Krzywanek, H. (1973). Untersuchungen zur Beurteilnug der aktuellen Leistungfähigkeit von Trabrennpferden. *Zbl. Vet. Med A*, **20**, 265–276.
Krzywanek, H. (1974). Lactic acid concentrations and pH values in trotters after racing. *J. S. Afr. Vet. Assoc.* **45**, 355–360.
Krzywanek, H., Milne, D. W., Gabel, A. A. and Smith, L. G. (1976). Acid-base values of Standardbred horses recovering from strenuous exercise. *Am. J. Vet. Res.* **37**, 291–294.
Kubo, K., Takagi, S., Murakami, M. and Kai, M. (1984). Heart rate and blood lactate concentration of horses during maximal work. *Bull. Equine Res. Inst.* **21**, 39–45.
Lindholm, A. and Saltin, B. (1974). The physiological and biochemical response of Standardbred horses to exercise of varying speed and duration. *Acta Vet. Scand.* **15**, 310–324.
Milne, D. W., Gabel, A. A., Muir, W. W. and Skarda, R. T. (1977). Effects of training on heart rate, cardiac output, and lactic acid in Standardbred horses, using a standardized exercise test. *J. Equine Med. Surg.* **1**, 131–135.
Nimmo, M. A. and Snow, D. H. (1983). Changes in muscle glycogen, lactate and pyruvate concentrations in the Thoroughbred horse following maximal exercise. *In: Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 237–244.
Panndorf, H., Schneider, J., Mohring, B. and Werner, G. (1980). Zum Verhalten der Milchsaurekonzentration im Blut beim Training von Galopprennpferden. *Mh. Vet. Med.* **35**, 734–739.
Persson, S. G. B. (1983). Evaluation of exercise tolerance and fitness in the performance horse. *In: Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 441–457.
Persson, S. G. B. and Ullberg, L. E. (1974) Blood volume in relation to exercise tolerance in trotters. *J. S. Afr. Vet. Assoc.* **45**, 293–299.
Snow, D. H. and MacKenzie, G. (1977). Some metabolic effects of maximal exercise in the horse and adaptations with training. *Equine Vet J.* **9**, 134–140.
Snow, D. H., Mason, D. K., Ricketts, S. W. and Douglas, T. A. (1983). Post-race blood biochemistry in Thoroughbreds. *In: Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 389–399.
Snow, D. H., Harris, R. C. and Gash, S. P. (1985). Metabolic response of equine muscle to intermittent maximal exercise. *J. Appl. Physiol.* **58**, 1689–1697.
Thornton, J., Essén-Gustavsson, B., Lindholm, A., McMiken, D. and Persson S. (1983). Effects of training and detraining on oxygen uptake, cardiac output, blood gas tensions, pH and lactate concentrations during and after exercise in the horse. *In: Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 470–486.
van den Hoven, R. (1983). Enkele aspecten van de lactaatvorming en verdwijning bij drawers tijdens in spanning. *Tijdschr. Diergeneeskd.* **108**, 760–764.
Wilson, R. G., Isler, R. B. and Thornton, J. R. (1983). Heart rate, lactic acid production and speed during a standardized exercise test in Standardbred horses. *In: Equine Exercise Physiology*. Snow, D. H., Persson, S. G. B. and Rose, R. J. (eds.) Granta Editions, Cambridge, pp. 487–496.

Bicarbonate Administration and Muscle Metabolism during High-Intensity Exercise

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Summary

The effect of bicarbonate administration on blood and muscle metabolite concentrations and pH before and after a supramaximal exercise bout was investigated. Six Thoroughbred horses ran 1600 m at maximal speed on two occasions after receiving either 4.0 l of water or 4.0 l of water which contained 0.4 g $NaHCO_3$/kg, using a double blind protocol. Blood and muscle samples were collected before fluid administration, prior to and after exercise, and after 5 and 10 min of recovery. Bicarbonate administration elevated blood pH and bicarbonate concentration ($P < 0.05$) prior to exercise, however, it did not affect pre-exercise muscle metabolites or pH. Exercise increased muscle and blood lactate concentration and muscle glucose-6-phosphate concentration, which remained elevated during recovery. Exercise also resulted in decreases in the concentration of adenosine triphosphate, creatine phosphate, glycogen, and pH ($P < 0.05$). There were no post-exercise metabolite or blood gas differences between water and bicarbonate treatments. These results do not support the contention that administration of bicarbonate improves the intracellular environment thereby allowing metabolic pathways to operate beyond normal levels.

Index terms: Muscle metabolites; pH; ergogenic aids; blood gases.

Introduction

Earlier studies of exercise physiology and biochemistry have demonstrated that the production of acid metabolites during heavy exercise leads to an acidification of muscle and blood. This acidification can produce deleterious effects on the contractile and metabolic capacities of muscle. The ability to buffer the acid produced by muscle could have beneficial effects on performance capacity. The idea of augmenting the buffering capacity of the blood, and perhaps of muscle, in the search for ergogenic aids is not new. Since the blood is readily accessible there have been many attempts to alter its buffering capacity and to determine the effects of such alterations on performance ca-

pacity. Considerable attention has been placed on the bicarbonate buffer since it is readily altered and plays a major role in regulation of blood pH. Reports of these studies cover a period of more than 50 years and the problem remains unsolved.

The studies reported here were undertaken as a result of the continued interest within the racing industry in devising ways of combating the acidosis of heavy exercise by the administration of substances which will increase blood buffering capacity. A double-blind design was used where horses were given a bicarbonate load via naso-gastric intubation prior to a maximal exercise test. Arterial and venous blood and muscle samples were obtained at rest and after exercise. Metabolite and pH concentrations in these samples were measured to determine whether there had been a significant metabolic effect resulting from changing the blood buffering capacity. The results reported in this paper do not support the contention that the administration of bicarbonate improves the intracellular environment or performance capacity.

Materials and Methods

Six moderately trained Thoroughbred horses, 5 geldings and 1 mare, were studied. Each animal ran 1600 m at maximal speed on two occasions. Each horse received either 4.0 l of water or 4.0 l of water containing sodium bicarbonate ($NaHCO_3$) (0.4 g/kg body weight) via a stomach tube 1 hr before each exercise.

Approximately 2 hr before exercise each horse was prepared for collection of muscle and blood samples. Biopsy samples were collected from the middle gluteal muscle using the method described by Bergström (1962). Areas over the jugular vein and a portion of the carotid artery, which had previously been translocated to a subcutaneous position (carotid loop), were aseptically prepared and catheters inserted. Muscle and blood samples were collected at rest 1 hr before fluid (water or $NaHCO_3$) administration, immediately prior to and after exercise, and at 5 and 10 min post-exercise. The immediate post-exercise samples were collected after slowing the horses to a complete stop (about 20 sec). Muscle samples were frozen (<7 sec) by being forcefully extruded from the biopsy needle into liquid nitrogen. The blood samples for lactate (LA) determination were placed in tubes containing potassium oxalate and sodium fluoride and placed on ice. Blood samples for blood gas and pH determination were collected into heparinized syringes under anaerobic conditions, capped, placed on ice, and subsequently analyzed for partial pressures of oxygen and carbon dioxide, bicarbonate, and pH using an Instrumentation Laboratories, Inc. model 813 blood gas analyzer. Muscle samples were analyzed for selected metabolites and pH. The preparation of the samples involved pulverization of the sample at the temperature of liquid nitrogen in a micromortar (Pette and Reichmann, 1982), extraction of the tissue with 2.0 N perchloric acid, centrifugation to remove the precipitated protein, neutralization of the protein-free extract and removal of potassium perchlorate, and an alkalization and neutralization sequence to remove any surviving enzymes, particularly adenylate kinase. The neutralized samples were analyzed for adenosine triphosphate (ATP), creatine phosphate (CP), creatine (Cr), glucose-6-phosphate (G-6-P), and LA using standard methods (Lamprecht and Trautschold 1974; Harris, *et al.*, 1974; Lamprecht *et al.*, 1974; Lang and Michal, 1974; Lowry and Passonneau, 1972). Blood samples were extracted in a manner similar to that used for muscle except the second alkalization and neutralization steps were omitted. The extracts were analyzed for LA. The muscle samples used to determine muscle

pH were pulverized and placed into a solution containing 145 mmol KCl, 10 mmol NaCl, 5 mmol iodoacetate and 0.3 mmol dinitrofluorobenzene (DNFB) at 22°. The pH was determined with an Orion model 701A pH meter with a glass electrode (Microelectrode Laboratories, Inc. model MI 410 microelectrode). Muscle glycogen was determined as described by Passonneau and Lauderdale (1974).

Results

Mean run time for the bicarbonate treated horses was 111.3 ± 1.2 sec and 114.0 ± 2.2 sec under control conditions ($0.05 < P < 0.10$). The administration of 0.4 g/kg of bicarbonate 1 hr prior to collecting blood samples produced an elevation of venous blood pH from a normal resting value of 7.39 ± .009 to 7.43 ± .004 ($P < 0.05$) and in the concentration of bicarbonate in the venous blood from 28.73 ± 0.78 to 31.02 ± 0.84 mmol/l ($P < 0.05$) (Fig. 1). Similarly, the bicarbonate concentration in arterial blood increased from 27.92 ± 0.88 to 29.73 ± 0.84 mmol/l (Fig. 1). There was no change in any of these variables as a result of the administration of an equal volume

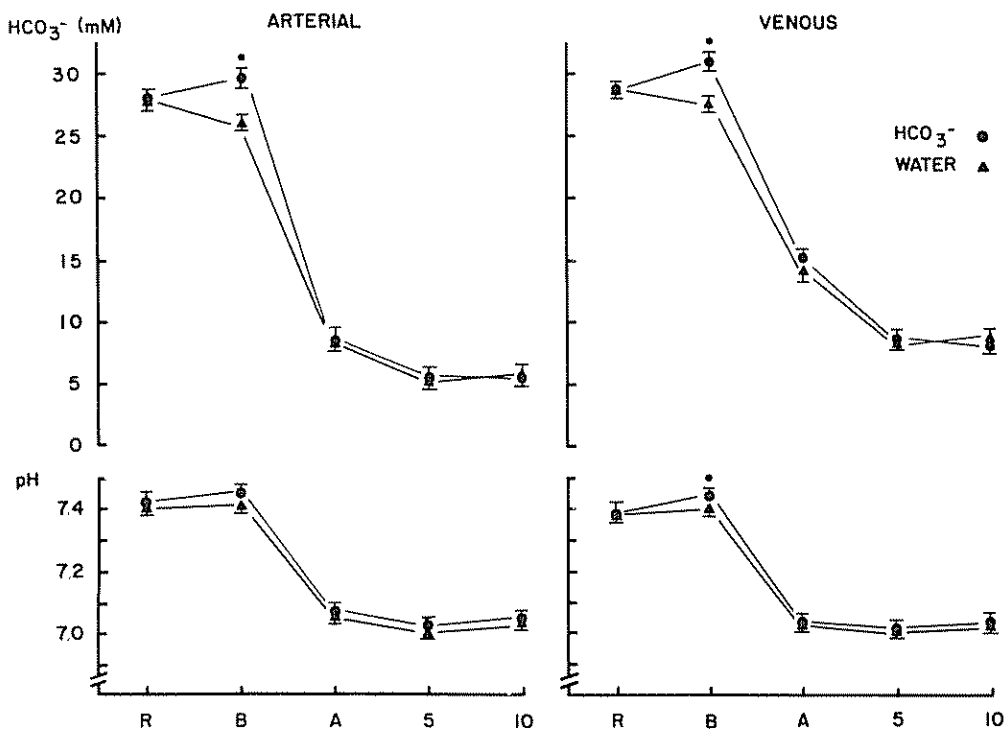

FIGURE 1. Arterial and venous pH and bicarbonate concentration. R = rest; B = before exercise (1600-m run); 5 = 5 min post-exercise; 10 = 10 min post-exercise. Bars represent SEM. *$P < 0.05$

of water Results of blood gas analysis are presented in Table 1. The concentrations of metabolites in blood and muscle, and muscle pH at rest were not influenced by the administration of either water or bicarbonate.

Immediately after running 1600 m at maximal speed, the concentrations of LA in the venous blood were 22.8- and 27.2-fold greater than for rest after the water and bicarbonate treatments, respectively (Fig. 2). The increase in blood LA after exercise was not statistically different for the two treatments. Lactate concentrations in blood were highest 5 min after termination of the exercise. A decline in pH and bicarbonate ($P < 0.05$) occurred in venous and arterial blood in response to exercise (Fig. 1). There was no difference in this response to exercise as a consequence of the administration of bicarbonate.

The exercise resulted in large increases in the concentration of LA and produced a lowering of the pH within the muscle. Bicarbonate treatment did not alter the accumulation of LA in muscle or the reduction in pH (Fig. 3). Muscle glycogen declined 36.7 and 31.0% for the bicarbonate and water trials, respectively. There was a 3.5-fold increase in G-6-P concentration after the exercise following water and bicarbonate treatments (Fig. 4). There was essentially no change in G-6-P concentration during the recovery period. CP and ATP declined about 39 and 20%, respectively ($P < 0.05$), for both treatments (Fig. 5). Neither CP nor ATP concentration had returned to a rest value by 10 min post-exercise.

Discussion

The possibility that oral administration of bicarbonate could improve physical performance capacity can be supported from two standpoints. First, as demonstrated in the present and from other studies, there is an accumulation of bicarbonate in the blood after such treatments. Since bicarbonate is one of the major physiological buffers, it can easily be envisaged that augmenting its availability could help control the reduction

TABLE 1. Effects of water and bicarbonate ingestion on blood gases at rest, immediately after exercise, and during recovery from exercise

Treatment	Rest	Pre-exercise	Post exercise	5 min rec.	10 min rec.
Water					
PaO_2	89.4 ± 1.9	85.2 ± 1.0**	104.3 ± 1.4	111.7 ± 3.0	113.3 ± 2.7
$PaCO_2$	45.1 ± 1.3	42.4 ± 0.8	30.3 ± 2.0	25.3 ± 1.3	23.2 ± 1.0
PvO_2	42.6 ± 1.0	40.8 ± 1.5	56.4 ± 3.8	73.1 ± 2.1	70.5 ± 6.2
$PvCO_2$	47.2 ± 0.3	45.0 ± 0.8*	57.7 ± 5.5	37.2 ± 0.8	34.3 ± 2.4
Bicarbonate					
PaO_2	88.0 ± 2.4	90.8 ± 1.7**	100.9 ± 1.6	113.1 ± 2.9	114.2 ± 1.3
$PaCO_2$	45.7 ± 1.4	44.6 ± 1.6	32.6 ± 2.1	23.3 ± 1.9	20.9 ± 1.1
PvO_2	42.6 ± 1.2	40.2 ± 0.9	55.6 ± 2.3	70.4 ± 1.7	75.0 ± 2.9
$PvCO_2$	48.2 ± 1.2	47.7 ± 1.5*	61.2 ± 3.9	36.6 ± 1.3	32.3 ± 0.8

Values are means ± SEM in torr for 6 horses. Rec indicates recovery. The increases or decreases (all $P < 0.05$) in the blood gases are consistent with a hyperventilatory response to the exercise. Bicarbonate administration elevated PaO_2 (**$P < 0.05$) and $PvCO_2$ (*$P < 0.1$).

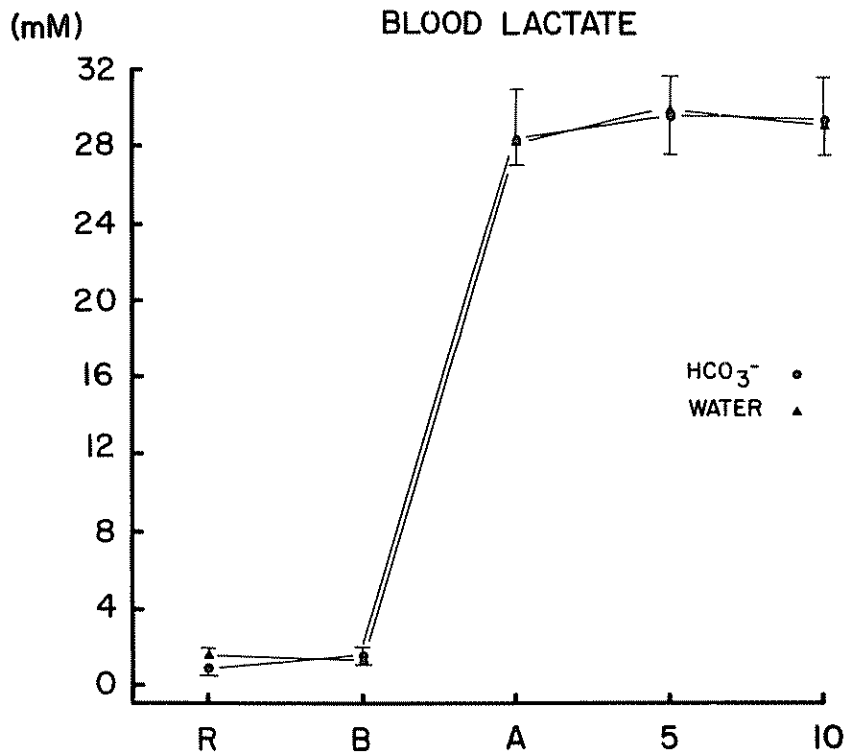

FIGURE 2 Blood lactate concentration 1 hr before treatment, immediately prior to and after exercise, and 5 and 10 min after completion of exercise. Symbols are as in Fig. 1.

in blood pH caused by LA production during heavy exercise. Secondly, increased blood bicarbonate concentration may promote the efflux of hydrogen ions from the muscle and therefore inhibit the deleterious effects of proton accumulation on further activity of the Embden-Meyerhof pathway and/or other cellular processes. However, it may be suggested from the results of the current study that there were no clear indications that bicarbonate administration significantly improved performance or altered muscle metabolism.

Studies attempting to demonstrate the effects of altering the bicarbonate pool in blood on exercise capacity are not new. There are a number of reports in the literature dealing with the effects of altering the acid-base status of man and its effect on work capacity. The results from these studies do not present a unified picture concerning the benefits of such treatments. Dennig *et al.* (1931) examined the effects of an experimentally imposed acidosis or alkalosis on blood chemistry and work capacity. Ingestion of either ammonium chloride or sodium bicarbonate was used to produce acidotic or alkalotic states. At rest, significant shifts in pH and total bicarbonate of arterial blood occurred in response to the treatments. With exercise, a more severe depression in blood pH and PCO_2 occurred in the acidotic, and a smaller depression in the alkalotic state, as compared to normal. It was concluded that run time was longer and the accumulated oxygen debt greater in the alkalotic as compared to either the normal or acidotic state. This is

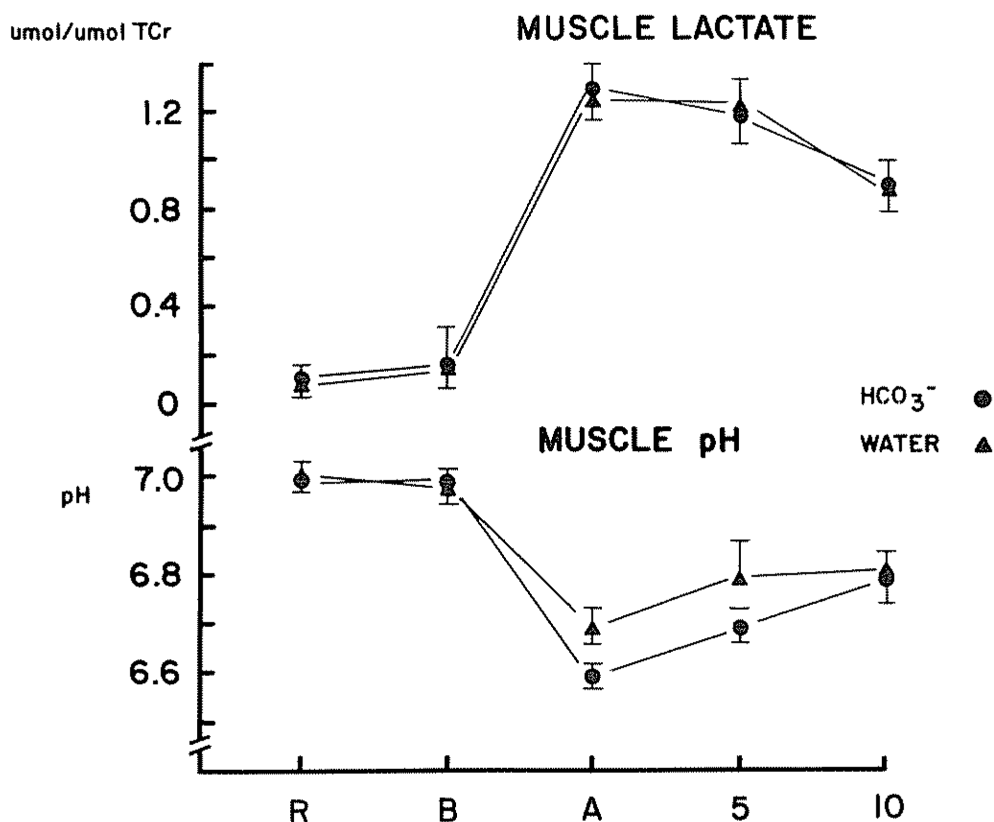

FIGURE 3. Muscle lactate and pH at rest, before, and after exercise and 5 and 10 min following exercise. Symbols are as in Fig. 1.

supported by the observation that ingestion of 0.3 g/kg over a 2 hr period prior to an 800 m race significantly improved performance (Wilkes *et al.*, 1983).

Margaria *et al.* (1971) studied subjects performing treadmill running after receiving either a placebo, a mixture of sodium bicarbonate and sodium-potassium citrate, or sodium bicarbonate. They concluded that performance during supramaximal exercise was not influenced by the induction of an alkalosis. Poulus *et al.* (1974) infused an 8% bicarbonate solution into the blood during exercise in an attempt to correct acidemia resulting from exercise. Although these treatments did produce a significant alkalosis as indicated by blood chemistry measures, they had little effect on work capacity. Johnson and Black (1953), Kowalchuk *et al.* (1984) and Kindermann *et al.* (1977) reached a similar conclusion that alkalosis did not increase exercise capacity under either competitive conditions or in a laboratory setting. The findings of Magaria *et al.* (1971) and Johnson and Black (1953) have been criticized on the grounds that the dose of bicarbonate was inadequate to cause a substantial increase in the buffer reserve (Wilke *et al.*, 1983).

There are reports that the induction of alkalosis is associated with enhanced endurance during moderately heavy but not supramaximal exercise (Jones *et al.*, 1977; Kowal-

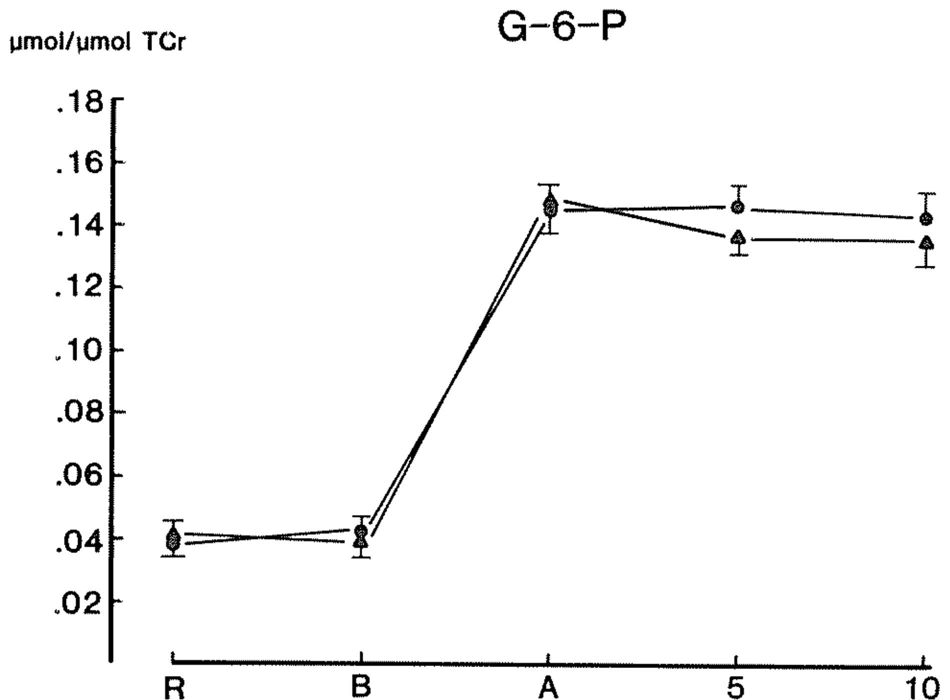

FIGURE 4 Glucose-6-phosphate concentration in muscle at rest before and after exercise and 5 and 10 min following exercise. Symbols are as for Fig. 1

chuck *et al.*, 1984; McCartney *et al.*, 1983; Sutton *et al.*, 1981). During supramaximal exercise there was no difference in plasma LA and pH between bicarbonate and placebo conditions (McCartney *et al.*, 1983). The proposed mechanism for this effect of enhanced endurance is one of increasing the efflux of protons from within the muscle cell to allow for a greater total glycolytic response as compared to the normal or acidotic state. This greater lactate efflux with bicarbonate administration is supported by studies on isolated muscles which have demonstrated that increasing the bicarbonate in the bathing medium results in a more rapid efflux of LA during electrically induced contractions (Mainwood and Worsley-Brown, 1975).

The concentrations of the metabolites in muscle and blood in the present study do not indicate that alkalosis significantly alters the metabolism of exercise. The depressions in glycogen, ATP, and creatine phosphate were similar under both the placebo and bicarbonate conditions. Moreover, the increases in LA in blood and muscle and G-6-P in muscle were not different between treatments. The decrease in ATP demonstrates that the intensity of exercise was such that its synthesis did not keep pace with its use. The increase in G-6-P is, together with the large LA accumulation, indicative of a major activation of the Embden-Meyerhof pathway. The maintenance of a high G-6-P concentration after exercise suggests either that its production continued at a high rate or that it was removed rather slowly. The rapid return of the $\dot{V}O_2$ towards rest after exercise and the observation that LA does not continue to rise supports the

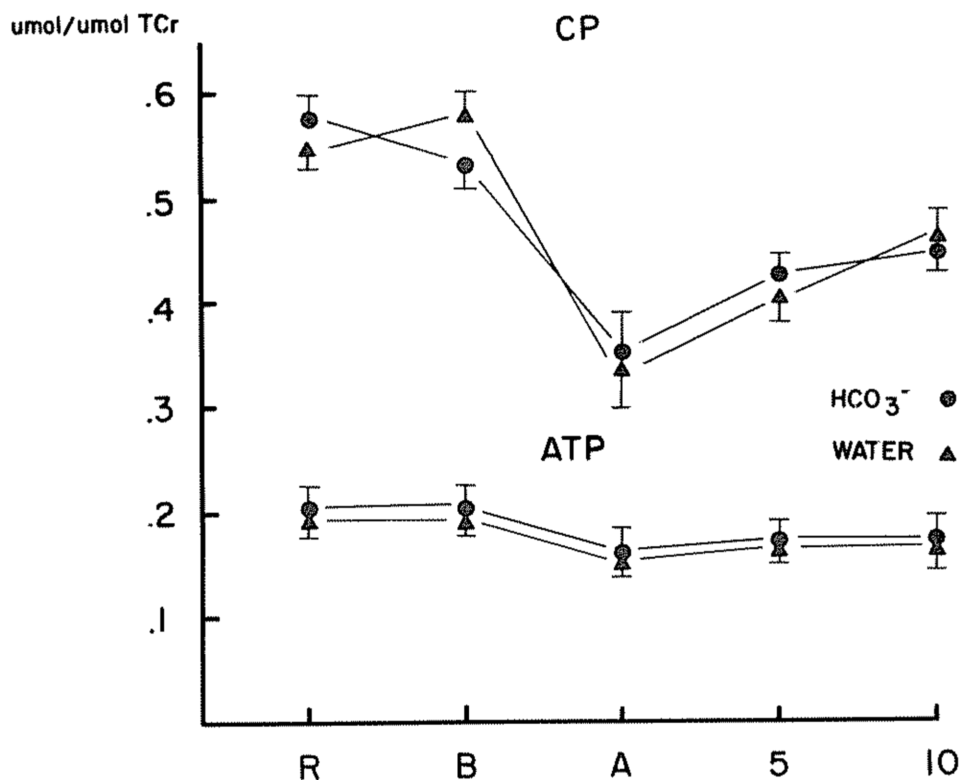

FIGURE 5 Creatine phosphate (CP) and adenosine triphosphate (ATP) changes in muscle at rest, before and after exercise and 5 and 10 min following exercise. Symbols are as in Fig. 1.

contention that it was being slowly removed rather than being rapidly produced.

The results of the present study revealed that the $PvCO_2$ increased following the oral administration of $NaHCO_3$. However, its effect on blood pH and PCO_2 in both arterial and venous blood during exercise was small. This can be attributed to the $PaCO_2$ being a function of the difference between the absorption of $NaHCO_3$ by the gut and the release of CO_2 from the blood perfusing the lung. The role of the ventilatory system in maintaining PCO_2 homeostasis of blood is illustrated by the differences in venous and arterial blood at rest. This was more apparent during exercise where the difference between the $PaCO_2$ and $PvCO_2$ for the control and bicarbonate treatments narrowed, and in the post-exercise period where it was practically nil. The similarity in blood pH during exercise between experimental conditions is indicative that the ventilatory response to exercise overrode the effects produced by bicarbonate administration. This could reflect a preferential distribution of blood flow to the skeletal muscle at the expense of the gut.

In this study the mean time to run 1600 m was 2.2 sec less ($P < 0.10$) after the bicarbonate treatment. This was equivalent to completing the 1600 m about 29 m prior to of the control condition. Secondly, the muscle pH after exercise for the bicarbonate

treatment was lower than that of the water condition. This could be explained by the faster run times which occurred for this condition. From some standpoints, the design of using an all out exercise test may not have been optimal for examining the effects of bicarbonate administration on the metabolic response to exercise. Thus, to fully elucidate the effects of a bicarbonate treatment on the metabolic response of muscle it would appear that these experiments should be repeated under conditions of a standard exercise test.

Acknowledgment

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References

Bergström, J. (1962). Muscle electrolytes in man. *Scand. J. Clin. Lab. Invest.* Suppl. 68.

Dennig, H., Talbott, J. H., Edwards, H. T. and Dill, D. B. (1931). Effect of acidosis and alkalosis upon capacity for work. *J. Clin. Invest.* **9**, 601–613.

Harris, R. E., Hultman, E. and Nordesjö, L.-O. (1974). Glycogen, glycolytic intermediates, and high-energy phosphates determined in biopsy samples of musculus quadriceps femoris of man at rest. Methods and variance of values. *Scand. J. Clin. Lab. Invest.* **33**, 109–122.

Johnson, W. R. and Black, D. H. (1953). Comparison of effects of certain blood alkalinizers and glucose upon competitive endurance performance. *J. Appl. Physiol.* **5**, 577–578.

Jones, N. L., Sutton, J. R., Taylor, R. and Toews, C. J. (1977). Effect of pH on cardiorespiratory and metabolic responses to exercise. *J. Appl. Physiol.* **43**, 959–964.

Kindermann, W., Keul, J. and Huber, G. (1977). Physical exercise after induced alkalosis (bicarbonate or tris-buffer). *Europ. J. Appl. Physiol.* **37**, 197–204.

Kowalchuk, J. M., Heigenhauser, G. J. F. and Jones, N. L. (1984). Effect of pH on metabolic and cardiorespiratory responses during progressive exercise. *J. Appl. Physiol.* **57**, 1558–1563.

Lamprecht, W. L. and Trautschold, I. (1974). Adenosine-5′-triphosphate determination with hexokinase and glucose-6-phosphate dehydrogenase. *In: Methods of Enzymatic Analysis*. Bergmeyer, H. U. (ed.). New York, Academic Press. pp. 2101–2110.

Lamprect, W. L., Stein, P., Heniz, F. and Weisser, H. (1974). Creatine phosphate, determination with hexokinase and glucose-6-phosphate dehydrogenase. *In: Methods of Enzymatic Analysis*. Bergmeyer, H. U. (ed.). New York, Academic Press. pp. 1775–1783.

Lang, G. and Michal, G. (1974). D-glucose-6-phosphate and D-fructose-6-phosphate. *In: Methods of Enzymatic Analysis*. Bergmeyer, H. U. (ed.). New York, Academic Press. pp. 1238–1242.

Lowry, O. H. and Passonneau, J. V. (1972). *A Flexible System of Enzymatic Analysis*. New York: Academic Press.

Mainwood, G. W. and Worsley-Brown, P. (1975). The effects of extracellular pH and buffer concentration on the efflux of lactate from frog sartorious muscle. *J. Physiol. (London)* **250**, 1–22.

Margaria, R., Aghemo, P. and Sassi, G. (1971). Effect of alkalosis on performance and lactate formation in supramaximal exercise. *Inter. Z. angew. Physiol.* **29**, 215–233.

McCartney, N., Heigenhauser, G. J. F. and Jones, N. L. (1983). Effects of pH on maximal power output and fatigue during short-term dynamic exercise. *J. Appl. Physiol.* **55**, 225–229.

Passonneau, J. V. and Lauderdale, V. R. (1974). A comparison of three methods of glycogen measurement in tissues. *Anal. Biochem.* **60**, 405–412.

Pette, D. and Reichmann, H. (1982). A method for quantitative extraction of enzymes and metabolites from tissue samples in the milligram range. *J. Histochem. Cytochem.* **30**, 401–402.

Poulus, A. J., Docter, H. J. and Westra, H. G. (1974). Acid-base balance and subjective feelings of fatigue during physical exercise. *Europ. J. Physiol.* **33**, 207–213.

Sutton, J. R., Jones, N. L. and Toews, C. J. (1981). Effect of pH on muscle glycolysis during exercise. *Clin. Sci.* **61**, 331–338.

Wilkes, D. Gledhill, N. and Smyth, R. (1983). Effect of acute induced metabolic alkalosis on 800-m racing time. *Med. Sci. Sports Exerc.* **15**, 277–280.

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 (Ehrsam *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