodium, potassium, and chloride in the diet. If the results of the equation are positive, the diet is considered cationic. Conversely, if the results of the equation are negative the diet is considered anionic. The milliequivalent units expressed in Mongin's equation indicate the charge on the ion, or the valence, as the primary factor affecting acid-base physiology. Recent research conducted by Tucker, et al. (1991) demonstrated that dietary chloride and sulfur had similar effects on the acid-base status of dairy cows. This concurs with Oetzel's (1991) research which analyzed previous studies conducted on acid-base balance in dairy cattle. He determined that sulfur is the primary ion affecting acid-base balance.

Therefore, both researchers recommended that sulfur be included in the equation for determining DCAB for lactating dairy cows.

Dietary Cation-Anion Balance Research in Different Species

Poultry. Poultry nutritionists recognize the importance not only of the inclusion of sodium, potassium, and chloride in the diet, but also the balance achieved by the ratios of these three elements. Mongin (1968) discovered that the acid-base status of the laying hen was altered during egg-shell calcification. As a result, nutritionists devised a method to alter the acid-base status of the hen to improve eggshell strength and thickness. Leach (1979) used the term cation-anion balance to refer to the relationship between sodium, potassium, and chloride, and their interaction to maintain acid-base balance. Since Mongin's 1968 study, specific research has been conducted on the resulting effect of DCAB on egg-shell strength and thickness (Austic, 1982; Hamilton and Thompson, 1979;).

Cohen, et al.(1972) analyzed the effect of sodium to chloride ratios on acid-base status in the diets of laying hens. He observed that venous blood parameters, pH, and bicarbonate are a function of the dietary Na^+/Cl^- ratio, regardless of the concentration of the two ions in the diet. Furthermore, by maintaining a constant level of dietary chloride, an alkalosis was produced with dietary sodium supplements. Conversely, an acidosis was produced when dietary sodium was held constant and chloride was supplemented to the diet. This is in agreement with research conducted by Hurwitz, et al. (1973) who reported that sodium is alkalogenic and chloride is acidogenic in the diets of chicks. Similar results occur when potassium is incorporated into the diet (Cohen and Hurwitz ,1974). Potassium produced the same alkalogenic effect as sodium. Sodium and potassium are additive in their effects and may offset the acidosis produced by elevated levels of dietary chloride. Such research concurs with findings by Nesheim, et al. (1964) who recommended supplementation of sodium and potassium as a means of counteracting the retardation of growth in diets of chicks with high levels of chloride. Mongin (1980) researched the interaction between acid-base balance of the blood and cations and anions. Studies involving DCAB in poultry indicated that chloride depresses blood pH, bicarbonate, base excess, and pCO_2 (Adekunmisi et al. 1987; Cohen et al. 1974; Hamilton et al. 1980; Riley et al. 1984; Teeter et al. 1986).

Research on growth in poultry related to DCAB has also received considerable attention. Nesheim, et al.(1964) examined the effect of the cations sodium and potassium and the anions chloride and sulfate on the growth of chicks. Unless higher levels of anions were offset with equimolar amounts of sodium or potassium, excess dietary chloride or sulfate ions lowered growth significantly. Melliere and

Forbes (1966) examined the addition of an acid, a base, or a combination of these two factors on food consumption and growth in chicks. Food consumption and weight gain were dependent on the dietary cation-anion ratio. Maximum gain and intake were realized at ratios of 1.2 to 1.8, and totally suppressed at a ratio of 0.6.

Nesheim et al.(1964) established the importance of an accurate balance of dietary electrolytes to maximize performance. Mongin and Sauveur (1977) reported the optimum level for growth was an electrolyte balance of 250 meq/kg diet DM. Johnson and Karuajeewa (1985) concluded that an electrolyte balance between 250 and 300 meq/kg provided for the highest level of growth. Adekunmisi and Robbins (1987) investigated the influence of DCAB on performance and the physiological and metabolic effects of growing broiler chicks. The electrolyte balance which provides for optimum growth is dependent upon the dietary crude protein content. The researchers supplemented the diet with sodium and potassium citrate to increase the electrolyte balance from 200 to 350 meq/kg. Improved gains and feed consumption of chicks consuming high protein diets (28.6%) were reported. Both gain and feed consumption were depressed in chicks fed diets low in protein (14.3%). The addition of sodium chloride and/or potassium chloride in the diets also suppressed growth in both high and low protein diets. Hurwitz et al. (1973) reported that optimal performance occurred when the sodium to chloride ratio (on a weight to weight basis) is 1:1. Furthermore, weight gain increased until the sodium chloride level reached .13%.

In addition to affecting acid-base balance, feed intake, and growth, DCAB may contribute to the occurrence of leg abnormalities in young chicks. Halley et al. (1984) manipulated the cation:anion ratio in the diets of chicks. A lower cation to anion ratio increased 3 week body weights and the incidence of both dyschondroplasia and varus deformation. Calcium or magnesium supplementation to diets containing high levels of anions decreased the incidence of leg problems and lowered three week body weights. Sauveur and Mongin (1978) used sodium, potassium, and chloride as variables to study tibial dyschondroplasia in chicks. The incidence of tibial dyschondroplasia was closely related to the chloride content of the diet and in turn was directly related to the acid-base balance of the blood.

As previously stated, chloride has a negative effect on acid-base parameters. However, Teeter, et al. (1985) suggested beneficial effects of chloride supplementation in thermostressed chicks. Respiratory alkalosis resulting from thermostress was reduced and live weight gain increased by the addition of chloride in the form of NH_4CL.

Swine. Dietary Cation-Anion Balance has recently been studied by swine researchers, investigating growth and feed intake (Austic et al. 1983; Coffey et al. 1985; Golz and Crenshaw 1991; Honeyfield et al. 1985; Patience et al. 1987; Yen et al. 1981). Yen et al. (1981) studied the effect of calcium chloride ($CaCl_2$) as a regulator of feed intake and weight gain in pigs. His research demonstrated the effect of chloride on acid-base balance in swine. The addition of 4% $CaCl_2$ to the basal diet of crossbred barrows lowered daily feed intake and suppressed both weight gain and feed efficiency. Calcium chloride supplementation resulted in increased plasma chloride concentration and lowered blood pH, HCO_3^-, total CO_2, and base excess. He also investigated the supplementation of 2.03% sodium bicarbonate ($NaHCO_3$) to the diet containing $CaCl_2$. The addition of $NaHCO_3$ prevented the harmful effects of chloride on acid-base status. When pigs were supplemented with $NaHCO_3$, higher weight gains, feed intakes, and feed efficiencies were demonstrated as compared to the $CaCl_2$ diets. Austic, et al. (1983) demonstrated an increased growth rate and improved feed efficiency in young growing swine when $NaHCO_3$ was supplemented to lysine deficient diets. A second experiment attempted to pinpoint the upper and lower confines in electrolyte balance which permit optimum growth performance in young swine. Six diets ranging from -100 to 500 meq/kg of diet dry matter were formulated using the equation Na^++K^+-Cl^-. Although not statistically significant, diets in the range of 100-300 mEq/kg of diet dry matter produced the highest level of performance.

Additional research of dietary electrolytes in swine nutrition, has primarily been concerned with the interaction of dietary potassium and lysine utilization. Higher levels of potassium, rather than DCAB, contributed to reduced growth in young pigs (Coffey et al. 1985; Froseth et al. 1983). This data is inconsistent with Austic et al. (1983) who suggests that the addition of potassium tended to improve performance of pigs fed diets low in lysine. Honeyfield et al. (1985) researched the effect of sodium and chloride on growing and finishing pigs. The optimal ratio for average daily gain and feed efficiency was suggested to be .13% sodium and .17% chloride. Patience, et al. (1987) examined the performance of young pigs fed diets with dietary cation-anion balances of -85 to 341 mEq/kg diet DM. An electrolyte balance between 0 and 341 mEq/kg diet DM of $((Na^+ + K^+) - Cl^-)$ maximized average daily gain and feed intake, whereas these parameters were lowered at -85 mEq/kg diet DM. Acid-base balance suffered deleterious effects at 0 mEq/kg diet DM. In addition, Honeyfield, et al (1985) studied the effect of DCAB on pigs fed lysine and tryptophan adequate diets compared to trytophan deficient diets. Two experiments were conducted where the basal diet contained a balance of 135 mEq/kg diet DM. Deficiencies of lysine and trytophan caused depressed growth, feed intake and feed efficiency. None of these responses were augmented by supplementing $NaHCO_3$ to the diet. A similar experiment conducted by Honeyfield, et al. (1985) in which the DCAB was 61 mEq/kg diet DM, $NaHCO_3$ supplementation significantly increased growth and feed intake when lysine and trytophan were limiting. They concluded the supplementation of $NaHCO_3$ in pigs deficient in lysine is dependent upon the electrolyte balance of the diet, and is also influenced by other dietary amino acids. Golz and Crenshaw (1990), investigated the response of dietary sodium, potassium and chloride on growth in young pigs. Potassium and chloride interacted reciprocally, affecting weight gain in young pigs. The weight gain was dependent upon the potassium to chloride ratio. Gain was depressed by .07 kg/day when dietary chloride was increased to .57% with .1% potassium in the diet. However, gain was increased by .16 kg/day when dietary chloride was increased with 1.1% potassium in the diet. Dietary sodium ranged from .03% to .60%. Within these confines no interactions between sodium and potassium or sodium and chloride were detected.

Dairy Cattle. Recent studies have examined the significant effects of DCAB on the performance parameters of dairy cattle. Kellaway et al. (1977) examined the results of supplementing the diet with 2, 11, 20, or 29 grams. of sodium chloride (NaCl) or sodium bicarbonate ($NaHCO_3$) in order to predict the effect of these supplements on food intake, growth and acid-base balance in calves. Total dry matter intake and growth were greater when 29g Na^+/kg DM was fed versus 2g Na^+/kg DM before weaning. Calves supplemented with $NaHCO_3$ exhibited linear increases in intake and growth rate with the high sodium diet when compared to the low sodium diet. The only significant response with NaCl was intake which was 16% higher on the diet with 11 g/kg DM sodium diet as compared to the 2 g/kg DM sodium diet. Observations of acid-base balance indicated that the addition of up to 20g Na^+/kg DM from NaCl or $NaHCO_3$ had no adverse effects on the calves. Concentrations above this level resulted in a marked increase in base excess and blood pH associated with the addition of $NaHCO_3$. Escobosa et al. (1984) lowered the DCAB to -144 meq/kg diet DM and raised it to +350 meq/kg diet DM as compared to a control diet of +204 meq/kg diet DM to examine the physiological responses of lactating dairy cattle in hot weather. The diet with a DCAB of -144 meq/kg diet DM lowered feed intake and body weight. Glucose was significantly higher for the cows consuming the diet high in sodium. The blood acid-base parameters of cows on the high chloride diet suffered adverse effects, including a partially compensated metabolic acidosis.

Block (1984) examined diets formulated to contain a low dietary cation-anion balance preventing the occurrence of parturient paresis in lactating dairy cattle. He utilized a slightly modified equation $(Na^+ + K^+) - (Cl^- + S^-)$ meq/100g diet DM). He formulated an anionic diet of -12.85 meq/100g diet DM and a cationic diet of 33.05 meq/g diet DM. Both diets contained .65% calcium and .25% phosphorous. This study prompted the initial research on the effect of monovalent ions and parturient paresis in lactating dairy cattle. Cows consuming the acidic diet had no milk fever, but cows consuming the alkaline diet had a 47.4% incidence. The cows consuming the anionic diet had significantly ($p<0.05$) higher milk yields. Adequate calcium and phosphorous levels were maintained through parturition for cows consuming the acidogenic diet, whereas cows consuming the alkalogenic diet encountered a decrease in plasma calcium and phosphorous around calving. Although, the diets contain a high calcium to phosphorous ratio, calcium mobilization from the bone may have occurred during calcium stress as a result of the acidogenic diet. This systemic reaction may be attributed to the result of a liver and kidney response to the drop in blood pH. Goff et al. (1991) analyzed the addition of anions to the prepartum diet of dairy cows to examine what effects 1,25 dihydroxyvitamin D had on preventing milk fever. Two diets were formulated; the cationic diet contained +1,060 meq/kg diet DM and the anionic diet contained -143 meq/kg diet DM. Six of the 23 cows on the highly cationic diet developed milk fever, whereas one of 24 cows on the highly anionic diet contracted milk fever. The assay for 1,25 dihydroxyvitamin D indicated that cows fed the alkalogenic diet failed to synthesize the necessary amounts of this hormone. The failure to synthesize 1,25 dihydroxyvitamin D could have contributed to the milk fever. In a related study Oetzel (1991) employed the technique of meta-analysis to analyze the data from 75 published trials to determine the nutritional risk factors for milk fever. Dietary sulfur and DCAB were the two nutritional factors most highly correlated to the incidence rate of milk fever. A regression analysis showed that dietary sulfur decreased the odds of developing milk fever, whereas higher levels of sodium and crude protein increased the odds of producing milk fever. There was a nonlinear relationship between incidence of milk fever and dietary calcium. This research supports the theory that the incidence rate of milk fever is primarily influenced by dietary cation-anion balance. Tucker, et al. (1988) showed that feeding a cationic diet of +20 meq/100g diet DM increased milk yield by 8.6%. There was a linear increase in blood pH and bicarbonate with increasing DCAB. Serum Cl^- is affected most dramatically by changes in DCAB and may be the ion primarily responsible for altering acid-base physiology.

Beighle, et al. (1988) studied the effect of dietary cation-anion balance on growth and serum inorganic phosphorous in dairy calves. The anionic diet, -14 meq/100g diet DM, exhibited marked improvement in performance over the cationic diet, +39 meq/100 g diet DM, at the lowest phosphorous concentration. The interaction of dietary phosphorous and cation-anion balance was responsible for significant differences in calf performance. The DCAB affected mean feed intake and average daily gain throughout the trial. Calves consuming the anionic diets had intakes of 4.0 kg/day and gained .94

kg/day compared with 3.7 and .84 kg/day for calves consuming the cationic diet. Calves receiving the high phosphorous diet (.37%) consumed 4.2 kg feed and gained .97 kg body weight/day compared with 3.8 and 9.2 kg for calves fed .29% phosphorous, and 3.6 and 7.8 kg for those fed .22% phosphorous. The results may indicate more efficient utilization of phosphorous by the calves receiving anionic diets when dietary phosphorous was low. Tucker, et al. (1991) evaluated the effects of dietary NaHCO3 on potassium metabolism in growing dairy calves. Average daily gain (ADG) increased with higher levels of potassium. Gain tended to be reduced by dietary $NaHCO_3$ supplementation. This study suggested the potassium requirement of the growing calf was between .40 and .55% of diet DM. Average daily gain and plasma potassium were considered accurate indicators of dietary potassium in the growing calf.

Tucker et al. (1991) studied the influence of calcium chloride ($CaCl_2$) on systemic acid-base status and calcium metabolism. Hydrogen ion concentration in the blood increased linearly whereas blood HCO_3^- decreased linearly with increasing levels of $CaCl_2$. The acidogenic capacity of Cl^- is the likely cause in the reduction of blood HCO_3^- as a result of $CaCl_2$ supplementation. Blood pCO_2 decreased when supplemental $CaCl_2$ was raised from 1% to 1.5%, and responded quadratically to the four levels of supplemental $CaCl_2$. Diets with 1.0 and 1.5% $CaCl_2$ created an acidic condition in the acid-base balance of dairy cows. Urine calcium excretion was elevated in cows consuming the 1.0 and 1.5% $CaCl_2$. This is most likely the result of increased mobilization of calcium. Blood HCO_3^- was greatly reduced when the diet was supplemented at the 1.5% level. Tucker, et al. (1991) recommended feeding 1% $CaCl_2$, for 3 weeks prepartum to prevent parturient paresis without compromising systemic acid-base balance.

West (1990) conducted a study to determine the effect of diets containing differing cation-anion balances on performance and physiologic responses of lactating dairy cows during cool or hot environmental conditions. Increasing the electrolyte balance in the diet resulted in increased milk production and feed intake. The environmental conditions had no significant affect on feed intake or milk yield.

Horses. Little research pertaining to the effect of DCAB on the exercising horse is presently available. However, previous research touches on the subject of electrolyte balance in horses. Milne (1974) conducted a series of experiments to examine the effect of exercise on blood gases, acid-base balance, electrolyte, and enzyme changes in exercising horses. The first experiment compared arterial and venous blood gases in unconditioned horses at rest. The standard exercise test (SET) consisted of two sub maximal work loads with the second the more strenuous of the two. Both arterial and venous samples were drawn. Acid-base balance suffered no serious imbalances during moderate work, but all horses experienced a partially compensated metabolic acidosis during heavy work. Arterial and venous pH, pCO_2, and HCO_3^- are linearly related, and arterial blood gases could be accurately predicted from a knowledge of venous blood during exercise. In the second experiment, horses with no previous conditioning were subjected to a sub maximal anaerobic exercise test. After a workload performed at 700 m/min. venous O_2 increased over two times the resting levels. In the third experiment, horses were subjected to an 80 km endurance ride. HCO_3^- and pCO_2 were significantly decreased at the completion of the ride, and pH significantly elevated during the middle of the ride. This may indicate that $NaHCO_3$ supplementation does not benefit the endurance horse as it would further elevate pH and produce a severe alkalosis. The severity of exercise may dictate what effect exercise has on blood gases and acid-base balance. Furthermore, the anaerobic threshold in unconditioned horses was reached when the work load is in excess of 350 m/min. As fitness levels improve it is thought that anaerobic threshold is increased as well as blood buffering capabilities.

Williamson (1974) attributed the cause of poor race track performance to abnormal serum electrolyte levels (Na^+, K^+, Cl^-, and HCO_3^-). Both hyperchloremic acidosis and hypochloremic alkalosis has been observed. These disorders were treated by electrolyte therapy by supplementing the diet, by drench, or intravenously. His results demonstrated, that electrolyte therapy to treat acidosis and alkalosis may improve poor race track performance in some horses.

The use of $NaHCO_3$ has enhanced the performance of equine athletes, particularly racehorses. Lawrence et al. (1987) examined the effects of sodium bicarbonate administration prior to exercise and recovery to determine if this practice would augment the blood bicarbonate system. During the exercise test, three horses received 300 mg/kg body weight of $NaHCO_3$ administered by drench 1.5-2.5 hours prior to the exercise test and the other three horses received a placebo by the same method. The time to fatigue was extended by the $NaHCO_3$ treatment. Preexercise venous pH and HCO_3^- levels were elevated by the treatment. Blood pH was reduced in both treatments, but remained significantly higher on the $NaHCO_3$ treatment during exercise and recovery. Sodium bicarbonate supplementation significantly elevated venous lactate levels during exercise and recovery. $NaHCO_3$ administration may aid horses competing in events that induce a significant metabolic acidosis.

Kelso et al. (1987) examined the effect of bicarbonate administration on blood and muscle metabolite concentrations and pH before and after a supra maximal exercise bout. Blood pH and HCO_3^- concentrations were significantly elevated ($p<0.05$) prior to exercise.

However, there were no significant blood gases differences between water and HCO_3^- treatments post exercise. This research is in agreement with Lawrence, et al. (1987) which showed that the administration of NaHCO3 elevated blood pH and HCO3⁻ concentrations prior to exercise, and raised muscle and blood lactate concentrations during exercise. Upon the cessation and recovery of exercise, differences between the water and bicarbonate treatments for metabolites and blood gases were not evident. These findings contradict the results of Lawrence et al. (1987). A more recent study by Lawrence et al. (1990) examined the effect of bicarbonate administration. Improved times were reported when horses were treated with $NaHCO_3$ 2.5 hours before racing. Blood lactate removal was faster with the $NaHCO_3$ treatment which may extend exercise before the onset of fatigue caused by intramuscular acidosis.

Roberts et al. (1991) studied the metabolic changes during exercise in horses given sodium bicarbonate. The administration of $NaHCO_3$ induced a metabolic alkalosis by raising blood pH, HCO_3^-, standard base excess and pCO_2. Treatment contributed no effect to pCO_2 or heart rate during exercise or recovery. Treatments did not affect lactic acid concentration, pCO_2 or heart rate at rest. However, heart rate and plasma lactic acid concentration were elevated due to exercise which resulted in decreased blood pH, HCO_3^-, SBE and pCO_2 in horses on both treatments. Horses treated with $NaHCO_3$ had significantly higher blood pH, HCO_3^-, and SBE, as compared to horses given the placebo ($p<0.05$). Although the treated horses tended to use more muscle glycogen the sodium bicarbonate drench did not elicit any significant changes in muscle glycogen.

Training Methods and Heart Rate Research

Exercise to a maximum where heart rates exceed 210 beats per minute initiated marked changes in the serum electrolytes Na^+, K^+, Cl^-, and HCO_3^- as well as blood gases and acid-base balance (Carlson et al. 1987, Judson et al. 1983, Milne, 1974, Snow et al. 1983 and Williamson 1974). Asheim et al. (1970) examined heart rate and blood lactate concentrations in Standardbred horses during training and racing. Heart rates varied from 210 to 238 bpm during maximum work. Interval training gave the most effective circulatory training. Interval training is a training method involving conditioning of athletes by performing many exercise bouts. Between the exercise bouts partial recovery (usually detected by heart rate) is allowed before resuming the next bout. The advantages of interval training may include development of an animal that can endure heavier work loads, and may delay the onset of fatigue.

Harkins and Kammerling (1990) compared interval training and conventional training. They reported that there were no significant differences in heart rates or run times between the two training methods. No refutable data supported the proposition that interval training was advantageous to conventional training. However, the interval trained horses did seem to have more endurance and finish stronger in exercise tests.

Foreman et al. (1990) examined a standard exercise test and daily heart rate response of Thoroughbred horses undergoing conventional race training and detraining. After nine week of training, he reported significant differences were seen training in the standard exercise test (SET) recovery heart rate at 0.5 through 5 minutes after exercise ($P<0.05$ to $P<0.01$). Differences were also seen after detraining in the SET recovery heart rate at 40 and 60 minutes after exercise ($P<0.05$ to $P<0.01$). Recovery heart rate in the post training race track SET was significantly decreased. The conventional training method used may increase cardiovascular fitness and the recovery heart rate is an accurate indicator of fitness. The heart rates for the horses that were lame during exercise were significantly higher than the heart rates for horses showing no visible signs of lameness. This may indicate that heart rate is a valuable tool in detecting lameness. This is in agreement with Erickson et al. (1987) who reported that detection of injury can be diagnosed from heart rates particularly if a heart rate computer is used.

Effect of Exercise on Blood Glucose

Normal levels of blood glucose in the horse range from

60-110 mg/dl. Stull, et al. (1987) reported that peak plasma glucose levels occurred three hours post feeding. Evans (1971) suggested horses that were physically conditioned went through an adjustment in their energy metabolism and used 150% more glucose than the horses in the control group. Webb, et al. (1987) looked at the physiological responses of cutting horses to exercise and reported that plasma glucose increased ($p<0.05$) with work. This is in agreement with Snow (1990) who studied the hematological changes in horses during the cross country stage of driving trials. Similarly, work in humans has also shown glucose to increase after exercise, particularly after ingestion of high levels of carbohydrate (Mitchell et al., 1989). The addition of dietary fat to rations of exercising horses and it effect on plasma glucose has recently been studied (Duren, et al. 1987; and Meyers, et al. 1989). Incomplete research is available on the relationship between acid-base balance and plasma glucose concentrations

<u>Acid-Base Status in Humans Performing Anaerobic Exercise</u>

Avenues to improve the athletic performance of humans is a never ending quest. Recently, highly technical methods including heart rate monitoring, retinal scanners, and computer imaging have been used in order to maximize human performance. Diet has also proven to be a beneficial tool in enhancing performance. Research on the effects of dietary cation-anion balance on the human athlete has yet to receive significant attention. Researchers have touched on the subject of dietary cation-anion balance by administering different electrolyte solutions to study their effect on acid-base balance and blood metabolites.

Jones et al. (1977) administered capsules containing calcium carbonate, (control), ammonium chloride, (acidosis), or sodium bicarbonate, (alkalosis), in a dose of 0.3 g/kg body weight to adult male subjects. Prexercise blood pH was altered by both experimental treatments. Blood pH was lowered to 7.21 $\pm$ 0.033 with NH_4Cl and raised to 7.43 $\pm$ 0.029 with $NaHCO_3$. When the subjects exerted maximum output, endurance times were lowered in the acidotic treatment and extended in alkalotic treatment. Acidosis produced an elevated level of alveolar ventilation. At all levels of exertion venous lactate was lowest in acidosis and highest in alkalosis. Alterations in blood pH and HCO_3^- concentrations appeared to exert some control on metabolism in exercise contributing to impaired endurance. Kowalchuk et al. (1984) examined the effect of pH on metabolic and cardiorespiratory responses during progressive exercise of adult males. The treatment protocol and the results are consistent with the previous research conducted by Jones et al. (1977). Kowalchuk and coworkers concluded that changes in blood parameters affect acid-base balance by controlling the maximum level of exertion that may be maintained in incremental dynamic exercise and modifying plasma lactate appearance, but have minimum effect on hydrogen ion seen in plasma.

Mitchell et al. (1990) administered sodium bicarbonate $NaHCO_3^-$ and sodium chloride NaCl intravenously to examine their effects on exercise in male subjects. Hydrogen ion concentration and HCO_3^- were similar in the control subjects and those subjects administered NaCl. Change was not detected from rest values in subjects administered $NaHCO_3$. Other parameters such as VO_2, VCO_2, and heart rate did not differ among the three treatments. Endurance was noticeably extended ($P<0.01$) in subjects administered $NaHCO_3$ and NaCl compared to the control. At exhaustion plasma glucose levels were higher ($P<0.025$) in the control group as compared to the $NaHCO_3$ and NaCl treatments. Administration of $NaHCO_3$ causes a prolonged metabolic alkalosis. The benefit of the alkalosis is increased buffering capacity of the blood during exercise. These findings are consistent with Wilkes et al. (1983).

CHAPTER III

MATERIALS AND METHODS

Four geldings and four mares of primarily Thoroughbred and Quarter Horse breeding were assigned to four blocks of two horses each according to sex. The blocks were used in two simultaneous 4x4 Latin squares so that each block was represented by one horse in each square. The horses were randomly assigned to four dietary treatments.

The trial was designed to subject the eight experimental animals to a six week conditioning period on a .8 km track consisting of Long Slow Distance (LSD) exercise six days/week. The purpose of the conditioning period was to establish an aerobic base and standardize the fitness level of all eight horses by increasing the distance galloped as well as the pace. The regimen for the first week of the conditioning period was galloping 1.6 km at a pace of .4 km/min, during the second week, 2.4 km at a pace of .4 km/min, for the third week 3.2 km at .4 km/min, during the fourth week, 3.2 km at .43 km/min (an increase in pace rather than distance) and finally during the fifth and sixth weeks 3.2 km at a pace of .46 km/min. During the pre-conditioning period all horses were conditioned on the ration presented in Table I. The ration was fed twice daily until the termination of the conditioning at which time experimental treatments were imposed.

During the experimental periods, horses combined aerobic exercise galloping 3.2 km at .46 km/min with an anaerobic interval training protocol. The interval training consisted of two .4 km sprints eliciting heart rates of 200 beats per minute (bpm). Between sprints, horses were slowed sufficiently for heart rates to recover below 110 bpm. Heart rates were recorded using a UNIQ[a] on-board heart rate monitor.

Ingredient compositions of the trial diets are shown in Table I and nutrient compositions are shown in Table II. Four diets were formulated to provide DCAB's of -50 (L), +50 (ML), +150(MH), and +250(H) meq/kg diet dry matter. Treatment diets consisted of a base concentrate of corn, soybean meal and cottonseed hulls. Calcium chloride and ammonium chloride were added to diet L, calcium chloride was added to diet ML, and potassium citrate and sodium bicarbonate were added to diet H to achieve the desired DCAB. The concentrate was fed with bermudagrass hay in a 60:40 ratio in amounts to maintain constant body weight.

Calcium and phosphorous were balanced at a ratio of 1.77 to 1 with ground limestone and dicalcium phosphate. Calcium was .5% of the diet and phosphorous was .3% of the diet. The digestible energy of all four diets were

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[a] UNIQ Computer Instruments Corp. Hempstead, NY.

TABLE I

INGREDIENT COMPOSITION OF HAY AND CONCENTRATES FED TO EXERCISING HORSES (DM BASIS)

			% of diet			
Ingredient	IFN	Pre[1]	L	ML	MH	H
Bermudagrass hay	1-09-210	40.00	40.00	40.00	40.00	40.00
Corn	4-02-985	33.25	33.24	33.25	33.25	33.20
Soybean meal	5-04-604	6.92	6.92	6.92	6.92	6.91
Cottonseed hulls	1-01-599	15.03	14.72	15.08	15.03	13.70
Limestone	-------	.78	.00	.22	.78	.77
Dicalcium Phosphate	-------	.19	.21	.21	.19	.20
Trace Mineralized salt	-------	.55	.55	.55	.55	.55
Sodium Bicarbonate	-------	.00	.00	.00	.06	.61
Potassium Citrate	-------	.00	.00	.00	.00	.89
Calcium chloride	-------	.00	.78	.54	.00	.00
Ammonium Chloride	-------	.00	.35	.00	.00	.00
Chromic oxide	-------	.22	.22	.22	.22	.22
Liquid cane Molasses	4-04-695	1.99	1.99	1.99	1.99	1.99

[1]Ration fed during the condition period.

TABLE II

NUTRIENT COMPOSITION OF HAY AND CONCENTRATES FED TO EXERCISING HORSES (DM BASIS)

	Diet			
Constituent	L	ML	MH	H
DE, Mcal/kg	2.7	2.7	2.7	2.7
Crude Protein,%	10.4	10.4	10.4	10.4
Calcium,%	.50	.53	.52	.54
Phosphorus,%	.28	.29	.28	.28
Magnesium,%	.15	.16	.15	.15
Potassium,%	1.12	1.14	1.13	1.39
Sulfur,%	.11	.12	.11	.13
Sodium,%	.29	.27	.30	.43
Chloride,%	1.38	1.00	.69	.68
DCAB	**+27**	**+130**	**+223**	**+354**

calculated to be 2.7 Mcal/kg DM based on NRC values. The equation developed by Anderson et al. (1983) was used to estimate the digestable energy requirements for work. Using an average body weight of 463 kg, the equation suggests feeding 25.43 Mcal/day. A horse weighing 463 kg doing moderate work requires approximately 982 g of crude protein. The treatment diets contained 936g of crude protein. Feeding took place at 1000 and 2200 hours. All horses were allowed 12 hours to consume their rations. Horses were weighed prior to the morning exercise bout once each week using a standard livestock scale.

After analyses of hay and concentrates, the actual cation:anion ratios in the complete diets were calculated to be +27.33, +130.48, +223.31, and 354.19 meq(Na^{+}+K^{+})-Cl^{-}/kg of diet dry matter for the four diets. Diet MH served as the control. Diets were fed for 21 days prior to the beginning of sample collection. Horses were maintained in individual box stalls with free access to water and were immunized and dewormed prior to the initiation of the trial. Standard animal health care was provided throughout the trial.

Beginning at the morning feeding on the 22nd day of the experimental period venous blood samples were drawn from the geldings hourly for 17 hours. The blood samples were used to examine how the four treatments effected blood parameters in relation to exercise, which preceded the morning feeding. An 18 gauge, 1.5 inch indwelling jugular catheter was inserted into the left jugular vein for the collection of venous blood into a 20cc syringe. After collection, 5 ml of heparinized saline was injected into the catheter to prevent clotting. Samples were transferred to an evacuated tube containing lithium heparin. The blood samples were immediately put into an ice water slurry and analyzed within two hours for blood pH, pCO_2, pO_2, HCO_3^-, TCO_2, SBC, BEecf, BEB, %sO_2c using an Instrumentation Laboratories Blood Gas Analyzer[b] that was standardized and calibrated prior to each sampling period. The same procedure was followed on the mares the third day of the experimental period.

On the last day of each experimental period, horses were subjected to a standard exercise test (SET) approximately 2 hours before the morning feeding. The SET consisted of a 1.6km sprint on the track at speeds to elicit a heart rate of 200bpm. Blood samples were drawn via jugular venipuncture into evacuated tubes containing lithium heparin and immediately placed in an ice water slurry and analyzed within 2 hours for blood pH, pCO_2, pO_2, HCO_3^-, TCO_2, SBC, BEecf, BEB, and %sO_2c. Venous blood also was collected into evacuated tubes containing potassium oxalate and sodium flouride for the analysis of lactate and plasma glucose concentrations. Samples were drawn before the SET at the end of the SET (time 0), and at 1, 2, 3, 4, 5, 10, 20, and 30 minutes post exercise.

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[b] Instrumentation Blood Gas Analyzer Model 1304, Lexington, MD.

One milliliter of whole blood was drawn from the potassium oxalate, sodium flouride tubes and transferred to a tube containing 2 ml. of cold 10% trichloroacetic acid for deproteinization and inverted back and forth for thirty seconds. It was then centrifuged 10 minutes at approximately 1500 x g. The clear supernatant was harvested into polyethylene tubes and the samples were frozen for subsequent lactate analysis. The remaining blood was centrifuged for 20 minutes and the plasma was harvested into polyethylene tubes. The samples were frozen for subsequent glucose analysis. Both glucose and lactate were analyzed on a Beckman Model DU64 Spectrophotometer[c] using commercially available analysis kits.

Statistical analyses which were suited to a Latin square experiment were performed as recommended by Steele and Torrie (1980). All data were analyzed using a general linear model procedure for repeated measures and least square means calculated for each parameter. The pDiff procedure was used to detect significant differences between means.

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[c] Beckman Model DU64 Spectrophotometer, Fullerton, CA.

CHAPTER IV

RESULTS AND DISCUSSION

Hourly Sample Analysis

Blood Acid-Base Status. Changes in blood pH over the seventeen hour collection, on day one of the experimental period are listed in Table III. Mean venous blood pH ranged from 7.342 in horses on the low diet to 7.402 in horses on the medium high diet. Blood pH (Figure 1) for horses consuming all dietary treatments dropped significantly the first hour post-feeding. Peak chloride absorption may have occurred at this time across treatments, corresponding to the lowest blood pH values. Chloride is absorbed in gastrointestinal tract in exchange for the secretion of a HCO_3^- ion. This is due to hydrogen ion being liberated from the intermediate carbonic acid by the enzyme carbonic anhydrase; resulting in increased systemic acid generation. Therefore, blood pH would be expected to decrease with decreasing DCAB. The decrease in venous blood pH in the medium high and high diets is probably not attributed to chloride but may be due to the exchange of potassium into the intracellular fluid for a hydrogen ion into the extracellular fluid at the cellular level.

TABLE III

EFFECT OF DIETARY CATION-ANION BALANCE ON
VENOUS BLOOD pH POST FEEDING IN
EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S E
10 am[a]	7 391^{b}	7.399^{b}	7.395^{b}	7.398^{b}	.005
11 am	7 342^{b}	7 368^{c}	7 365^{c}	7 370^{c}	004
12 pm	7.354^{b}	7 378^{c}	7 376^{c}	7 384^{c}	006
1 pm	7.366^{b}	7 380^{c}	3 379^{c}	7 390^{c}	004
2 pm	7 366^{b}	7 381^{c}	7.380^{c}	7 387^{c}	004
3 pm	7.366^{b}	7 381^{c}	7.381^{c}	7 383^{c}	.005
4 pm	7 370^{b}	7 385^{c}	7.385^{c}	7 398^{c}	004
5 pm	7 371^{b}	7 376^{bc}	7 387^{cd}	7.390^{d}	005
6 pm	7 374^{b}	7 383^{bc}	7.394^{cd}	7 395^{d}	.004
7 pm	7.375^{b}	7 392^{c}	7.392^{c}	7 400^{c}	004
8 pm	7.382^{b}	7.398^{c}	7.398^{c}	7 395^{c}	004
9 pm	7 383^{b}	7.388^{b}	7 395^{b}	7 391^{b}	004
10 pm[a]	7 396^{b}	7 397^{b}	7.402^{b}	7 398^{b}	003
11 pm	7.348^{b}	7.368^{b}	7 359^{b}	7 356^{b}	007
12 am	7.360^{b}	7.378^{c}	7 379^{c}	7 384^{c}	006
1 am	7 374^{b}	7 381^{bc}	7 398^{d}	7 392^{cd}	005
2 am	7 374^{b}	7.377^{bc}	7.392^{d}	7 387^{cd}	.005

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

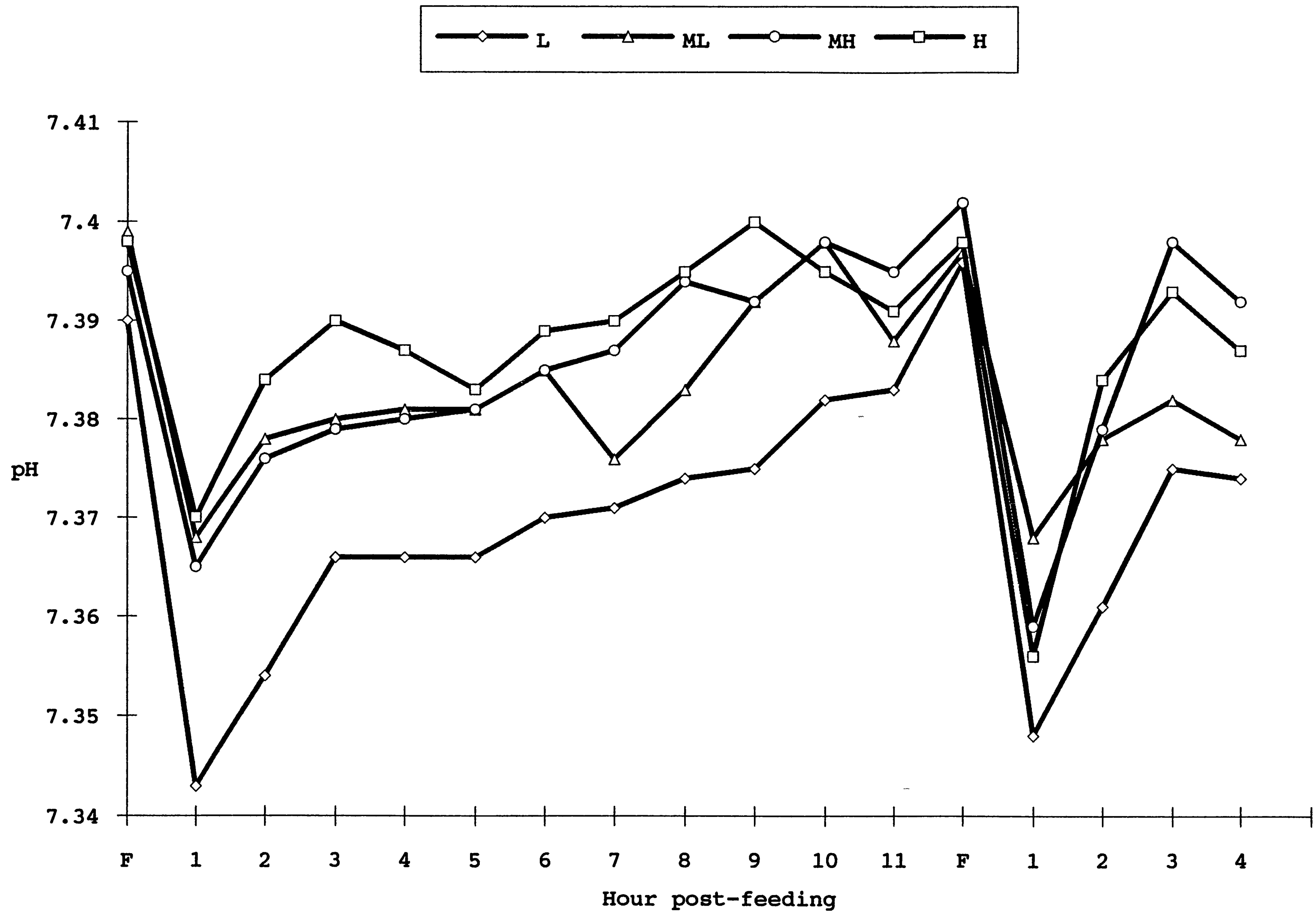

Figure 1. Effect of Dietary Cation-Anion Balance on Venous Blood pH Post Feeding

Mean venous blood pH decreased with decreasing DCAB. Venous blood pH was lower ($p<0.001$) in horses consuming the low diet as compared to horses consuming the medium low, medium high, and high diets throughout the twelve hour post-feeding interval. The drop in blood pH at one hour post feeding may indicate a metabolic or respiratory acidosis due to the high chloride level in the low diet. This is in agreement with Tucker et al. (1988) who reported a decrease in blood pH in dairy cows fed highly anionic diets. Blood pH did not recover to normal values until the time of the evening feeding.

Changes in venous blood pCO_2 over the seventeen hour collection on day one of the experimental period are listed in Table IV. Venous blood pCO_2 (Figure 2) responded similarly across all dietary treatments by rising sharply at one hour post feeding indicating a respiratory acidosis. This rise was followed by a dramatic drop at two hours post feeding. This drop may be explained by the respiratory compensation that occurs due to elevated levels of pCO_2. Horses consuming the low diet had significantly lower ($p<0.05$) pCO_2 levels as compared to horses consuming the high diet at ten of the seventeen hours measured. Mean venous blood pCO2 ranged from 46.450 mmol/L in horses on the low diet to 55.200 mmol/L in horses consuming the high diet. Venous blood pCO_2 levels were lower after the morning feeding versus the evening feeding. This may have

TABLE IV

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pCO_2 (mmHg) POST FEEDING IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	48.02[b]	49.86[b]	50.67[b]	49.51[b]	1.11
11 am	52.58[b]	50.76[bc]	53.75[d]	52.97[bd]	0.87
12 pm	48.56[b]	48.27[b]	49.05[b]	49.43[c]	1.08
1 pm	46.45[b]	47.70[bc]	48.57[c]	48.81[c]	0.72
2 pm	47.08[b]	47.66[b]	48.93[c]	49.00[c]	0.44
3 pm	46.98[b]	47.91[b]	49.37[c]	50.50[c]	0.48
4 pm	47.18[b]	48.37[bc]	50.46[c]	50.62[c]	1.07
5 pm	47.10[b]	49.06[bc]	49.13[bc]	50.28[c]	0.98
6 pm	47.75[b]	48.75[b]	49.28[b]	49.66[b]	0.80
7 pm	48.18[bc]	47.87[b]	49.82[cd]	50.20[d]	0.64
8 pm	47.28[b]	47.96[bc]	49.58[cd]	50.45[d]	0.71
9 pm	48.05[b]	49.95[b]	49.77[b]	50.88[b]	1.13
10 pm[a]	47.63[b]	48.71[bc]	49.01[bc]	49.78[c]	0.65
11 pm	53.03[b]	51.45[b]	53.11[b]	55.20[b]	1.27
12 am	49.26[b]	48.31[b]	50.95[b]	49.71[b]	0.98
1 am	47.62[b]	48.05[b]	48.15[b]	48.08[b]	0.80
2 am	47.65[b]	49.20[bc]	48.73[bc]	50.38[c]	0.74

[a]Indicates feeding time.

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

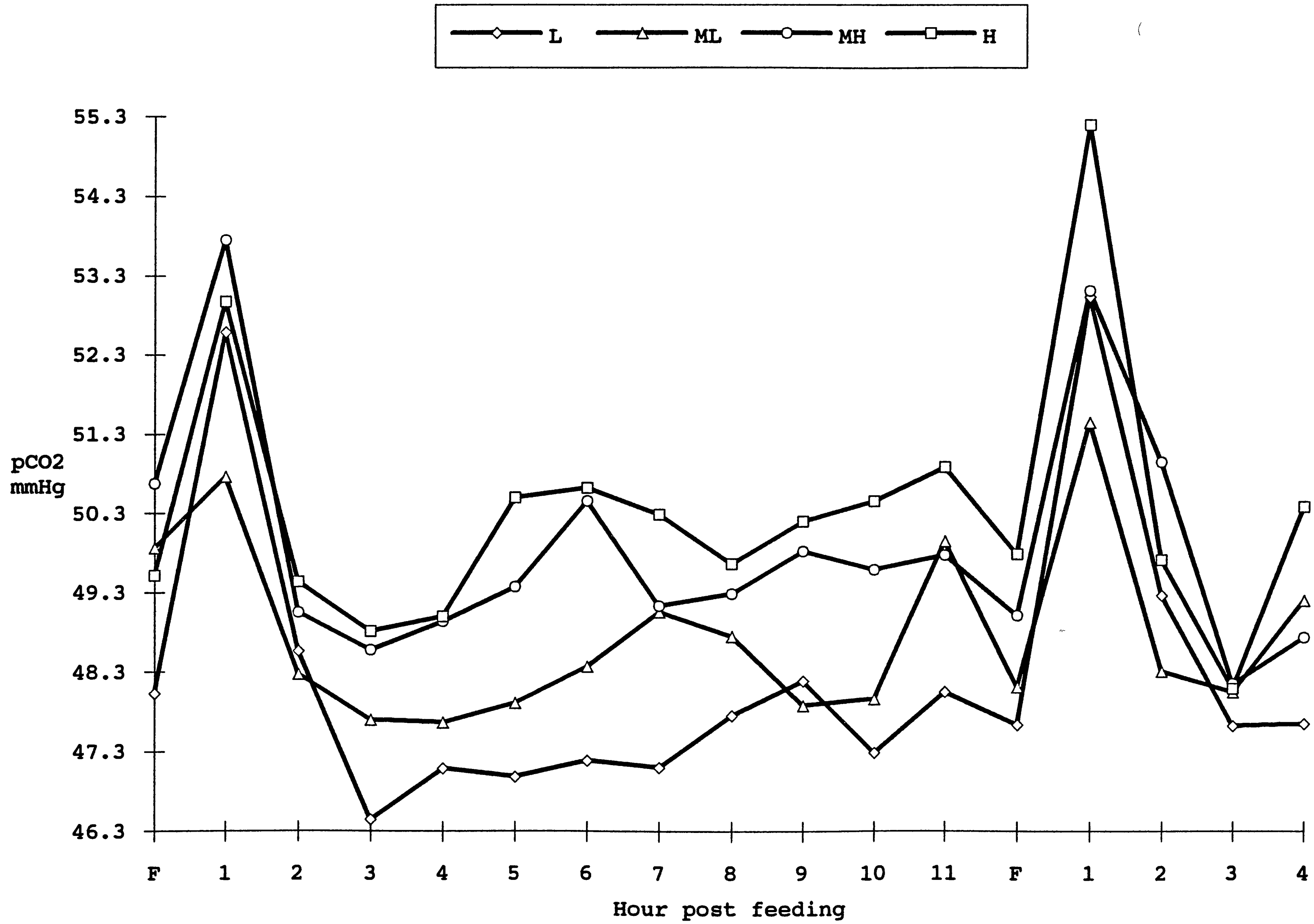

Figure 2. Effect of Dietary Cation-Anion Balance on Venous Blood pCO_2 Post Feeding

resulted from the exercise bout the horses performed that morning.

Diet had no significant effect on venous pO_2, except at one hour post feeding A.M.. Partial pressure of O_2 in horses on the low diet was significantly lower ($p<0.05$) than horses on the medium low diet and high diets (Table V and Figure 3). Levels of pO_2 in horses on the low and medium high diets produced reciprocal results as compared to pCO_2 at hours one and two post feeding A.M.. This would indicate that alveolar ventilation did increase to compensate for the rise in pCO_2. Means for venous pO_2 ranged from 35.37 mmol/L in horses consuming the low diet to 47.87 mmol/L in horses on the medium high diet.

Changes in venous blood HCO_3^- over the seventeen hour collection period are listed in Table VI. Bicarbonate treatment means ranged from a low of 26.95 mmol/L for horses on the low diet to a high of 31.43 mmol/L for horses on the high diet. Results of venous blood HCO_3^- (Figure 4) showed that horses consuming the low diet had significantly lower ($p<0.05$) mean venous HCO_3^- levels as compared to those horses consuming the medium high and high diets at all measured intervals. The decrease in HCO_3^- indicates a metabolic acidosis, and may be directly related to the drop in blood pH. This is probably due to chloride absorption in the gastrointestinal tract. Bicarbonate levels dropped post feeding in all treatments. Horses consuming the medium high and high diets bicarbonate values returned to

TABLE V

EFFECT OF DIETARY CATION-ANION BALANCE
ON VENOUS BLOOD pO_2 (mmHg)
IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	41.50[b]	42.25[b]	44.37[b]	42.00[b]	2.22
11 am	36.50[b]	43.87[c]	38.00[bc]	44.50[c]	2.36
12 pm	46.37[b]	45.75[b]	45.25[b]	43.37[b]	2.18
1 pm	47.37[bc]	46.00[bc]	47.87[b]	42.50[c]	1.76
2 pm	45.87[b]	47.25[b]	47.62[b]	43.00[b]	1.62
3 pm	45.25[b]	44.00[b]	46.37[b]	44.75[b]	2.19
4 pm	46.62[b]	44.00[b]	43.50[b]	42.75[b]	1.88
5 pm	46.75[b]	45.87[bc]	45.25[bc]	42.37[c]	1.45
6 pm	46.37[b]	44.37[b]	45.25[b]	43.50[b]	1.77
7 pm	46.12[b]	47.25[b]	47.12[b]	44.75[b]	1.41
8 pm	45.75[b]	43.62[b]	44.62[b]	46.50[b]	1.60
9 pm	43.12[b]	38.75[b]	44.37[b]	43.87[b]	1.58
10 pm[a]	41.37[b]	42.25[b]	43.50[b]	41.12[b]	1.29
11 pm	35.37[b]	38.12[b]	37.62[b]	35.50[b]	2.02
12 am	43.25[b]	44.87[b]	43.50[b]	44.75[b]	2.05
1 am	48.25[b]	45.50[b]	47.12[b]	47.12[b]	1.51
2 am	45.50[b]	44.62[b]	44.50[b]	43.12[b]	1.60

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

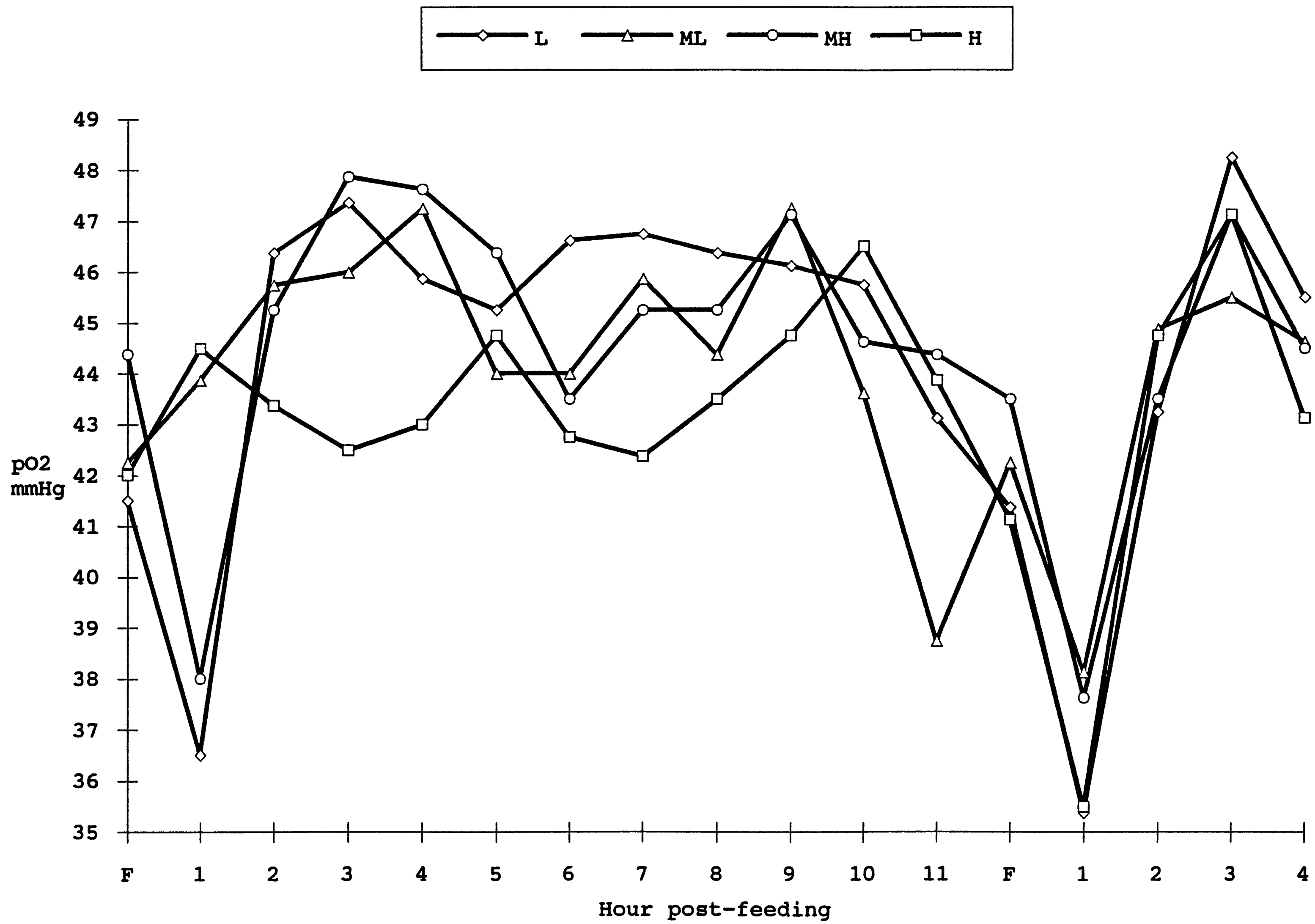

Figure 3. Effect of Dietary Cation-Anion Balance on Venous Blood pO_2 Post Feeding

TABLE VI

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD HCO_3^- (mmol/L) POST FEEDING IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	29.48[b]	31.12[c]	31.23[c]	30.92[b]	0.31
11 am	28.86[b]	29.53[b]	31.06[c]	30.91[c]	0.33
12 pm	27.36[b]	28.73[c]	29.037	29.85[d]	0.25
1 pm	26.95[b]	28.52[c]	29.00[c]	30.23[d]	0.28
2 pm	27.28[b]	28.70[c]	29.27[c]	29.77[d]	0.35
3 pm	27.27[b]	28.71[c]	29.55[c]	30.36[d]	0.53
4 pm	27.57[b]	29.22[c]	30.50[c]	30.90[d]	0.71
5 pm	27.66[b]	29.05[b]	30.47[c]	30.76[c]	0.56
6 pm	28.18[b]	29.32[b]	30.42[c]	30.72[c]	0.47
7 pm	28.60[b]	29.41[b]	30.58[c]	31.43[d]	0.51
8 pm	28.46[b]	29.87[b]	30.90[c]	31.20[c]	0.56
9 pm	29.03[b]	30.57[b]	30.80[c]	31.20[c]	0.47
10 pm[a]	29.60[b]	29.97[b]	30.82[c]	31.05[c]	0.40
11 pm	29.48[b]	29.87[b]	31.17[c]	31.28[d]	0.46
12 am	28.15[b]	28.81[b]	30.36[c]	29.60[c]	0.43
1 am	27.91[b]	28.82[b]	29.95[c]	29.55[c]	0.37
2 am	28.13[b]	29.27[c]	29.97[c]	30.58[d]	0.40

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

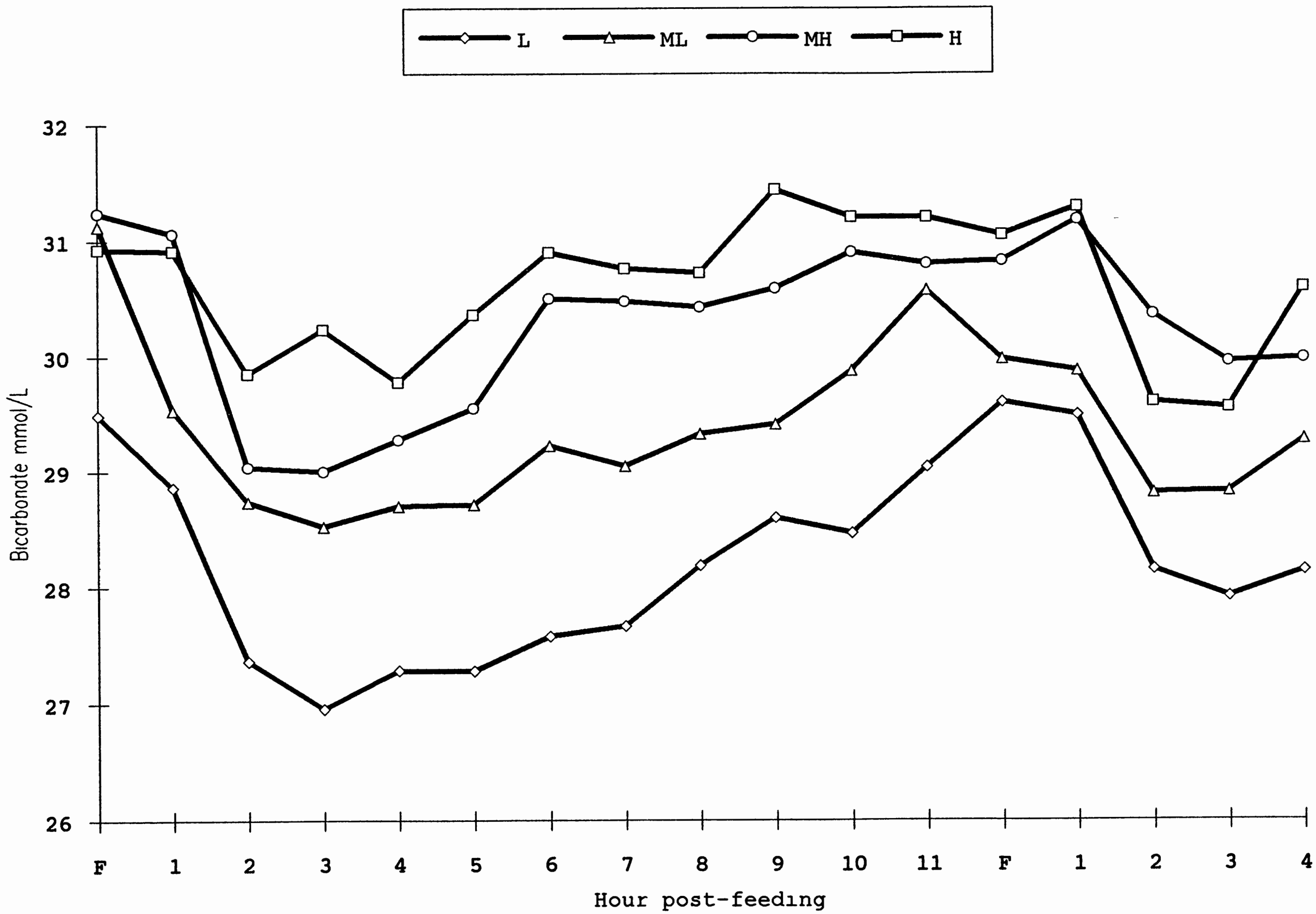

Figure 4. Effect of Dietary Cation-Anion Balance on Venous Blood HCO_3^- Post Feeding

pre-feeding levels between hour 6 and 7 post feeding, where as bicarbonate values in horses consuming the low and medium low diets never did return to pre-feeding levels.

Total carbon dioxide (tCO_2) is the total concentration of carbon dioxide (free + bound) in the blood. Treatment means for tCO_2 in venous blood for exercising horses ranged from 28.374 mmol/l on the low diet to 32.925 mmol/L on the high diet (Table VII and Figure 5). There were significant differences ($p<0.05$) in tCO_2 in horses on the low diet compared to horses on the medium high diet at all measured intervals. Horses on the low diet had significantly lower tCO_2 levels compared to horses on the high diet at sixteen of the seventeen measured intervals.

Base Excess is an indicator of the overall buffering capacity of the blood. Mean base excess in exercising horses ranged from 1.612 mmol/L for horses on the low diet to 5.975 mmol/L for horses on the high diet (Table VIII). Base excess in the blood decreased immediately post feeding in all dietary treatments; indicating removal of base from the blood. Base excess in horses on the low diet was significantly lower ($p<0.05$) at the morning and evening feedings as compared to the medium low, medium high, and high diets. Horses consuming the medium high and high diets had significantly higher ($p<0.05$) levels of base excess as compared to horses consuming the low diet at all measured intervals (Figure 6).

TABLE VII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD TOTAL CO_2 (mmol/L) IN EXERCISING HORSES

Time	TREATMENT				
	L	ML	MH	H	S.E.
10 am[a]	30.95[b]	32.65[c]	32.86[c]	32.45[bc]	0.56
11 am	30.45[b]	31.08[b]	32.71[c]	32.53[c]	0.34
12 pm	28.83[b]	30.23[c]	30.53[cd]	31.37[d]	0.35
1 pm	28.37[b]	30.02[c]	30.50[c]	31.77[d]	0.26
2 pm	28.72[b]	30.16[c]	30.77[cd]	31.26[d]	0.28
3 pm	28.72[b]	30.20[c]	31.07[cd]	31.90[d]	0.36
4 pm	29.00[b]	30.70[c]	32.07[cd]	32.47[d]	0.56
5 pm	29.10[b]	30.56[bc]	31.37[c]	32.32[c]	0.62
6 pm	29.65[b]	30.81[bc]	31.93[c]	32.26[c]	0.58
7 pm	30.16[b]	30.90[bc]	32.12[cd]	33.00[d]	0.47
8 pm	29.91[b]	31.31[bc]	32.45[c]	32.76[c]	0.54
9 pm	30.50[b]	31.97[bc]	32.33[c]	32.75[c]	0.59
10 pm[a]	31.07[b]	31.43[bc]	32.33[c]	32.58[c]	0.42
11 pm	31.12[b]	31.47[bc]	32.86[cd]	32.92[d]	0.49
12 am	29.66[b]	30.28[bce]	31.91[d]	31.10[de]	0.45
1 am	29.38[b]	30.30[bc]	31.41[c]	31.02[c]	0.39
2 am	29.60[b]	30.78[bc]	31.45[cd]	32.13[d]	0.42

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

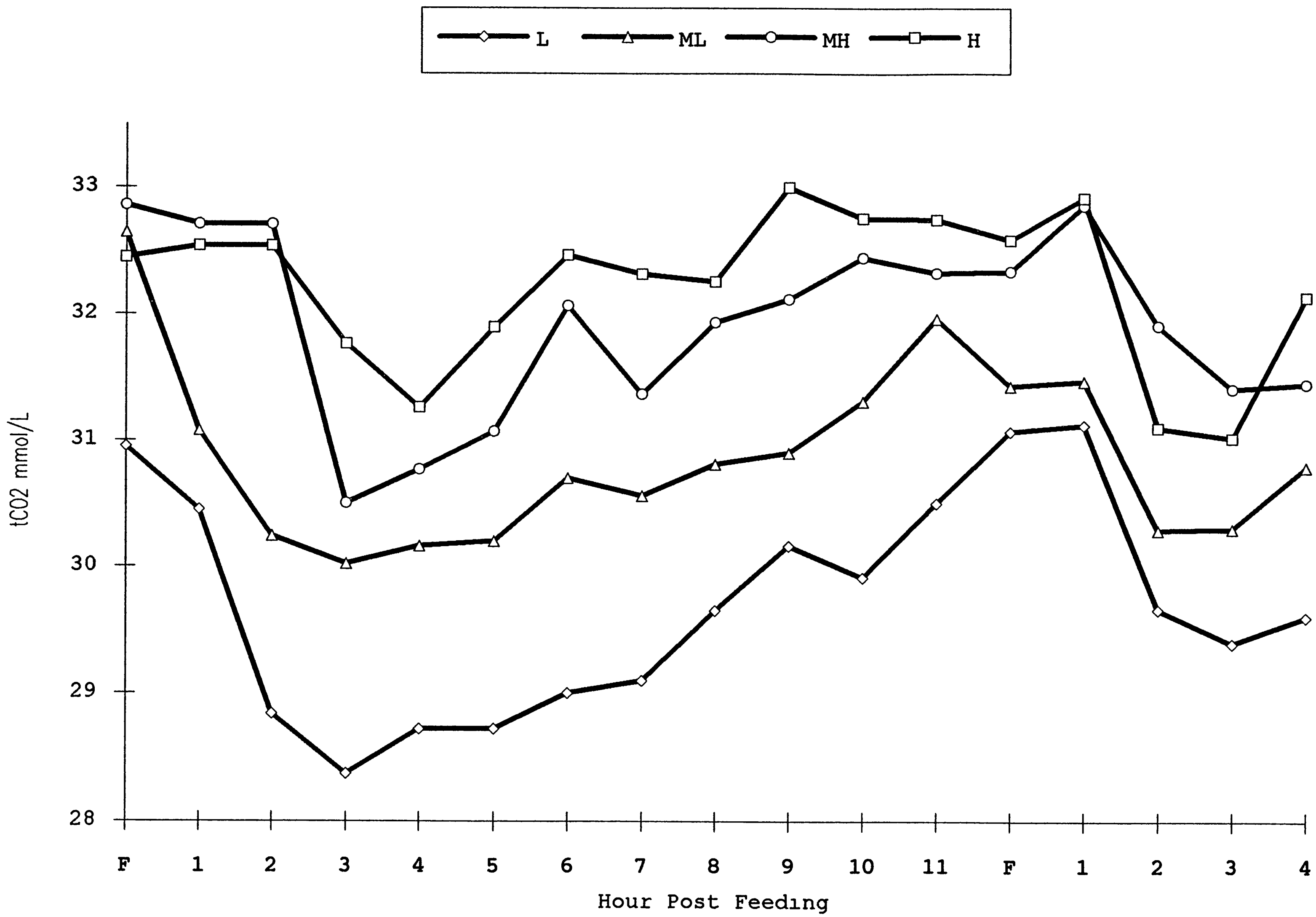

Figure 5. Effect of Dietary Cation-Anion Balance on Venous Blood Total CO_2 Post Feeding

TABLE VIII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD EXCESS BASE (mmol/L) IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	4.17^{b}	5.70^{c}	5.72^{c}	5.51^{c}	0.43
11 am	2.58^{b}	3.71^{c}	4.87^{de}	4.82^{e}	0.24
12 pm	1.68^{b}	3.31^{c}	3.48^{c}	4.32^{d}	0.24
1 pm	1.61^{b}	3.17^{c}	3.52^{c}	4.72^{d}	0.20
2 pm	1.85^{b}	3.38^{c}	3.75^{cd}	4.31^{d}	0.28
3 pm	1.88^{b}	3.35^{c}	4.01^{cd}	4.67^{d}	0.36
4 pm	2.18^{b}	3.38^{c}	4.87^{cd}	5.28^{d}	0.41
5 pm	2.30^{b}	3.51^{bc}	4.40^{cd}	5.17^{d}	0.51
6 pm	2.76^{b}	3.87^{bc}	5.00^{c}	5.25^{c}	0.50
7 pm	3.22^{b}	4.18^{c}	5.06^{c}	5.97^{c}	0.42
8 pm	3.15^{b}	4.66^{bc}	5.45^{c}	5.71^{c}	0.46
9 pm	3.66^{b}	4.91^{bc}	5.33^{c}	5.52^{c}	0.44
10 pm[a]	4.37^{b}	4.73^{bc}	5.51^{c}	5.62^{c}	0.35
11 pm	3.21^{b}	3.98^{bd}	4.83^{c}	4.78^{cd}	0.37
12 am	2.41^{b}	3.36^{bd}	4.60^{c}	4.11^{cd}	0.39
1 am	2.47^{b}	3.45^{c}	4.71^{de}	4.26^{ce}	0.33
2 am	2.71^{b}	3.73^{c}	4.60^{cd}	4.97^{d}	0.36

[a]Indicates feeding time.

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

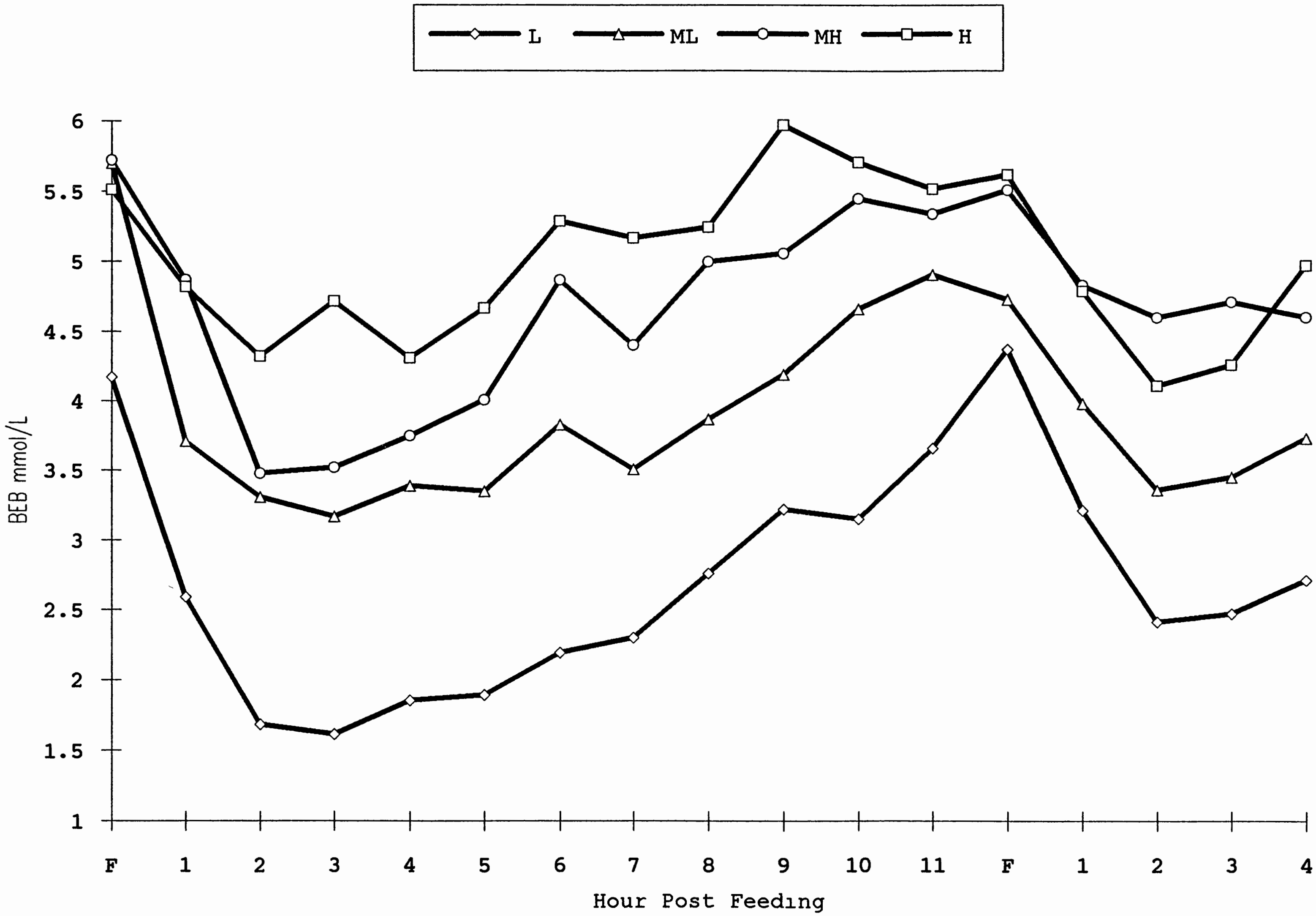

Figure 6. Effect of Dietary Cation-Anion Balance on Venous Blood Excess Base Post Feeding

There were significant differences in standard bicarbonate and base excess extracellular fluid ($p<0.05$) between horses on the low diet and horses on the medium high and high diet at all measured intervals. Treatment means for standard bicarbonate ranged from 25.637 mmol/L in horses consuming the low diet to 29.050 mmol/L in horses consuming the high diet (Table IX and Figure 7). Base excess extracellular fluid treatment means ranged from 1.1437 mmol/L in horses on the low diet to 6.462 mmol/L in horses on the high diet (Table X and Figure 8). Treatment means for percent oxygen saturation are shown in Table XI. Means ranged from 63.075% in horses on the high diet. Values for all treatments are shown graphically in (Figure 9).

<u>STANDARD EXERCISE TEST</u>

<u>Blood Acid-Base Status.</u> During strenuous exercise, blood pH, bicarbonate and pCO_2 decreased across all treatments producing an incompletely compensated metabolic acidosis. This is in agreement with Milne (1974). The treatment means for venous blood pH at time 0 post exercise in horses performing anaerobic work ranged from 7.147 in horses on the low diet to 7.201 in horses consuming the high diet. At 30 minutes post exercise means ranged from 7.299 for the low diet to 7.361 for the high diet (Table XII). Although treatment did not significantly affect measures of blood pH in horses performing anaerobic work in

TABLE IX

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD STANDARD HCO_3^- (mmol/L) IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	27.50[b]	28.68[c]	28.75[c]	28.58[c]	0.29
11 am	25.95[b]	27.16[c]	27.85[de]	28.03[e]	0.21
12 pm	25.63[b]	27.00[c]	27.13[c]	27.71[d]	0.20
1 pm	25.67[b]	26.90[c]	27.18[c]	28.02[d]	0.18
2 pm	25.92[b]	27.10[c]	27.36[c]	27.66[c]	0.22
3 pm	25.85[b]	26.96[c]	27.52[cd]	28.00[d]	0.26
4 pm	26.16[b]	27.32[c]	28.10[cd]	28.48[d]	0.31
5 pm	26.18[b]	27.20[bc]	27.82[cd]	28.35[d]	0.39
6 pm	26.55[b]	27.33[bc]	28.31[c]	28.46[c]	0.40
7 pm	26.82[b]	27.75[bc]	28.45[cd]	29.05[d]	0.35
8 pm	26.86[b]	28.00[c]	28.65[c]	28.85[c]	0.34
9 pm	27.17[b]	27.96[bc]	28.52[c]	28.65[c]	0.32
10 pm[a]	27.71[b]	28.00[bc]	28.66[c]	28.62[c]	0.28
11 pm	26.43[b]	27.07[bc]	27.71[c]	27.65[c]	0.28
12 am	26.13[b]	27.03[c]	27.87[d]	27.68[cd]	0.29
1 am	26.38[b]	27.18[c]	28.16[d]	27.78[cd]	0.24
2 am	26.52[b]	27.26[bc]	28.02[cd]	28.17[d]	0.28

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

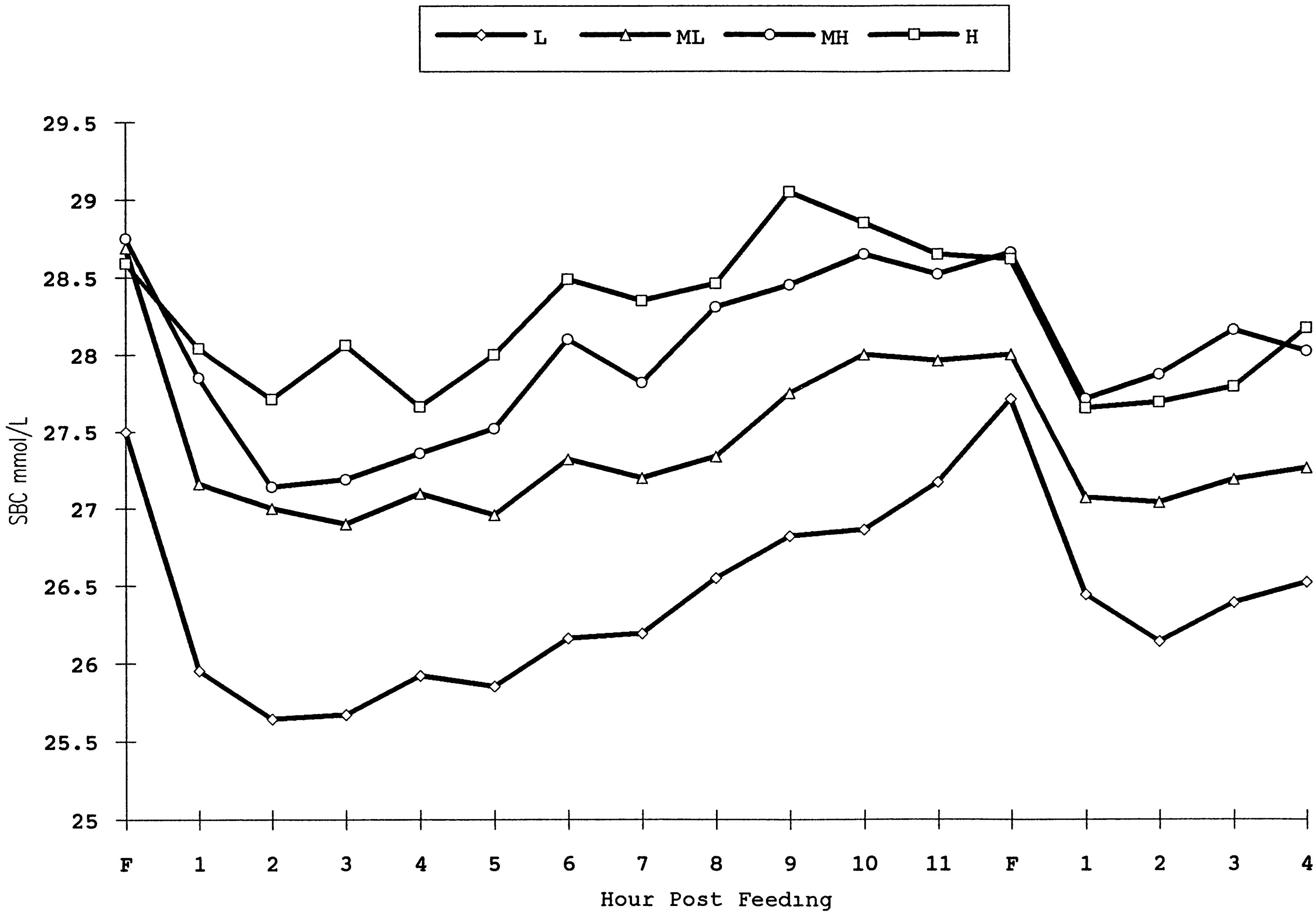

Figure 7. Effect of Dietary Cation-Anion Balance on Venous Blood Standard HCO_3^- Post Feeding

TABLE X

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD EXCESS EXTRACELLULAR FLUID (mmol/L) IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	4.33[b]	6.12[c]	6.25[c]	5.93[c]	0.53
11 am	2.95[b]	4.03[c]	5.51[d]	5.42[d]	0.30
12 pm	1.63[b]	3.42[c]	3.66[c]	4.62[d]	0.31
1 pm	1.43[b]	3.22[c]	3.68[c]	5.06[d]	0.25
2 pm	1.75[b]	3.40[c]	3.95[cd]	4.57[d]	0.32
3 pm	1.77[b]	3.42[c]	4.25[cd]	5.08[d]	0.41
4 pm	2.08[b]	3.98[c]	5.28[cd]	5.73[d]	0.52
5 pm	2.21[b]	3.67[bc]	4.66[cd]	5.61[d]	0.61
6 pm	2.77[b]	4.06[bc]	5.36[c]	5.65[c]	0.59
7 pm	3.23[b]	4.32[bc]	5.46[cd]	6.46[d]	0.50
8 pm	3.18[b]	4.76[bc]	5.87[c]	6.17[c]	0.56
9 pm	3.77[b]	5.30[bc]	5.76[c]	6.05[c]	0.55
10 pm[a]	4.52[b]	4.95[bc]	5.86[c]	6.03[c]	0.42
11 pm	3.67[b]	4.36[bc]	5.55[c]	5.52[c]	0.44
12 am	2.53[bc]	3.45[ce]	4.91[d]	4.35[de]	0.47
1 am	2.48[bc]	3.57[be]	4.92[d]	4.45[de]	0.38
2 am	2.72[bc]	3.95[cd]	4.86[de]	5.38[e]	0.42

[a]Indicates feeding time.
[b,c,d,e]Means in row with different superscripts differ ($p<.05$).

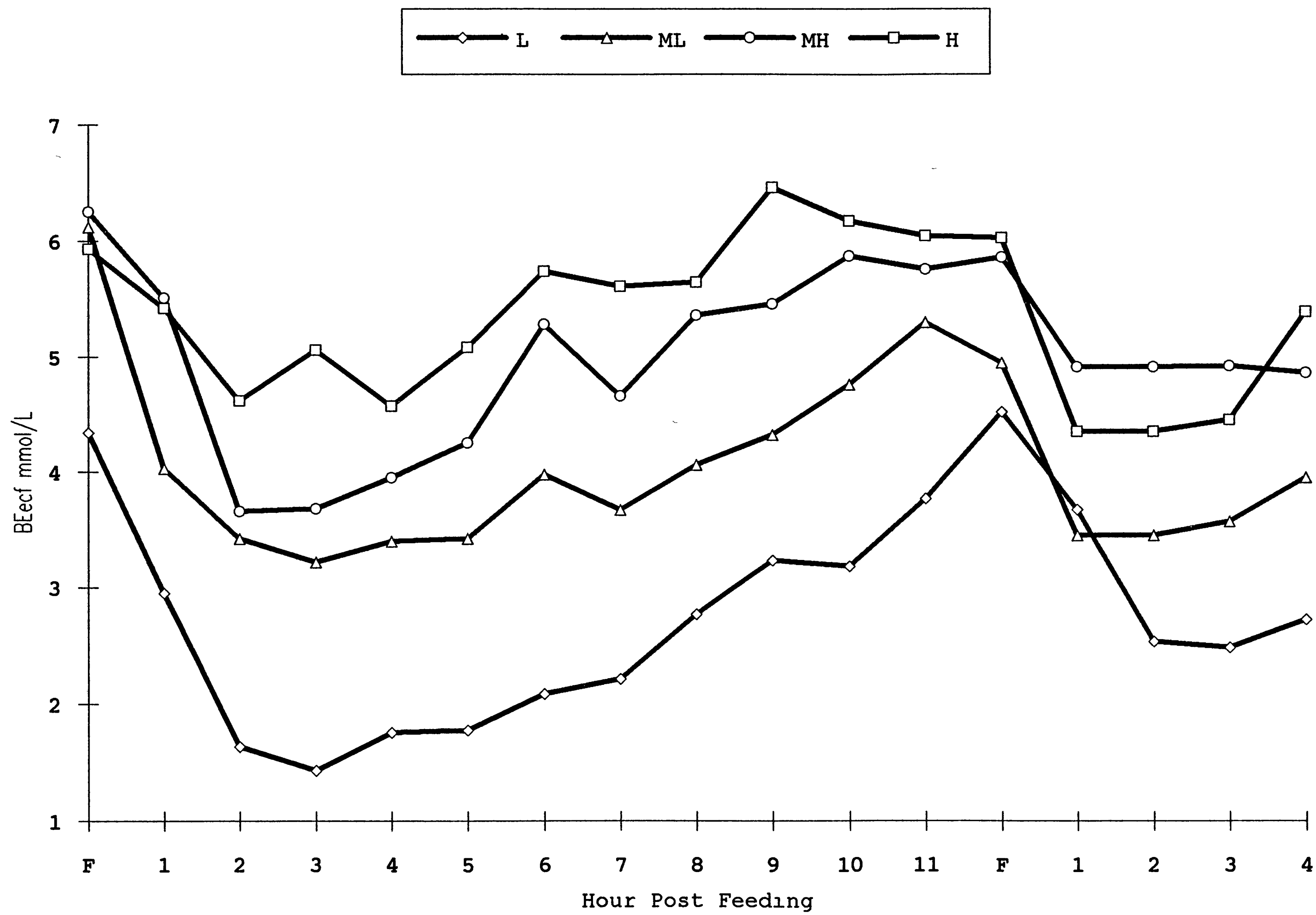

Figure 8. Effect of Dietary Cation-Anion Balance on Venous Blood Base Excess Extracellular Fluid Post Feeding

TABLE XI

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD %O_2 SATURATION (mmol/L) IN EXERCISING HORSES

	TREATMENT				
Time	L	ML	MH	H	S.E.
10 am[a]	74.32[b]	74.35[b]	75.52[b]	75.66[b]	3.07
11 am	63.28[b]	74.81[cd]	68.08[bcd]	75.23[b]	2.97
12 pm	77.54[b]	79.42[b]	78.48[b]	77.10[b]	2.73
1 pm	80.68[b]	79.72[b]	81.16[b]	75.81[b]	2.30
2 pm	78.53[bc]	81.61[b]	80.15[bc]	76.70[c]	1.62
3 pm	78.27[b]	77.38[b]	79.27[b]	77.57[b]	2.25
4 pm	80.43[b]	76.41[b]	76.60[b]	76.26[b]	2.62
5 pm	79.92[b]	79.17[b]	79.46[b]	76.07[b]	1.57
6 pm	79.38[b]	77.12[b]	79.77[b]	77.76[b]	1.86
7 pm	79.07[b]	81.38[b]	81.67[b]	79.36[b]	1.34
8 pm	79.96[b]	78.11[b]	78.95[b]	79.21[b]	1.51
9 pm	77.07[b]	65.48[b]	78.48[b]	77.08[b]	4.70
10 pm[a]	75.58[b]	76.73[b]	78.05[b]	74.58[b]	1.71
11 pm	63.08[b]	69.02[b]	67.05[b]	63.03[b]	3.96
12 am	72.98[b]	78.35[b]	76.00[b]	78.15[b]	2.72
1 am	81.30[b]	79.11[b]	81.20[b]	81.25[b]	1.41
2 am	78.83[b]	78.47[b]	78.46[b]	76.38[b]	1.71

[a]Indicates feeding time.

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

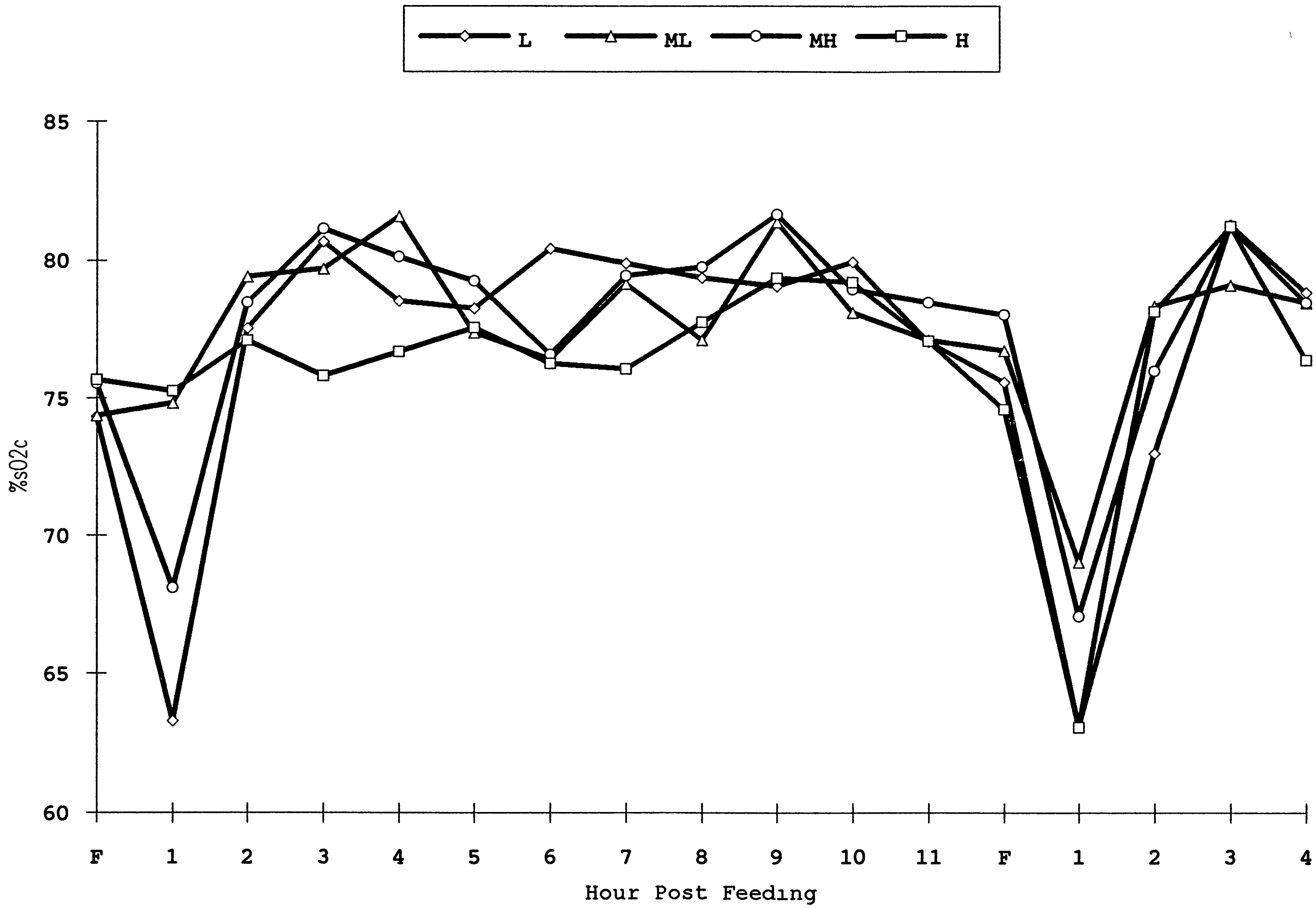

Figure 9. Effect of Dietary Cation-Anion Balance on $\%O_2$ Saturation in Venous Blood Post Feeding

TABLE XII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pH IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	7.380[a]	7.394[a]	7.380[a]	7.393[a]	.012
0	7.148[a]	7.175[a]	7.190[a]	7.201[a]	.024
1	7.158[a]	7.184[a]	7.203[a]	7.223[a]	.023
2	7.163[a]	7.167[a]	7.193[a]	7.200[a]	.024
3	7.154[a]	7.173[a]	7.189[a]	7.202[a]	.027
4	7.147[a]	7.174[a]	7.185[a]	7.202[a]	.030
5	7.149[a]	7.181[a]	7.188[a]	7.205[a]	.032
10	7.175[a]	7.198[a]	7.213[a]	7.244[a]	.062
20	7.251[a]	7.280[a]	7.282[a]	7.312[a]	.034
30	7.299[a]	7.338[a]	7.329[a]	7.361[a]	.026

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

experiment, the data do show a trend in buffering capacity (Figure 10).

The treatment means for venous blood pCO_2 are shown in Table XIII. There was a sharp decrease in pCO_2 at time 0 and 1 minute post exercise in horses performing anaerobic work. The decrease is probably due to hyperventilation occurring during the SET (Figure 11). At the same time there was a reciprocal rise in pO_2 (Figure 12) due to increased alveolar ventilation. Treatment means for venous blood pO_2 are shown in Table XIV. There were no significant differences in pO_2 between treatments, however significance was detected at 30 minutes between the low diet and medium high diet.

Treatment means for venous blood bicarbonate in horses performing anaerobic work are shown in Table XV. The means ranged from 13.900 mmol/L at 5 minutes post exercise in horses consuming the low diet to 24.637 mmol/L at 30 minutes post exercise in horses consuming the high diet. With strenuous exercise there is a marked decrease in bicarbonate, indicating a metabolic acidosis (Figure 13). This decrease is due to bicarbonate assisting with the neutralization of hydrogen ions and lactic acid that is produced during maximal exercise. There was a significant difference at time 0 in bicarbonate between treatments in horses performing anaerobic work. There is a trend that the high diet may be better able to assist in buffering

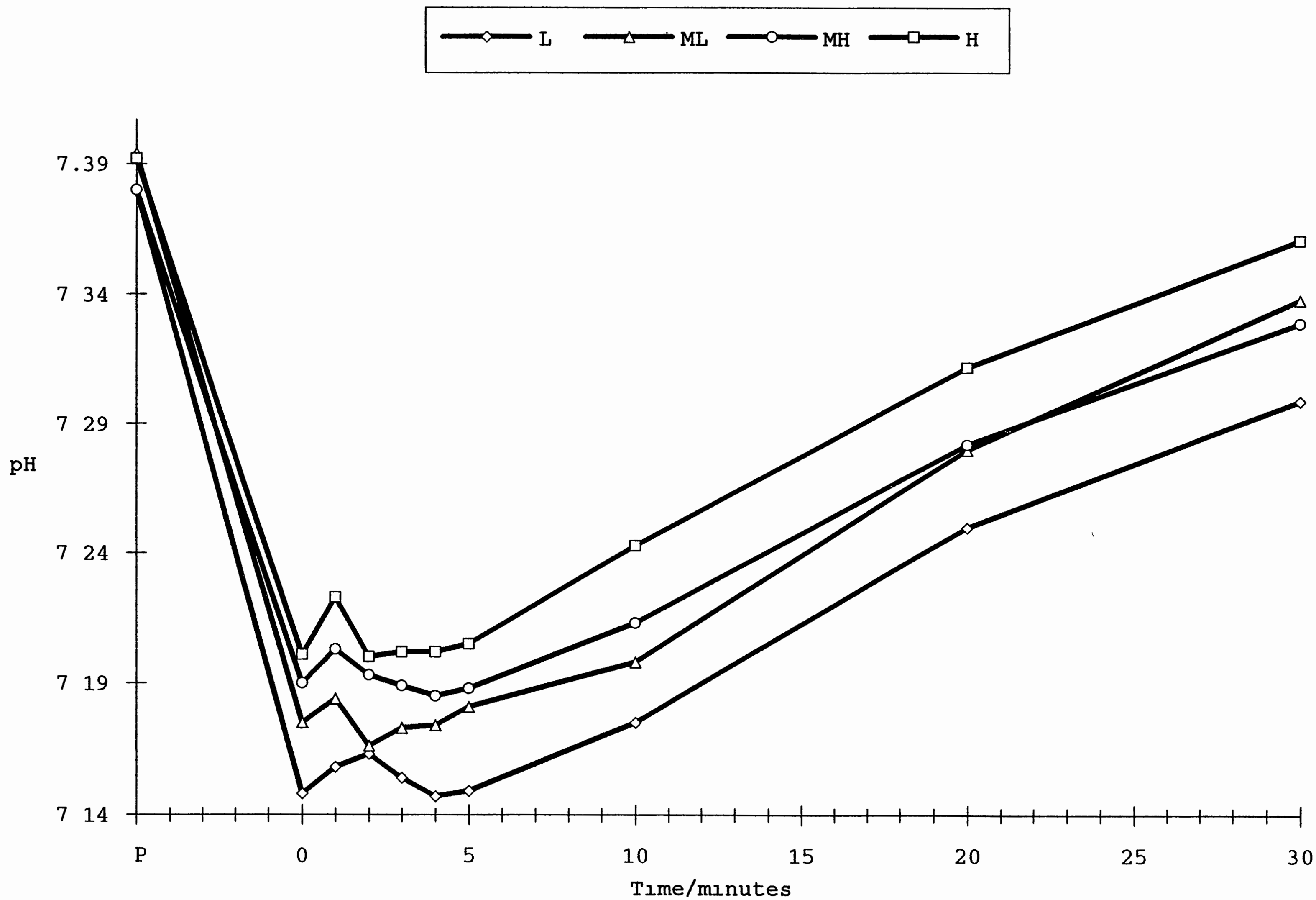

Fıgure 10. Effect of Dıetary Catıon-Anıon Balance on Venous Blood pH ın Anaerobıcally Exercısed Horses

TABLE XIII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pCO_2 (mmHg) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	47.87[a]	49.20[a]	48.57[a]	49.46[a]	0.95
0	45.65[a]	41.41[a]	48.33[a]	46.63[a]	2.51
1	40.77[a]	37.58[a]	40.25[a]	39.62[a]	1.39
2	37.97[a]	39.80[a]	40.13[a]	40.05[a]	1.31
3	38.00[a]	38.01[a]	40.72[a]	40.36[a]	1.23
4	39.16[a]	37.72[a]	39.67[a]	39.45[a]	0.89
5	38.05[a]	37.31[a]	39.26[a]	39.71[a]	1.06
10	36.15[a]	37.85[a]	39.50[a]	39.45[a]	1.28
20	38.11[a]	38.42[a]	39.53[a]	41.10[a]	1.28
30	39.62[a]	40.10[ac]	43.43[bd]	42.58[cd]	1.05

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

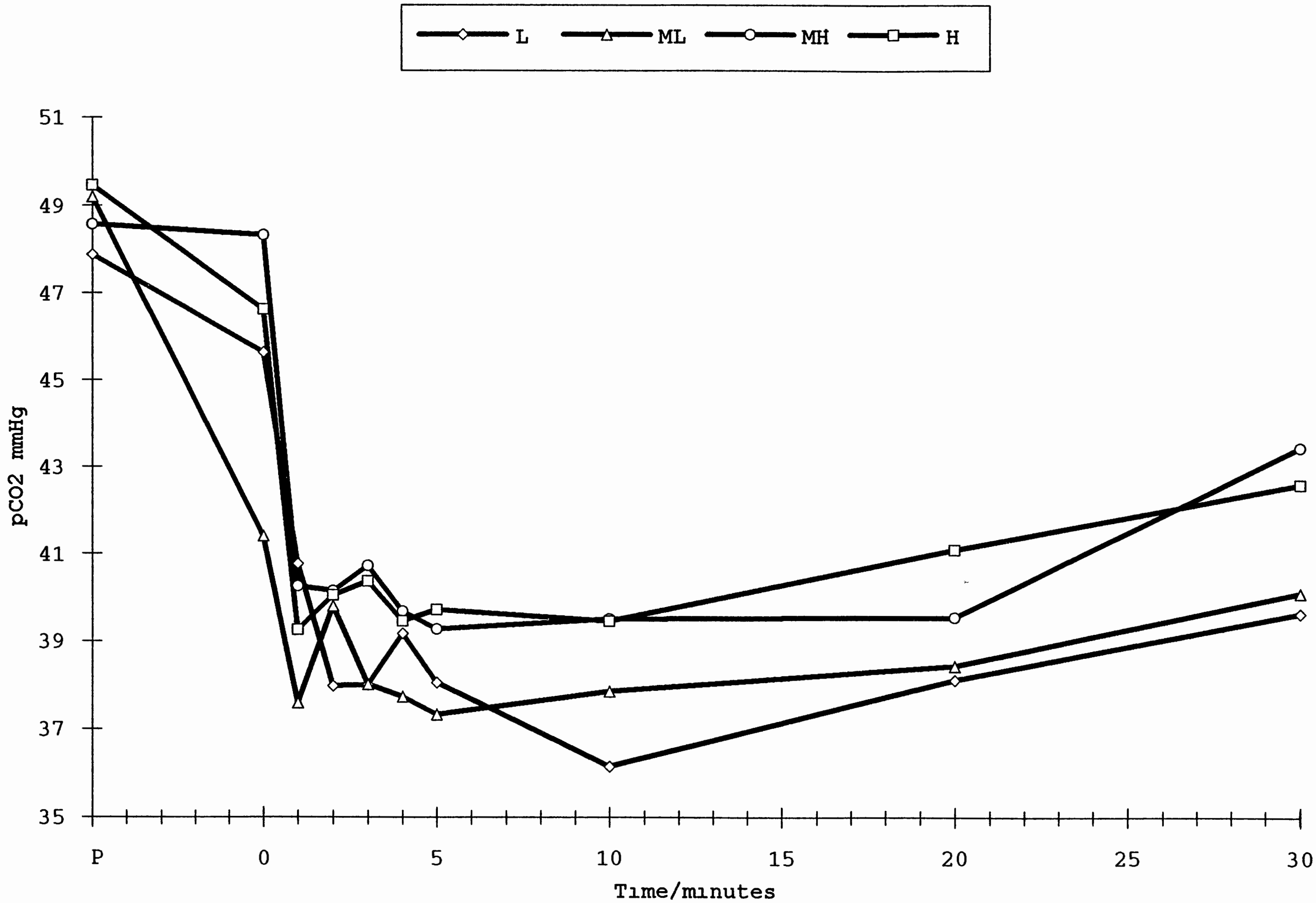

Figure 11. Effect of Dietary Cation-Anion Balance on Venous Blood pCO_2 in Anaerobically Exercised Horses

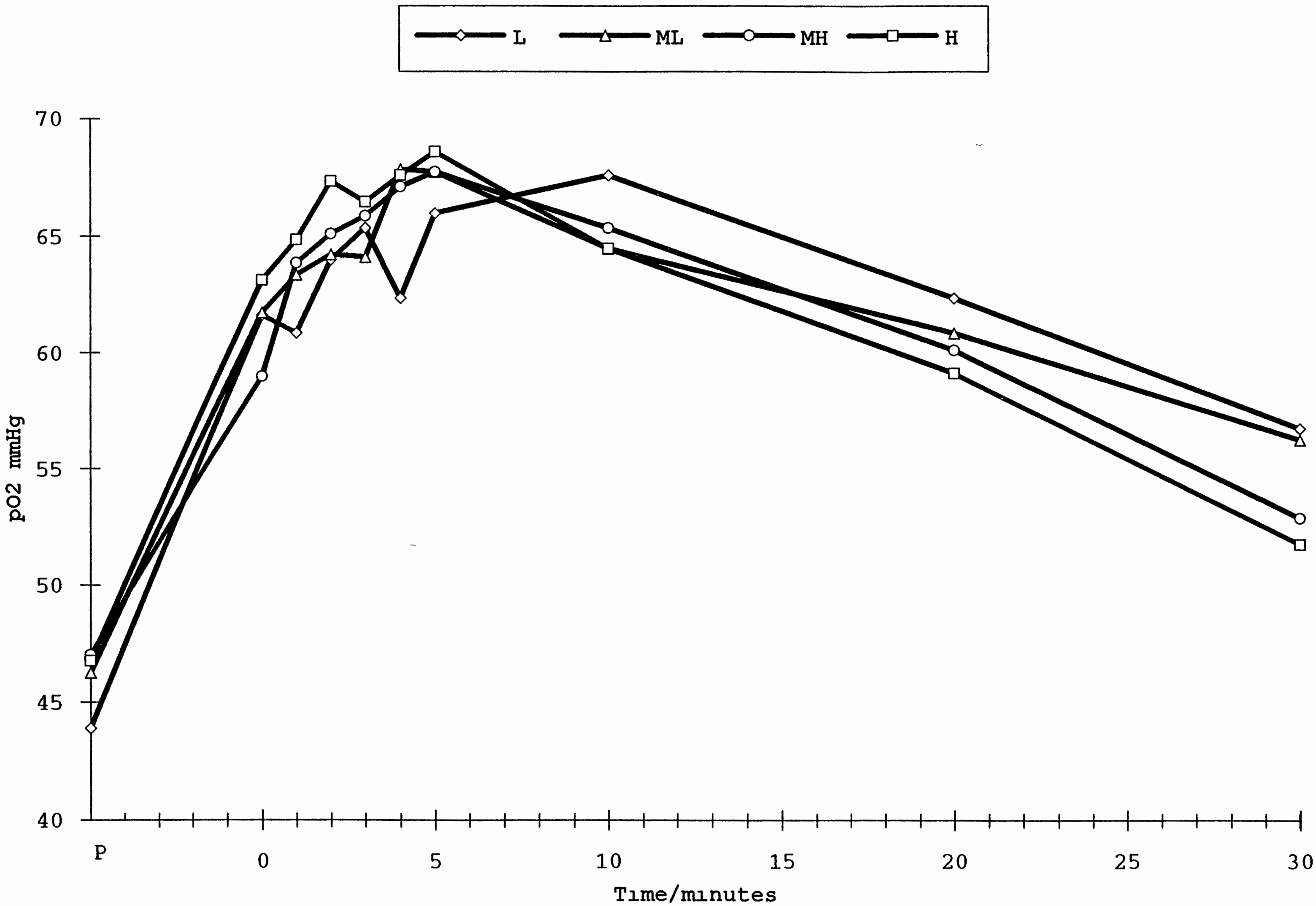

Figure 12. Effect of Dietary Cation-Anion Balance on Venous Blood pO_2 in Anaerobically Exercised Horses

TABLE XIV

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD pO_2 (mmHg) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	43.87[a]	46.25[a]	47.00[a]	46.75[a]	2.55
0	61.62[a]	61.75[a]	59.00[a]	63.12[a]	2.10
1	60.87[a]	63.37[a]	63.87[a]	64.87[a]	2.11
2	64.00[a]	64.25[a]	65.12[a]	67.37[a]	2.43
3	65.37[a]	64.12[a]	65.87[a]	66.50[a]	2.83
4	62.37[a]	67.87[a]	67.12[a]	67.62[a]	2.56
5	66.00[a]	67.75[a]	67.75[a]	68.62[a]	2.85
10	67.62[a]	64.50[a]	65.37[a]	64.50[a]	3.13
20	62.37[a]	60.87[a]	60.12[a]	59.12[a]	2.73
30	56.75[a]	56.25[a]	52.87[a]	51.75[a]	2.44

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

TABLE XV

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD HCO_3^- (mmol/L) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	28.72[a]	30.31[ab]	29.77[ab]	30.41[b]	0.57
0	16.45[ac]	15.70[a]	18.58[b]	18.31[bc]	0.72
1	14.77[a]	14.70[a]	16.25[a]	16.43[a]	0.86
2	14.07[a]	14.93[a]	15.92[a]	15.95[a]	1.07
3	14.00[a]	14.47[a]	16.08[a]	16.05[a]	1.08
4	14.15[a]	14.71[a]	15.67[a]	15.83[a]	1.06
5	13.90[a]	14.63[a]	15.67[a]	16.03[a]	1.16
10	14.42[a]	15.37[a]	16.85[a]	17.46[a]	1.49
20	17.98[a]	18.83[a]	19.90[a]	21.30[a]	1.73
30	20.52[a]	22.06[ab]	23.63[ab]	24.63[a]	1.53

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

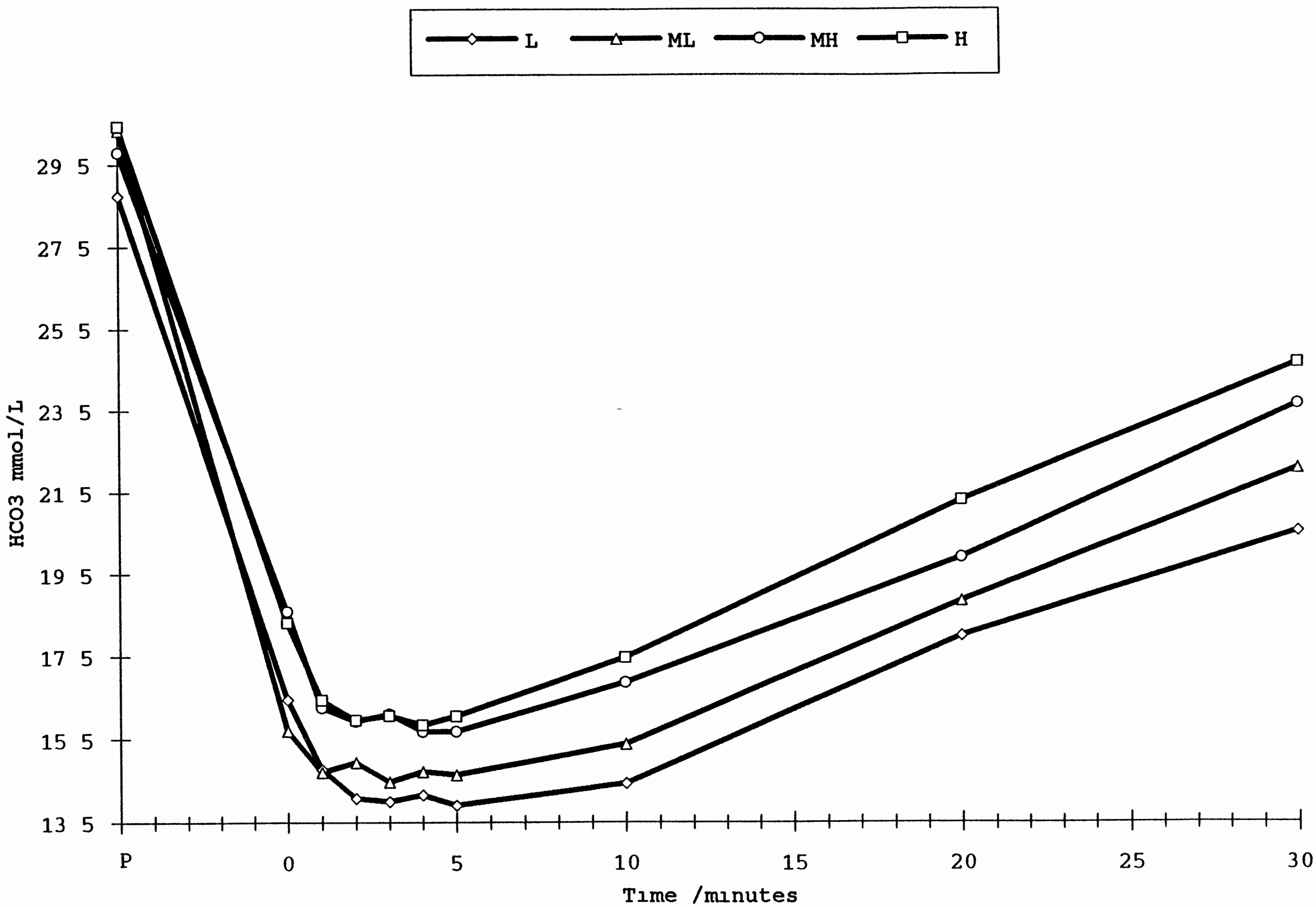

Figure 13. Effect of Dietary Cation-Anion Balance on Venous Blood HCO_3^- in Anaerobically Exercised Horses

systemic acids produced during exercise as compared to the low diet.

Treatment means for total CO_2 in horses performing anaerobic work are shown in Table XVI, and ranged from 15.06 mmol/L in horses on the low diet to 25.93 mmol/L at 30 minutes post exercise in horses on the high diet. Total CO_2 followed the same pattern as pCO_2, which is probably due to hyperventilation (Figure 14). There were significant differences between treatments pre-exercise and at time 0 for total CO_2.

Treatment means for base excess in horses performing anaerobic work are shown in (Table XVII). Means ranged from -13.70 mmol/L art 5 minutes post exercise in horses consuming the low diet to -0.33 mmol/L at 30 minutes post exercise in horses consuming the high diet. The overall buffering capacity of the blood is indicative of blood base excess. A negative base excess indicates acid in the blood or removal of base. Differences between treatments were not significant for base excess during the recovery phase in horses performing anaerobic exercise (Figure 15).

Standard bicarbonate followed the same pattern as bicarbonate by dropping sharply during the exercise bout (Figure 16). Treatment means ranged from 13.87 mmol/L at 5 minutes post exercise in horses on the low diet to 24.26 mmol/L at 30 minutes post exercise in horses on the high diet (Table XVIII). Base excess extracellular fluid followed the same pattern as base excess by dropping into

TABLE XVI

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD TOTAL CO_2 (mmol/L) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	30.17^{a}	31.82^{a}	31.42^{a}	31.95^{a}	0.60
0	17.88^{ac}	16.96^{a}	20.11^{b}	19.76^{bc}	0.75
1	16.02^{a}	15.88^{a}	17.63^{a}	17.66^{a}	0.89
2	15.23^{a}	16.15^{a}	17.57^{a}	17.18^{a}	1.01
3	15.16^{a}	15.63^{a}	17.47^{a}	17.30^{a}	1.11
4	15.33^{a}	15.63^{a}	16.88^{a}	17.05^{a}	1.04
5	15.06^{a}	15.78^{a}	16.88^{a}	17.27^{a}	1.18
10	15.53^{a}	16.53^{a}	18.07^{a}	18.67^{a}	1.52
20	19.16^{a}	20.01^{a}	21.11^{a}	22.56^{a}	1.77
30	21.75^{a}	23.30^{a}	24.96^{a}	25.93^{a}	1.58

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

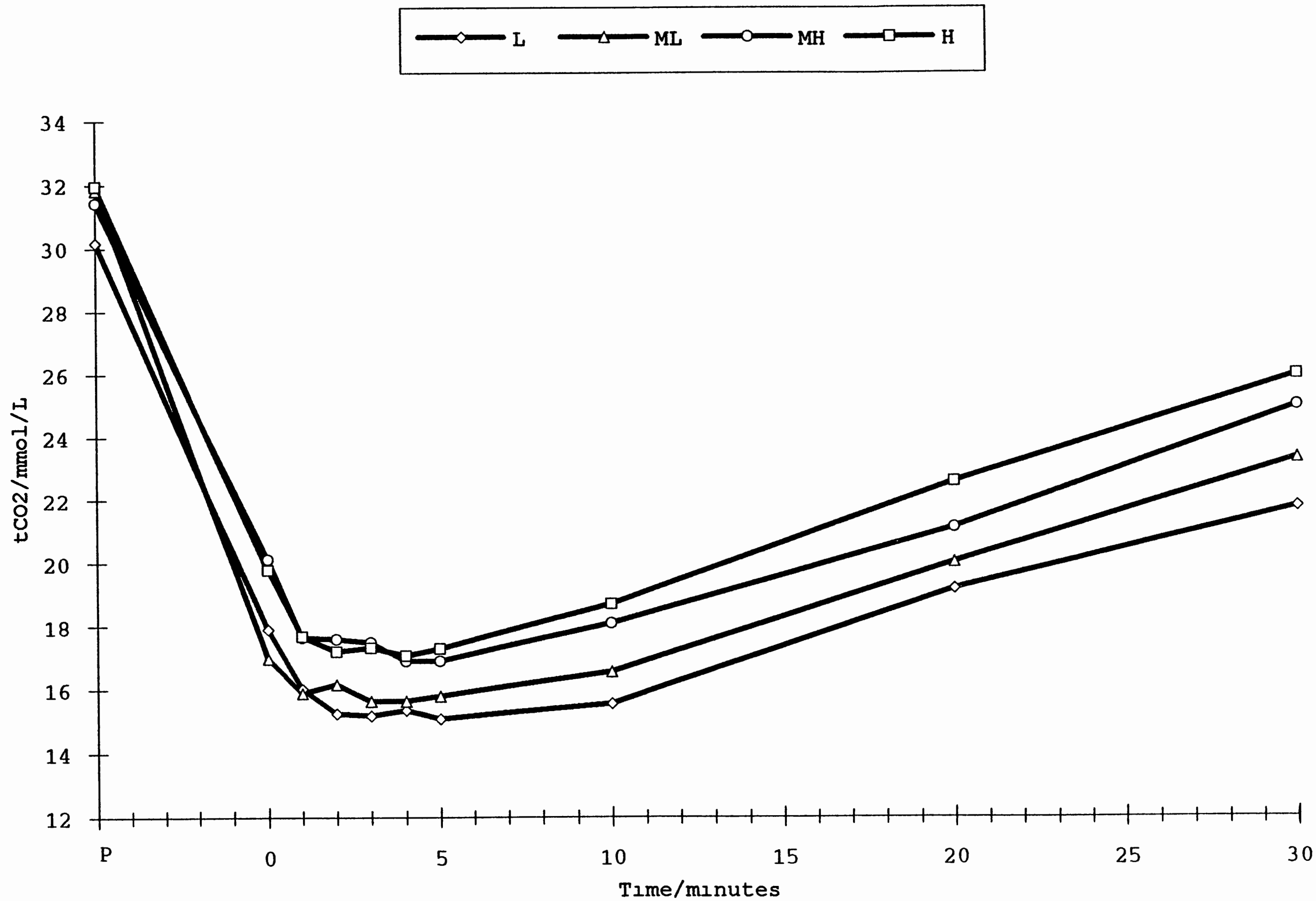

Figure 14. Effect of Dietary Cation-Anion Balance on Venous Blood Total CO_2 in Anaerobically Exercised Horses

TABLE XVII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD EXCESS BASE (mmol/L) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	3.33^{a}	4.83^{a}	4.81^{a}	4.97^{b}	0.52
0	-11.68^{a}	-11.70^{a}	-9.00^{a}	-9.00^{a}	0.95
1	-12.78^{a}	-12.27^{a}	-10.62^{a}	-9.98^{a}	1.15
2	-13.25^{a}	-12.47^{a}	-11.10^{a}	-10.91^{a}	1.36
3	-13.50^{a}	-12.71^{a}	-11.06^{a}	-10.77^{a}	1.44
4	-13.55^{a}	-12.67^{a}	-11.47^{a}	-10.95^{a}	1.48
5	-13.70^{a}	-12.387	-11.38^{a}	-10.73^{a}	1.62
10	-12.68^{a}	-11.41^{a}	-9.92^{a}	-8.71^{a}	1.94
20	-8.16^{a}	-6.80^{a}	-5.92^{a}	-4.12^{a}	2.13
30	-4.92^{a}	-2.91^{a}	-1.85^{a}	-0.33^{a}	1.81

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

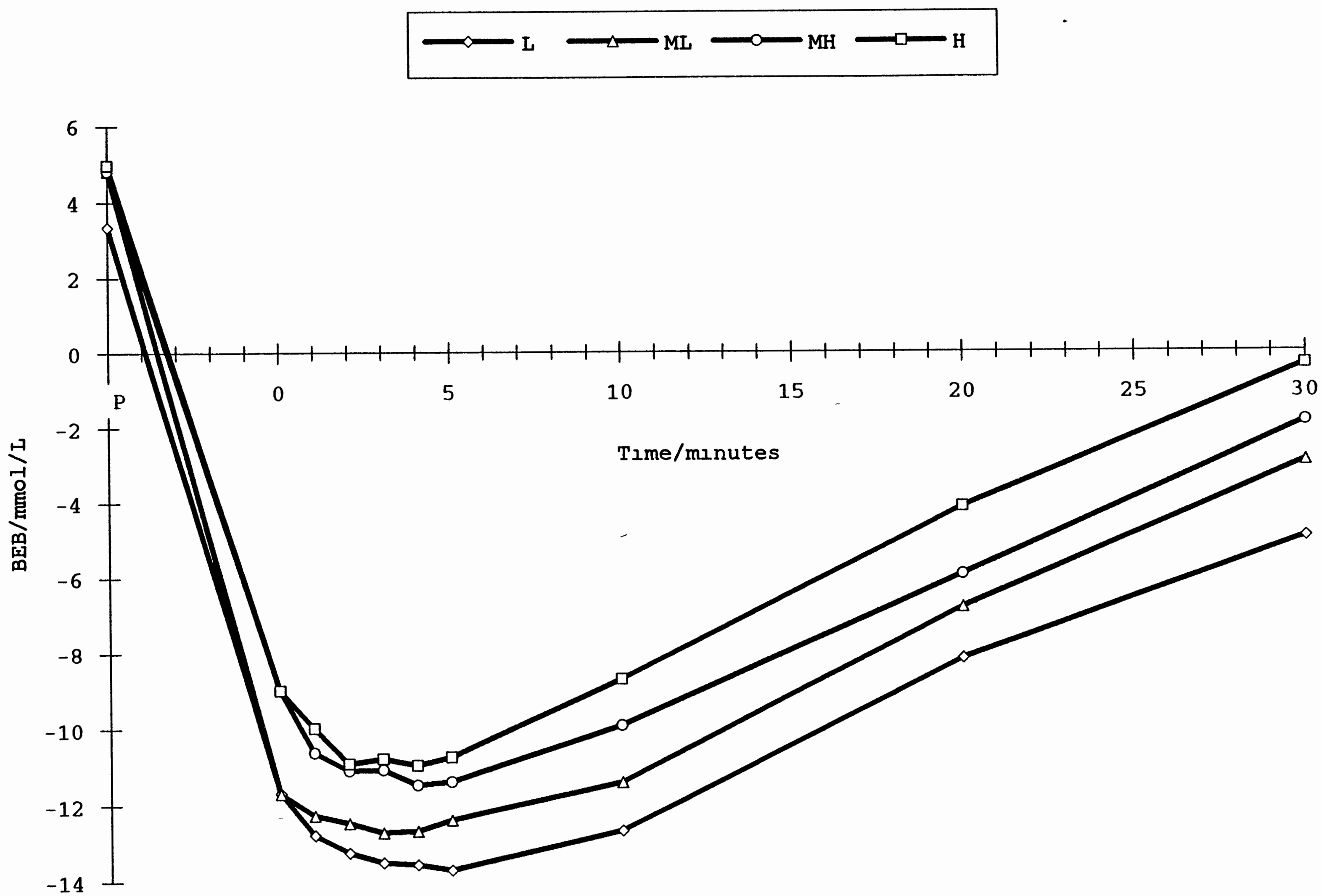

Figure 15. Effect of Dietary Cation-Anion Balance on Venous Blood Excess Base in Anaerobically Exercised Horses

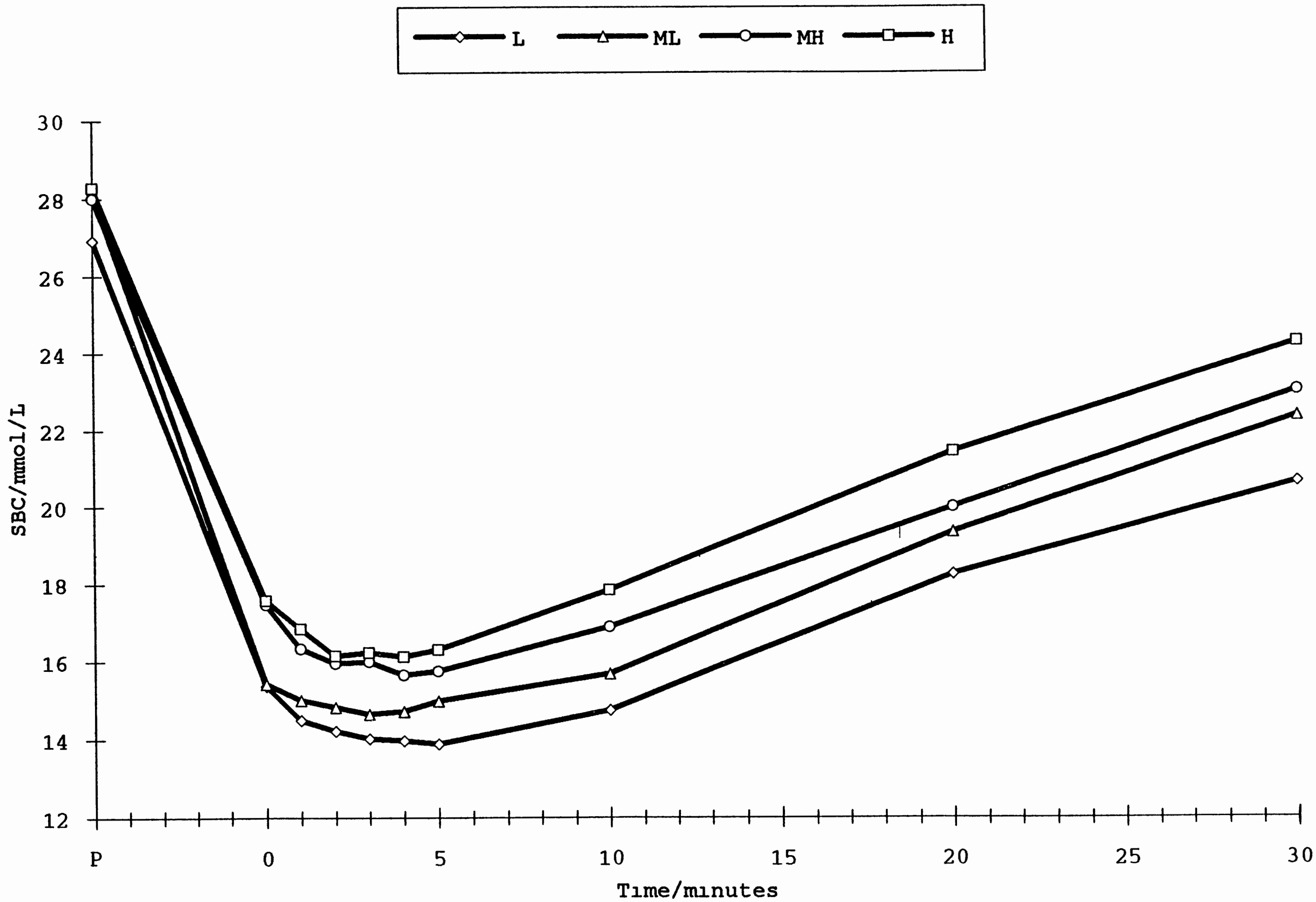

Figure 16. Effect of Dietary Cation-Anion Balance on Venous Blood Standard HCO_3^- in Anaerobically Exercised Horses

TABLE XVIII

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD STANDARD HCO_3^- (mmol/L) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	26.92[a]	28.17[b]	28.00[b]	28.28[b]	0.41
0	15.35[a]	15.41[ab]	17.45[ab]	17.56[b]	0.76
1	14.48[a]	15.00[a]	16.31[a]	16.82[a]	0.91
2	14.21[a]	14.81[a]	15.93[a]	16.13[a]	1.06
3	14.01[a]	14.63[a]	15.97[a]	16.22[a]	1.12
4	13.95[a]	14.71[a]	15.63[a]	16.11[a]	1.16
5	13.87[a]	14.97[a]	15.73[a]	16.28[a]	1.26
10	14.73[a]	15.66[a]	16.87[a]	17.82[a]	1.52
20	18.22[a]	19.32[a]	19.97[a]	21.42[a]	1.67
30	20.62[a]	22.32[a]	23.01[a]	24.26[a]	1.48

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

the negative range (Figure 17). Treatment means ranged from -15.12 mmol/L at 5 minutes post feeding in horses consuming the low diet to -0.97 at 30 minutes post exercise in horses consuming the high diet (Table XIX). There were significant differences between treatments for standard bicarbonate at pre-exercise between the low and high diets and base excess extracellular fluid at pre-exercise and at the end of exercise.

The treatment means for percent oxygen saturation in venous blood in horses performing anaerobic work are shown in Table XX. Treatment means ranged from 77.57% for the low diet pre-exercise to 79.93% for the high diet pre-exercise. Treatment means post exercise test ranged from 82.27% on the low diet to 88.15% on the high diet. The rise in percent O_2 saturation is probably due to less extraction of O_2 due to a maximal exercise bout (Figure 18). There were no significant differences in percent oxygen saturation between treatments during the recovery phase of the standard exercise test.

Blood Glucose and Heart Rate. There were significant differences in plasma glucose concentrations (Figure 19) at 10 minutes post exercise between the low diet and the high diet. At 20 minutes post exercise, there was a significant difference between the low and medium low, and the medium high and high diets. At 30 minutes post exercise, there were significant differences between the low diet and the medium low, medium high and high diets. Treatment means

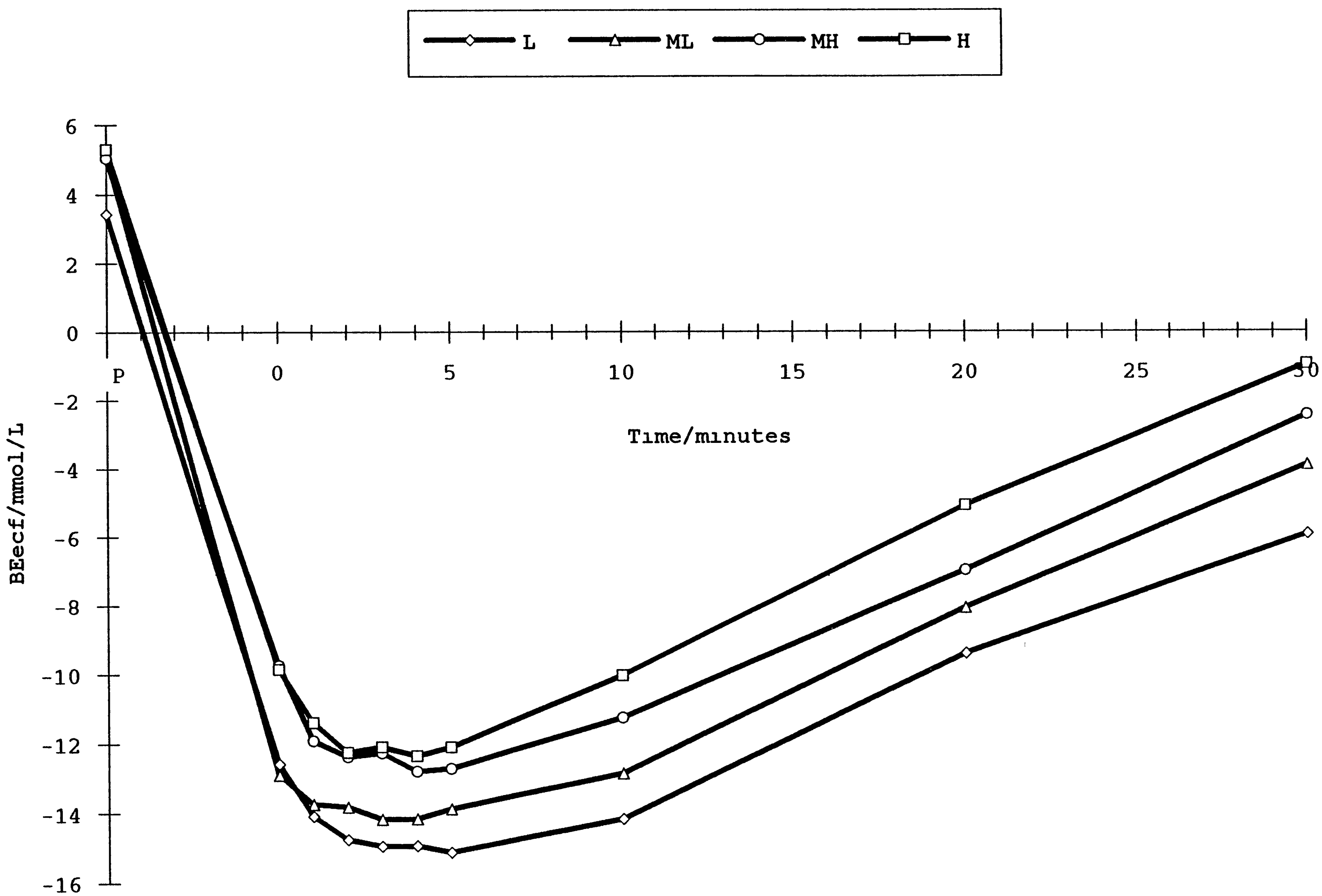

Figure 17. Effect of Dietary Cation-Anion Balance on Venous Blood Base Excess extracellular fluid in Anaerobically Exercised Horses

TABLE XIX

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD BASE EXCESS EXTRACELLULAR FLUID (mmol/L) IN HORSES FOLLOWNG ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	3.42[a]	5.17[ab]	5.03[b]	5.30[b]	0.61
0	-12.58[ac]	-12.91[a]	-9.76[b]	-9.87[bc]	0.96
1	-14.08[a]	-13.75[a]	-11.92[a]	-11.40[a]	1.18
2	-14.75[a]	-13.82[a]	-12.38[a]	-12.25[a]	1.42
3	-14.93[a]	-14.17[a]	-12.27[a]	-12.10[a]	1.49
4	-14.92[a]	-14.16[a]	-12.78[a]	-12.35[a]	1.49
5	-15.12[a]	-13.87[a]	-12.71[a]	-12.10[a]	1.64
10	-14.17[a]	-12.86[a]	-11.26[a]	-10.03[a]	2.02
20	-9.41[a]	-8.07[a]	-6.98[a]	-5.10[a]	2.27
30	-5.93[a]	-3.91[a]	-2.46[a]	-0.97[a]	1.99

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

TABLE XX

EFFECT OF DIETARY CATION-ANION BALANCE ON VENOUS BLOOD %SO_2 SATURATION (mmol/L) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	77.57[a]	79.20[a]	78.78[a]	79.93[a]	5.17
0	82.27[a]	84.40[a]	82.11[a]	85.87[a]	5.39
1	82.83[a]	85.73[a]	86.23[a]	87.51[a]	5.49
2	84.85[a]	85.15[a]	86.25[a]	87.87[a]	1.21
3	85.36[a]	85.32[a]	86.97[a]	87.52[a]	1.50
4	83.70[a]	87.21[a]	87.08[a]	88.15[a]	5.40
5	85.57[a]	87.73[a]	87.36[a]	88.18[a]	5.78
10	87.32[a]	85.98[a]	87.01[a]	87.71[a]	5.20
20	86.68[a]	87.30[a]	86.16[a]	87.82[a]	1.41
30	85.42[a]	86.17[a]	82.11[a]	83.90[a]	2.66

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

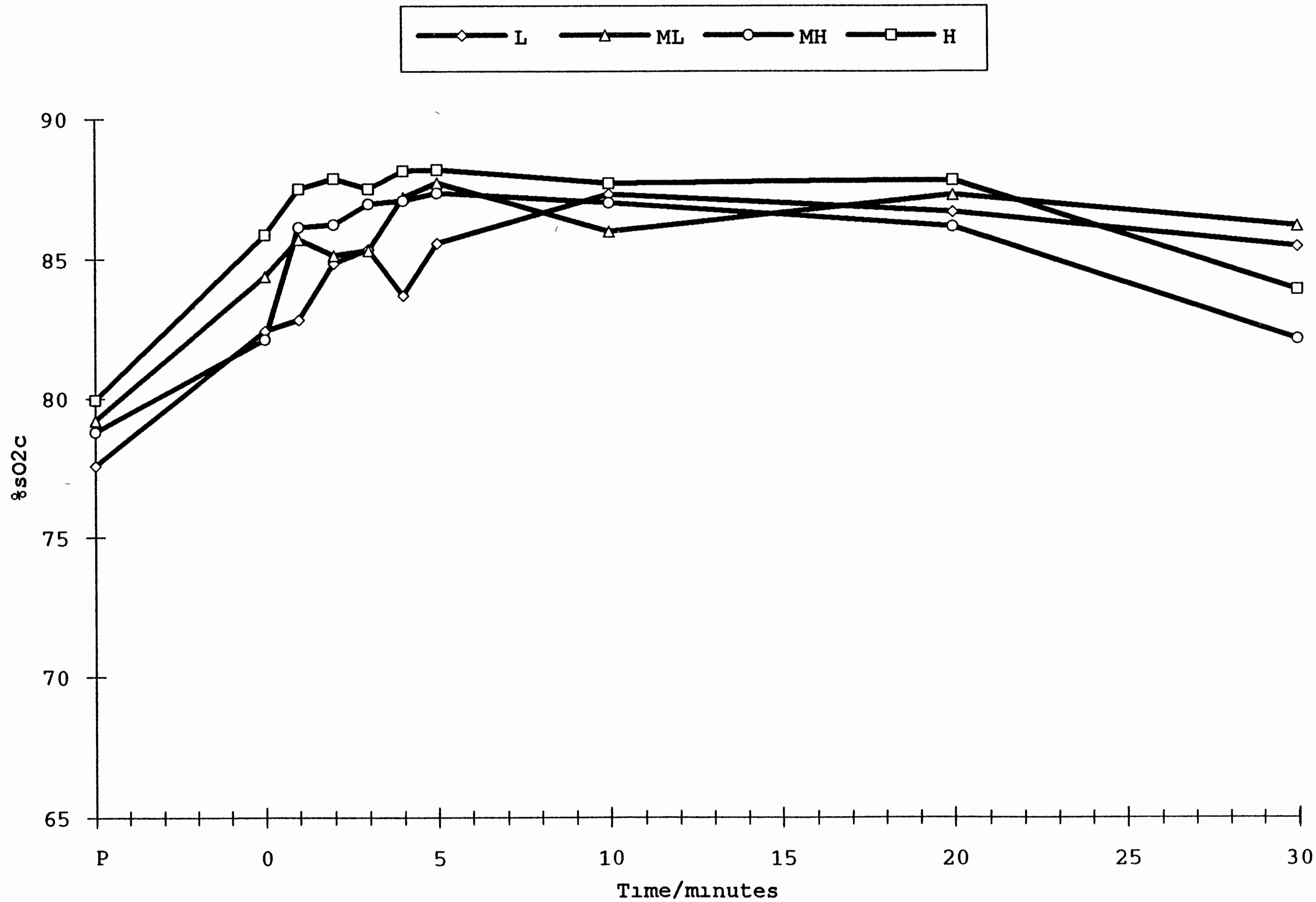

Figure 18. Effect of Dietary Cation-Anion Balance on %O_2 saturation in Venous Blood in Anaerobically Exercised Horses

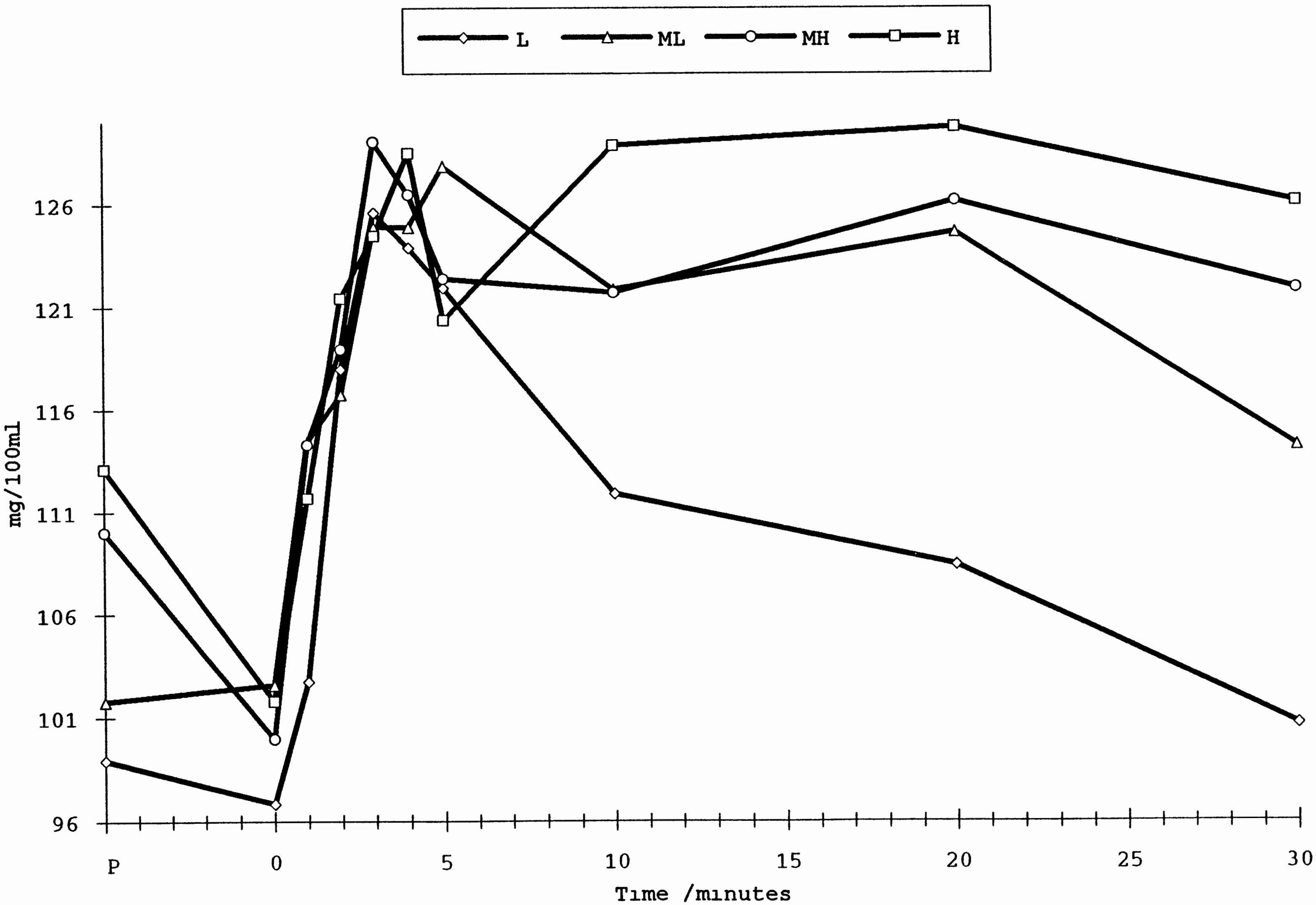

Figure 19. Effect of Dietary Cation-Anion Balance on Plasma Glucose in Anaerobically Exercised Horses

for plasma glucose concentrations ranged from 96.83 mg/100ml on the low diet to 126.07 mg/100ml on the high diet (Table XXI). The significantly lower plasma glucose concentrations between 10 and 30 minutes post exercise for the low dıet, may be due to that horses on the low diet expend more energy compensating for metabolic acidosis. Horses on the low diet experienced a lower venous blood pH throughout the SET and recovery. Due to the lower pH, enzymes are less efficient, therefore there may be less glycogen mobılized. There were no significant differences ın heart rates between treatments during the exercise test or durıng the recovery phase (Figure 20). Venous blood lactate samples were collected, but due to technician error data is not available.

TABLE XXI

EFFECT OF DIETARY CATION-ANION BALANCE ON BLOOD GLUCOSE CONCENTRATIONS (mg/100ml) IN HORSES FOLLOWING ANAEROBIC EXERCISE

	TREATMENT				
Time(min)	L	ML	MH	H	S.E.
PRE	98.93^{a}	101.77^{ab}	109.98^{ab}	113.08^{b}	4.67
0	96.83^{a}	102.54^{a}	9.94^{a}	101.77^{a}	4.63
1	102.70^{a}	114.41^{b}	114.25^{ab}	111.66^{ab}	4.72
2	117.96^{a}	116.73^{a}	118.93^{a}	121.42^{a}	5.47
3	125.59^{a}	124.89^{a}	129.00^{a}	124.47^{a}	5.80
4	123.89^{a}	124.89^{a}	126.46^{a}	128.45^{a}	6.94
5	121.93^{a}	127.83^{a}	122.37^{a}	120.36^{a}	6.16
10	111.87^{a}	121.84^{ab}	121.70^{ab}	128.82^{b}	5.18
20	108.38^{a}	124.63^{b}	126.16^{b}	129.69^{b}	3.90
30	100.67^{a}	114.20^{b}	121.84^{bc}	126.07^{c}	4.60

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

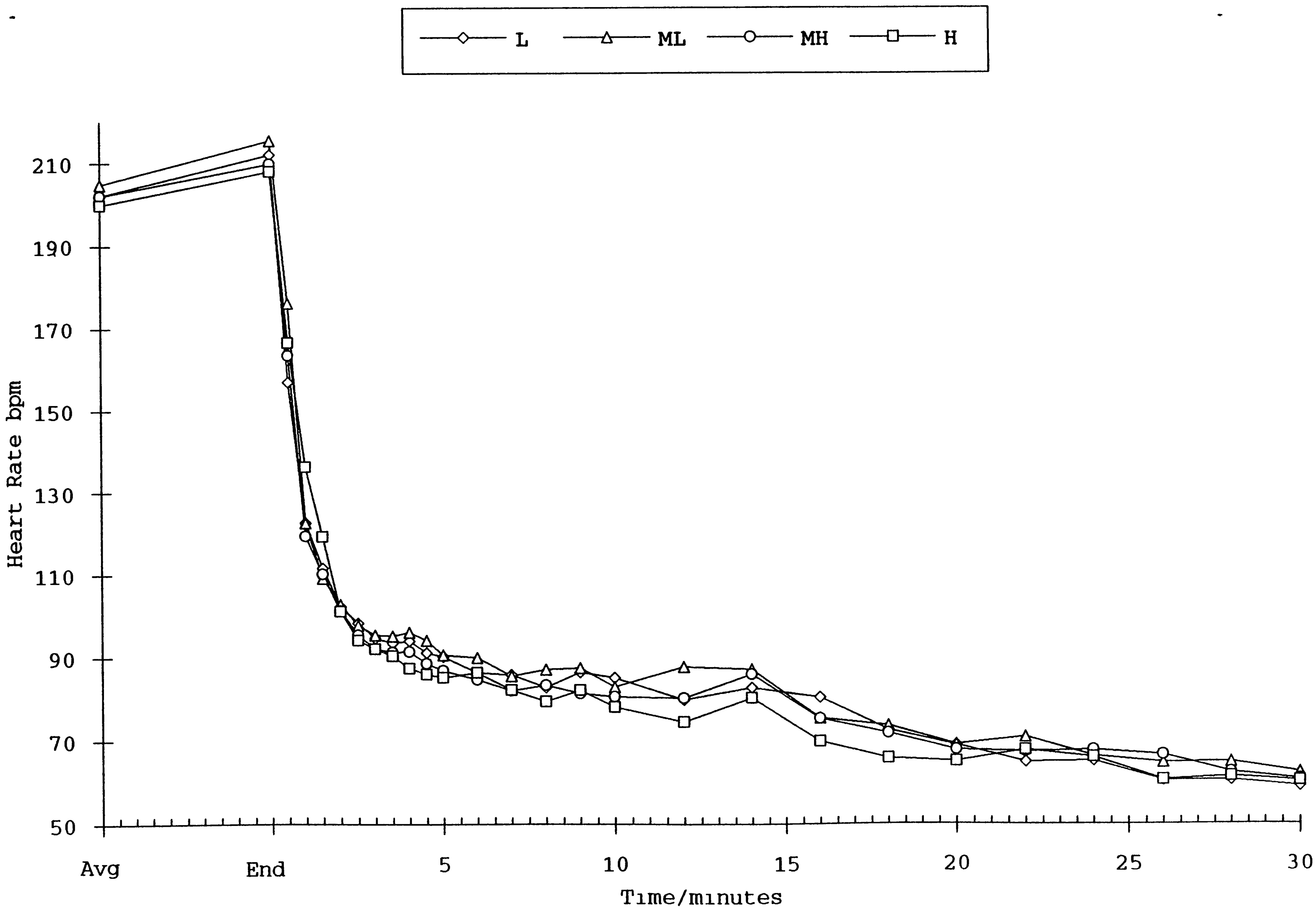

Figure 20. Effect of Dietary Cation-Anion Balance on Heart Rate in Anaerobically Exercised Horses

CHAPTER V

SUMMARY

Horses consuming diets with a low DCAB experienced decreased blood pH and altered acid-base status. The data from the seventeen hour blood collection period indicate four hours post feeding may be the most beneficial time to perform exercise on horses consuming diets high in DCAB. No statistically significant buffering affect on blood pH was found following the standard exercise test (SET) for horses consuming the medium high and high diets. However, there was a nonsignificant trend for horses consuming the high diet to have higher venous blood pH and bicarbonate levels following anaerobic exercise as compared to horses consuming diet L. The SET produced a partially compensated metabolic acidosis in all horses across all treatments. Horses consuming diet L had lower plasma glucose concentrations during the recovery phase, indicating more energy was expended compensating for metabolic acidosis. Horses consuming diet H had significantly higher glucose concentrations at 10, 20 and 30 minutes during the recovery phase as compared to horses consuming diet L. The elevated glucose concentrations in horses consuming diet H may indicate that horses on the high diet produced more lactate

during anaerobic exercise.

Horses performing anaerobic exercise may benefit from a diet containing a DCAB higher than that currently fed in the industry. The results of this study, suggest that feeding a diet high in DCAB to horses performing anaerobic exercise may improve performance. A diet high in DCAB coupled with initiation of the exercise bout at four hours post feeding may help alleviate acid-base disturbances associated with anaerobic exercise.

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