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The Effect of Sodium Bicarbonate Ingestion on Blood Parameters in Exercising Horses

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Summary

Six Quarter Horse mares were used in a crossover design to evaluate the effects of sodium bicarbonate administration during exercise and recovery. Each mare performed two exercise tests, approximately one month apart. The exercise test consisted of work to fatigue on a motorized treadmill at 4.5 m/sec on an 11% grade. Each horse carried 27 kg of lead during the test. For the first exercise test, 3 horses received sodium bicarbonate (300 mg/kg body weight) and 3 received a placebo (control). The treatments were then reversed for the second test. The treatments were administered by drench 1-1/2 to 2-1/2 h prior to beginning each exercise bout. Blood samples were taken via indwelling jugular catheters inserted 30 min prior to starting the exercise test. The average time to fatigue was 1191.8 ± 87.2 sec for the control treatment and 1218.5 ± 53.5 for the sodium bicarbonate treatment. Pre-exercise venous pH and bicarbonate levels were elevated by the sodium bicarbonate treatment ($P < .05$). Blood pH decreased during the exercise in both treatments, but was higher on the sodium bicarbonate treatment during exercise and recovery ($P < .05$). Venous lactate levels were higher ($P < .05$) during exercise and recovery in the sodium bicarbonate treatment also. During the last minute of exercise lactates were 10.9 ± 1.0 mmol/l and 15.2 ± 1.5 mmol/l for control and sodium bicarbonate treatments, respectively.

Index terms: Blood buffering; alkalosis; lactic acid.

Introduction

Lactic acid accumulation in muscle has been cited as a cause of fatigue during high intensity exercise (Gledhill, 1984). Lactic acid build-up creates an acidosis which may alter the contractile properties of muscle (Gollnick *et al.*, 1986) and possibly inhibit glycolysis (Ehram *et al.*, 1982). In vitro experiments have suggested that lactate and hydrogen ion efflux from muscle is inhibited when the pH of the perfusate is reduced (Mainwood and Worsley-Brown, 1975). Thus the removal of lactic acid from muscle may be impaired as blood pH decreases during intense exercise.

Oral ingestion of sodium bicarbonate by humans elevates blood pH and improves performance during intense exercise (Wilkes *et al.*, 1983). After sodium bicarbonate

ingestion, blood bicarbonate levels increase, which is believed to promote the efflux of lactate and hydrogen ions out of the muscle and into the blood. This theory is supported by the observation that blood lactate levels during and after exercise are higher in individuals administered sodium bicarbonate (Jones *et al.*, 1977; Wilkes *et al.* 1983).

This study was conducted to determine whether ingestion of sodium bicarbonate would alter the metabolic response of horses to intense exercise. In particular it was of interest to evaluate effects on blood pH and lactate levels.

Materials and Methods

Six Quarter Horse mares that had been conditioned for 7 weeks on a motorized treadmill were used in a cross-over design. Each mare performed 2 exercise tests, approximately 1 month apart. In the first test, 3 mares received the sodium bicarbonate treatment (300 mg/kg body weight) and 3 received a placebo (control treatment). In the second test the treatments were reversed. The treatments were administered by drench 1-1/2 to 2-1/2 h before each horse performed the test. For administration, the sodium bicarbonate was mixed with 30 ml corn syrup and enough water to make a thick paste. The placebo consisted of water and corn syrup. The horses had received their last meal 16–18 h before the exercise test.

The exercise test consisted of 1 min at 2 m/sec and 2 min at 3.5 m/sec followed by work to fatigue at 4.5 m/sec on an 11% grade. The horses carried 27 kg of lead during the test. Fatigue was defined as the point at which the horses would no longer willingly continue. At least 30 min prior to the exercise test a jugular catheter was placed in each horse. Blood samples were taken before (0 min), and at 2, 4, 8, 12, and 16 min of exercise, during the last minute of exercise (LAST), and at 2, 5, 15, 30 and 60 min of recovery. Heart rate was monitored by hardwire electrocardiogram before and during exercise and at 2 and 5 min of recovery. Respiration rate was determined within the first 2 min of recovery.

Blood for lactate analysis was immediately deproteinized in chilled perchloric acid. The supernatant was removed and assayed for lactate (Sigma Co., St. Louis, MO). Blood for pH and blood gas analysis was collected in chilled heparinized glass syringes that were immediately sealed without air bubbles and stored on ice. Determinations were made within 2 h of collection using an automated blood gas analyzer (Corning Equipment Co., Corning, N.Y.). Effects of treatment on the pre-exercise samples, respiration rates and fatigue time were evaluated with a paired t-test. The exercise and recovery data sets were each analyzed as a split-plot with repeated measures (Freund and Littell, 1979). The whole unit factor was treatment and time was the subunit factor. Differences due to treatment were tested using the horse by treatment interaction as the error term. The model used was

$$Y_{ijkl} = u + P_j + D_k + (HD)_{ik} + T_l + (HT)_{il} + (PT)_{jl} + (DT)_{kl} + E_{ijk}$$

where u = overall mean
 P_j = effect of cross-over
 D_k = effect of treatment (control vs sodium bicarbonate)
 $(HD)_{ik}$ = interaction between horse and treatment
 T_l = time
 $(HT)_{il}$ = interaction between horse and time

(PT)_{jl} = interaction between crossover and time
 (DT)_{kl} = interaction between treatment and time
 E_{ijk} = experimental error associated with the subunit

Results

Time to fatigue was 1191.8 ± 87.2 sec with the control treatment and 1218.5 ± 53.5 sec with the sodium bicarbonate treatment. The exercise test caused heart rate to in-

TABLE 1. Effect of sodium bicarbonate on venous pH and blood bicarbonate before exercise*.

Treatment	pH	HCO ₃ [†]
Control	7.385 ± .011	31.7 ± .45
Sodium bicarbonate	7.436 ± .011 ^{††}	35.8 ± .96 ^{††}

*Samples taken 1-1/2-2-1/2 h following administration; mean ± S.E.

[†]Blood bicarbonate, meq/l

^{††}Significantly different than control (P < .05).

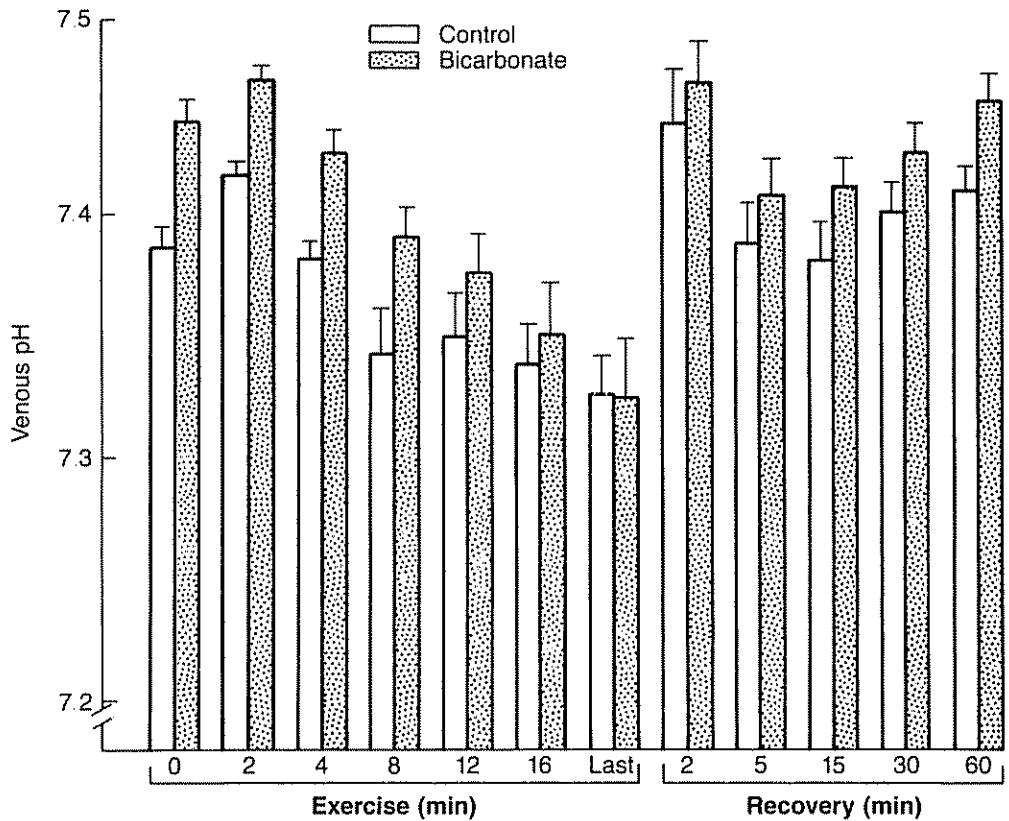


FIGURE 1 Effect of sodium bicarbonate on venous blood pH during exercise and recovery (means ± S.E.; n = 6).

crease to 199.2 ± 8.1 bpm with the control group and to 204.0 ± 6.1 bpm with the sodium bicarbonate group at 16 min. There was no effect of treatment on heart rate. Respiration rate in the second minute of recovery was 86.7 ± 3.7 with the control and 97.3 ± 3.2 with the sodium bicarbonate treatment ($P < .05$).

The sodium bicarbonate treatment significantly ($P < .05$) elevated pH and bicarbonate in the blood samples taken prior to exercise (Table 1). Sodium bicarbonate treatment elevated blood pH during the exercise bout also ($P < .05$), although the effect diminished over the course of the exercise bout (Fig. 1). During the recovery period pH rebounded quickly in both groups, with an effect of sodium bicarbonate being evident ($P < .05$). Venous blood bicarbonate decreased during exercise and into 2 min of recovery (Fig. 2). During exercise and recovery there were significant time \times treatment interactions ($P < .05$) for blood bicarbonate. Blood bicarbonate was higher with the sodium bicarbonate treatment during early exercise but not at the end of the exercise bout. Bicarbonate levels were higher with the sodium bicarbonate only at the end of recovery.

Lactate levels increased steadily during exercise to a maximum of 10.9 ± 1.0 mmol/l for the control treatment and 15.2 ± 1.5 mmol/l for the sodium bicarbonate treatment during the last minute of exercise (Fig. 3). Sodium bicarbonate supplementation resulted in higher lactate levels during exercise and recovery ($P < .05$).

Exercise produced a significant ($P < .05$) decrease in venous P_{CO_2} (Fig. 4). There was no effect of sodium bicarbonate supplementation on P_{CO_2} during exercise, but dur-

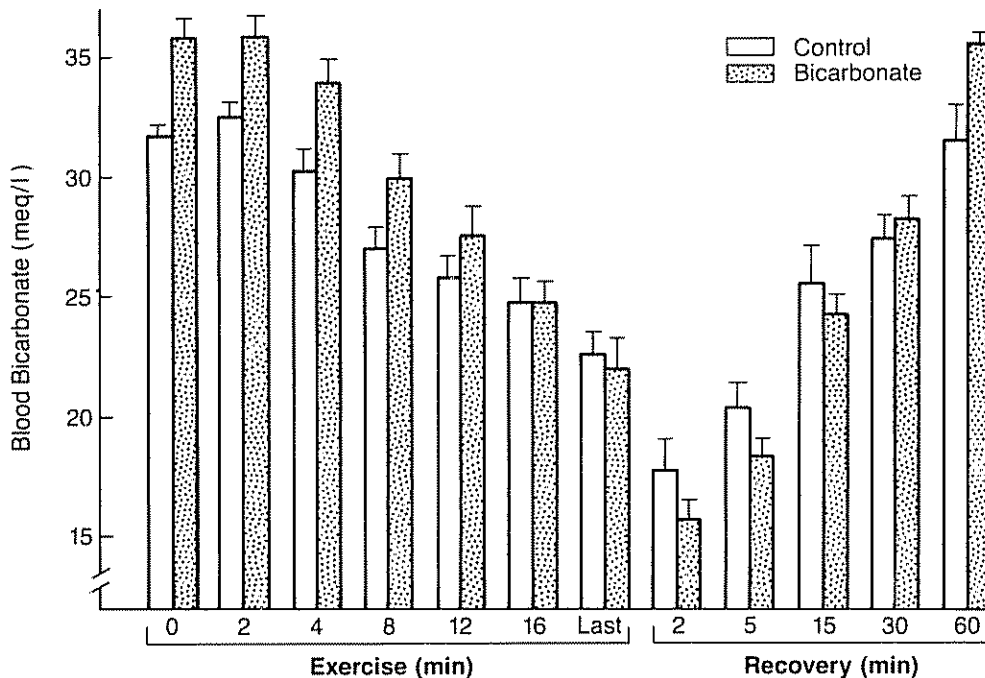


FIGURE 2. Effect of sodium bicarbonate on blood bicarbonate levels (meq/l) during exercise and recovery (means \pm S.E.; $n = 6$).

ing early recovery venous P_{CO_2} was lower for the sodium bicarbonate treatment than for the control treatment ($P < .05$).

Discussion

As early as 1931, altered acid-base status was found to alter work performance (Dennig *et al.*, 1931). Since then several studies have examined the effects of metabolic or respiratory acidosis and/or metabolic alkalosis on exercise performance (Jones *et al.*, 1977; Sutton *et al.*, 1981; Ehrsam *et al.*, 1982; Wilkes *et al.*, 1983). Metabolic alkalosis is most frequently induced by ingestion of sodium bicarbonate which augments the bicarbonate buffer system in the blood. In one study with human athletes, sodium bicarbonate raised pre-exercise blood pH from 7.39 and 7.49 and blood bicarbonate from 26.2 to 33.5 meq/l (Wilkes *et al.*, 1983). The changes in this study for blood pH and bicarbonate were slightly smaller in magnitude but still significant, indicating that in horses an alkalosis can be induced by sodium bicarbonate administration. The dose used in this study was 300 mg/kg body weight given 1-1/2 to 2-1/2 h prior to exercise. This is the same method reported by Jones *et al.*, 1977 and Wilkes *et al.*, 1983. Lower doses in humans have not produced beneficial effects on work performance (Gledhill,

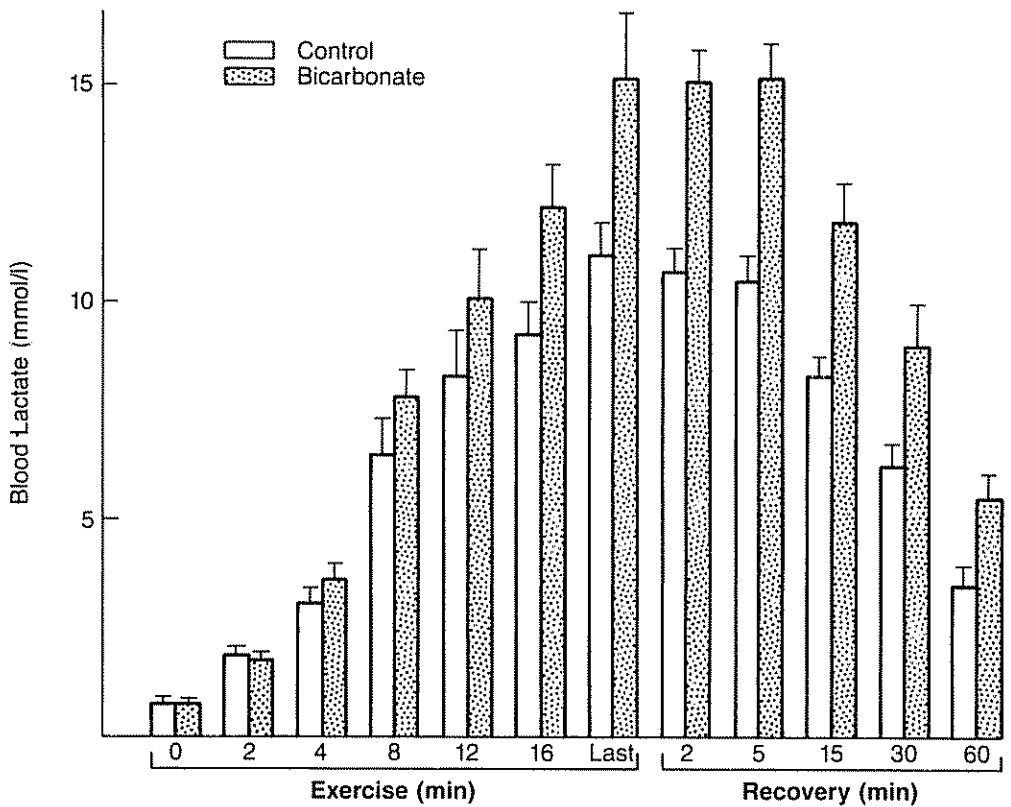


FIGURE 3. Effect of sodium bicarbonate on blood lactate (mmol/l) during exercise and recovery (means \pm S.E.; n = 6)

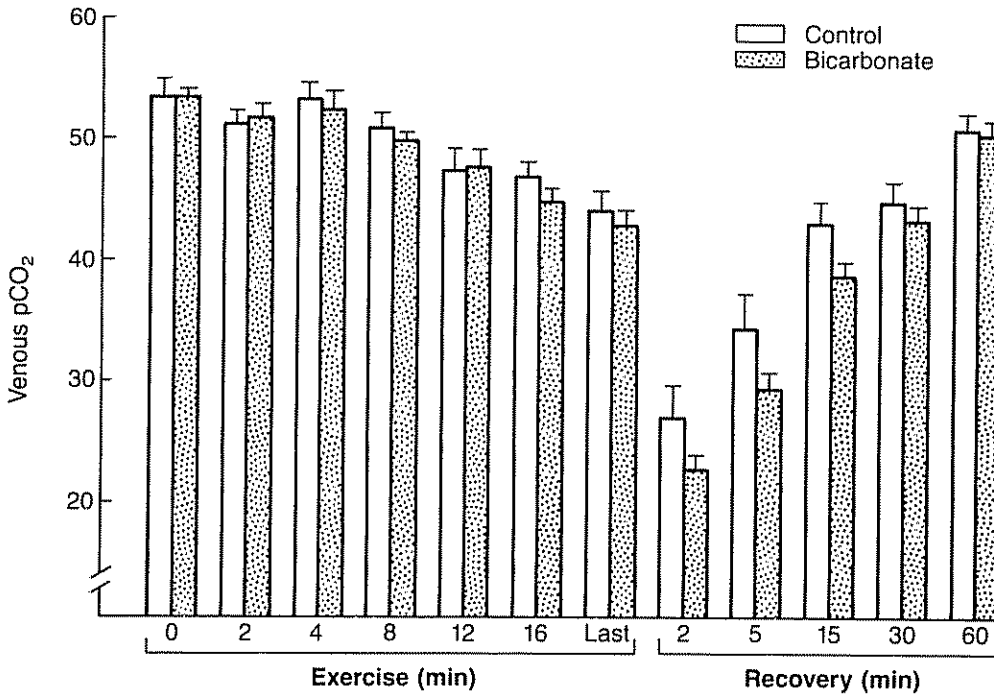


FIGURE 4 Effect of sodium bicarbonate on venous pCO₂ (mm Hg) during exercise and recovery (means \pm S.E.; n = 6)

1984). In a previous study from our laboratory, horses that received approximately 150 mg/kg mixed in a meal did not have a significant elevation in pH at 2 h post feeding (Hank *et al.*, 1985).

During anaerobic glycolysis, lactate and H⁺ are released in stoichiometric equal amounts. When the H⁺ leave the muscle and enter the blood they are sequestered by both bicarbonate and non-bicarbonate buffering systems and therefore simply measuring the pH decline is insufficient in assessing proton efflux (Keul *et al.*, 1972). However blood lactate concentrations reasonably reflect the quantity of H⁺ entering the blood since the efflux of the lactate anions and protons are closely correlated in higher vertebrates (Bouhuys *et al.*, 1966; Keul *et al.*, 1972). In this study, lactate level increased throughout exercise and at the end of the test lactate was approximately 40% higher with the sodium bicarbonate treatment than with the control treatment. Despite the heavier lactate load at the last minute of exercise, blood pH with the sodium bicarbonate treatment was similar to the control treatment. This suggests that under the influence of sodium bicarbonate supplementation the buffering capacity of the blood can contend with a higher level of lactic acid. There are several mechanisms for buffering metabolically produced H⁺, but a major role is played by the blood bicarbonate system. A depletion of blood bicarbonate is associated with metabolic acidosis and a mirror image relationship exists between blood bicarbonate and lactate (Milne *et al.*, 1976; Buono and Roby, 1982). In this study it was expected that sodium bicarbonate administration would augment the blood bicarbonate system. At rest blood bicarbonate levels were

higher with sodium bicarbonate treatment. Furthermore, when blood bicarbonate levels are compared at equivalent lactate levels, there is a higher bicarbonate reserve in the supplemented treatment. For example, at a lactate level of 10.9 mmol/l, blood bicarbonate in the sodium bicarbonate treatment is about 26 meq/l compared to 22.7 meq/l for the control.

The respiratory component of pH regulation was most evident during early recovery. The rebound in pH at the second minute of recovery reflected the decrease in P_{CO_2} while lactate remained high and bicarbonate remained low.

The higher blood pH during early recovery with sodium bicarbonate treatment appeared to be a consequence of P_{CO_2} , since blood bicarbonate was not different between treatments during recovery. The lower P_{CO_2} could be a result of an increased respiratory rate during recovery in the sodium bicarbonate treatment. Other studies using sodium bicarbonate to produce a metabolic alkalosis have not reported venous P_{CO_2} or respiration rates during recovery. The increased respiratory rate may be related to a larger oxygen debt incurred with greater lactate production.

Sodium bicarbonate administration is applicable only to horses competing in events that generate a significant metabolic acidosis and would be contraindicated for endurance type horses. The results of this study are encouraging in that the pH and lactic acid responses resembled those reported in human studies where performance was improved. However, further research is required to evaluate optimal dose rate and schedule.

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Blood and Muscle Ammonia Concentrations in Horses during Treadmill Work and after Racing

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Summary

Blood ammonia and lactate concentrations were analyzed during treadmill work of different intensities. In addition, muscle biopsies were obtained from 3 horses before and after standardized treadmill work and in 14 horses after racing for analysis of ammonia, lactate and adenosine triphosphate (ATP) concentrations, as well as citrate synthase activity and fiber type composition. An accumulation in blood of ammonia and lactate was only found at higher work intensities. Fiber type composition differed between horses and so did metabolite concentrations after racing. Ammonia concentrations in muscle were increased after exercise and positively correlated to lactate concentrations while a negative correlation was seen with ATP concentrations. Furthermore, the percentage of type II fibers in the biopsy was positively correlated with ammonia concentrations after exercise and negatively correlated to ATP concentrations.

It is concluded that ammonia concentrations increase in both muscle and blood at higher work intensities. Furthermore, the data indicate that during intense exercise adenosine monophosphate deamination may play an important role in type II fibers.

Index terms: Horse; fiber types; lactate; ammonia.

Introduction

Skeletal muscle is composed of fibers having slow twitch (type I) or fast twitch (type II) characteristics and during exercise the fibers are recruited in different patterns which depend on the intensity and duration of the performed work (Saltin and Gollnick, 1983; Armstrong and Laughlin, 1985). The immediate source for energy during muscular work is adenosine triphosphate (ATP) which continuously is replenished either through oxidative processes, through glycolysis with lactate formation or through phosphagen breakdown. However, the capacity for ATP supply through oxidative or glycolytic pathways differs between fiber types (Saltin and Gollnick, 1983; Pette, 1985).

In rats when ATP turnover rate is high, such as with intense treadmill work or electrical stimulation, not only lactate accumulates in muscle but also ammonia and IMP (Meyer and Terjung, 1979; Meyer *et al.*, 1980). Activation of the purine nucleotide cycle and deamination of AMP give rise to IMP and ammonia in muscle (Lowenstein,