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EFFECT OF SODIUM BICARBONATE INGESTION ON BLOOD AND MUSCLE PH AND EXERCISE PERFORMANCE

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EFFECT OF SODIUM BICARBONATE INGESTION ON BLOOD AND MUSCLE pH AND EXERCISE PERFORMANCE

DISSertation

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of The Ohio State University

By

Jeffrey C. Rupp, B.S., M.A.

* * * * *

The Ohio State University

1982

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School of Health, Physical Education and Recreation
Dedicated to
Juanita B. Rupp
and
Corey M. Rupp
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Studies on causes of Muscular Fatigue. Professors Robert L. Bartels, E. L. Fox and Will Zuelzer, M.D.
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CHAPTER 1
INTRODUCTION

For many years intensive research has been under way in order to more fully understand the mechanisms underlying muscular fatigue. It is interesting to note that while this research has been performed, little is known about the cause of muscular fatigue. Simonsen (1) has defined fatigue as a reversible state of decreased physical and mental work capacity resulting from preceding work.

A number of theories have been advanced concerning the causes of muscular fatigue: (1) depletion of substances necessary for activity, (2) accumulation of substances producing fatigue, (3) changes of physicochemical state of substrate, and (4) disturbance of regulation and coordination (2). In addition it has recently been suggested that the muscle may fail to contract due to fatigue at the neuro-muscular junction (3).

Depletion of substances necessary for activity include adenosine triphosphate (ATP), creatine phosphate (CP), and muscle glycogen. The depletion of phosphagen stores as a cause of muscular fatigue was investigated by Karlson and Saltin (4) who found that exhaustion was not related to low CP levels in the working muscle. Fitts and Holloszy (5)
also showed in isolated frog sartorius muscle that the large
decline in muscle phosphagens during contractions occurred
before the decline in peak tension. Even when this muscle
was fully fatigued, there was still 76% of the resting
concentration of ATP available. These authors also found
a rapid rise in phosphagen levels during the first minutes
of recovery with little change in the ability of the muscle
to develop force. Recently however Sahlin, Palmskog, and
Hultman (6) determined that the amount of energy liberated
per mole of ATP utilized decreased from 12.9 Kcal at rest
to 11.0 Kcal after maximal exercise. The mechanism of this
phenomenon is unknown, but could possibly be related to the
hydrogen ion (H\(^+\)) concentration (3).

In a series of investigations by Gollnick and co-workers
(7,8) it was shown that glycogen levels were depleted at a
greater rate in fast-twitch (FT) muscle fibers when sprint-
like activities were employed and in slow-twitch (ST) fibers
when endurance activities were performed. These authors
concluded that during prolonged endurance activities, glyco-
gen stores in ST muscles are essentially exhausted. This
conclusion was suggested as a possible cause of muscular
fatigue by Saltin (9).

The theory that muscular fatigue is caused by the
accumulation of waste-products has received much attention.
In the classic work by Hill (10) over 50 years ago, it was
postulated that the accumulation of lactic acid might be a
cause of muscular fatigue. Since this time other investigators have suggested lactic acid and the resulting decline in pH to be a factor in muscular fatigue (11, 12, 13, 14). Recently it has been shown that an increase in H\(^+\) concentration can affect enzyme activity and their substrates (15).

Finally evidence has been accumulating that fatigue at the neuromuscular junction might be possible (15, 16, 17). Komi and Tesch (18) have suggested that this type of fatigue may be related to the ability of the nerve ending to release acetylcholine.

Thus it can be seen that many factors have been suggested as a cause of muscular fatigue. Of the theories presented, the accumulation of acidic waste products has special applications to athletic performance. It has been hypothesized by many investigators (18, 19, 20, 21) that the ingestion or infusion of alkaline substances may improve performance by increasing the buffering capacity of the blood and/or muscle. The results of these investigations have been conflicting. Some authors have found increased performances after alkalinization (18, 19, 20, 21), while others have failed to find any effects on performance (22, 23, 24). The reasons for this controversy may lie in the fact that the type of exercise (continuous or intermittent), duration, type of buffer used, and amount of buffer ingested or infused differed among the investigations.
It should also be noted that in all these investigations no measurements of muscle pH were made. Since the concentration of lactate in the blood is a result of not only the production in the muscle, but the rate of release, rate of removal by other organs like liver, heart and other skeletal muscles, and fluid shifts which occur as a result of exercise (25, 26), it is difficult if not impossible to make conclusions about the effect of buffers on intracellular mechanisms without measures of muscle pH. Thus it is clear that in order to help resolve this controversy more information is needed in order to clarify the effectiveness of buffers like bicarbonate as an ergogenic aid.

Statement of the Problem. It shall be the purpose of this investigation to quantify the effects of ingestion of 0.3 grams of sodium bicarbonate (NaHCO₃) per kilogram (Kg) of body weight on exercise performance, muscle and blood pH on four college age males. Specifically the following hypotheses will be tested:

\[ H_0 : \text{Performance time to exhaustion at 95% VO}_{2\text{max}} \text{ will not be significantly different compared to control values.} \]

\[ H_A : \text{Performance time to exhaustion will be significantly higher with ingestion of bicarbonate compared to control values.} \]
$H_0$: The muscle pH at the end of the bicarbonate ingestion period will not be significantly different from control values.

$H_A$: Muscle pH at the end of the bicarbonate ingestion period will be higher than control values.

$H_0$: The muscle pH at the end of the exercise tests will not be significantly different between conditions.

$H_A$: The muscle pH will be significantly higher at the end of exercise during bicarbonate ingestion compared to control values.

$H_0$: The cardiovascular variables, heart rate maximum ($HR_{max}$), $VO_{2max}$ will not differ between conditions.

$H_A$: Bicarbonate ingestion will result in significantly higher maximum heart rates and maximum oxygen consumptions.

Significance of the Problem. As a result of this investigation more information should be gained pertaining to the theory that muscular fatigue is caused by a decline in muscle pH.

Definition of Terms.

$VO_2$: Amount of oxygen consumed per minute in liters

$VCO_2$: Amount of carbon dioxide produced per minute in liters

$V_E$: Expired ventilation per minute in liters BTPS

$NaHCO_3$: Sodium bicarbonate
$L_c$ : Lactose control group

KPM: min$^{-1}$ : Kilopond meter per minute

ADP : Adenosine diphosphate

AMP : Adenosine monophosphate

ATP : Adenosine triphosphate

CP : Creatine phosphate

Hr : Hour

LDH : Lactate dehydrogenase

NAD : Nicotinamide adenine dinucleotide

NADH : Nicotinamide adenine dinucleotide reduced form

PCO$_2$ : Carbon dioxide tension in arterialized venous blood

pH : $-\log [H^+]

PO$_2$ : Oxygen tension in arterialized venous blood

Pi : Inorganic phosphate

SD : Standard Deviation

SE : Standard Error of Mean
CHAPTER 2
REVIEW OF LITERATURE

It is rather well established that the major cause of the decrease in pH seen during maximal exercise is due to the accumulation of lactic acid. Since the production of lactic acid results in an equivalent release of $H^+$, it becomes essential that the concentration of $H^+$ be controlled by various buffering processes in order that the pH does not decline to a point incompatible with life.

Buffering Processes in Muscle. Sahlin and co-workers (6) have found that the concentration of inorganic phosphate (Pi) in the quadriceps muscle of man is 12 mmoles per liter. Thus the potential for buffering during metabolic acidosis is high since the intracellular concentration of Pi is high. It has been calculated that the stoichio-
metrical uptake of $H^+$ ions when muscle pH decreases from 7.08 to 6.6 is 27 moles per mole of Pi, thus resulting in a total $H^+$ uptake of 3.2 mmole per liter of muscle water (2).

The bicarbonate ion ($HCO_3^-$) can combine with the $H^+$ during high intensity activity which forms carbonic acid ($H_2CO_3$) which at physiological pH values immediately dissociates into carbon dioxide ($CO_2$) and water ($H_2O$) both
of which are easily removed. Sahlin and co-workers (27) have shown that the bicarbonate concentration decreases from 10.2 mmole per liter at rest to 3 mmole per liter in muscle at the end of exhaustive exercise, with an equivalent uptake of H⁺.

Muscle proteins also play an important role in the uptake of H⁺ ions during exercise. Since the concentration of protein in human muscle is high the potential for buffering by amino acids is high. However, the pKₐ for free amino acids is less than 3 for the alpha carboxal group and greater than 9 for the alpha amino group and thus are fully ionized in the cell and cannot contribute any buffer capacity (2). Thus the buffering power of proteins, amino acids and peptides is dependent on the acid-base characteristics of the R group. Only histidine has a pKₐ value that falls within physiological pH ranges (2). Bergstrom and coworkers (28) determined the concentration of histidine in the quadraceps muscle to be .38 mmol/liter. If this value is assumed to be correct then the buffering capacity of this amino acid is relatively small. Possibly more important is the fact that when the amino acids are incorporated into peptides the pKₐ of the R group changes. Lenz and Martell (29) found that when histidine is incorporated into the depeptide carnosine the molar buffer value for carnosine will be higher than for histidine. Since the concentration of carnosine is high in many muscles this
may be a more important buffering source. In fact Bergstrom and coworkers (28) determined that the carnosine content of human quadraceps muscle is 5.5 mmol/liter of muscle water.

Finally there are metabolic processes which can either consume or produce $H^+$ ions. Quantitatively the most important metabolic processes are lactate production and the synthesis or degradation of CP. The regulation point of glycolysis is generally agreed to be the enzyme phosphofructokinase. This enzyme is extremely sensitive to changes in pH (30). The utilization of CP causes an absorption of $H^+$ ions while synthesis causes a release of $H^+$ ions (2). Since the content of CP is higher in fast twitch fibers it seems reasonable to suspect that those muscles which have a high percentage of FT fibers have a higher capacity for metabolic buffering.

It had also been shown by a number of investigators (31, 32, 33), that the myosin ATPase activity in skeletal muscle decreased by 25% when pH decreased from 7.0 to 6.5. It was also shown that an increased amount of calcium was required to develop 50% of maximum ATP-splitting with a lowered pH.

In summary it is clear that changes in $H^+$ ion activity can have very definite effects on the rates of reactions and possibly the ability of the muscle to develop force. The next question that needs to be addressed is what effect does this have on exercise performance.
Acid-Base Balance and Exercise Performance. The idea that altered acid-base status may affect exercise performance is not new. As early as 1931 Denning and others (18) found an increase in performance plus a higher peak lactate concentration in runners when subjects were given bicarbonate. It was also concluded that during ammonium chloride ingestion the ability to buffer lactic acid and to accumulate an oxygen debt is greatly decreased during exercise. Dorrow and coworkers (19) found similar results in swimmers.

In a study by Poulus and Doeter and Westra, (34) changes in acid-base parameters by infusion of bicarbonate and the subjective feeling of fatigue during bicycle exercise was investigated in six subjects. The amount of bicarbonate infused average 270 ml. Subjective feelings of fatigue, heart rate, blood pressure, ventilation and oxygen consumption were measured on two test occasions, one with sodium chloride infusion and the other with bicarbonate infusion. Results showed no difference in maximum performance or cardiovascular variables with the exception of CO₂ output on the bicarbonate infusion. Results also showed that bicarbonate infusion was effective in keeping the H⁺ ion concentration close to resting levels. These authors concluded that bicarbonate infusion had no effect on the subjective feeling of fatigue and that pH changes during exercise do not affect cardiovascular function or maximal oxygen consumption.
A number of observations should be noted in this study. First the authors do not give information on the time to exhaustion, only what the maximal workload was. It is possible that cardiovascular parameters were not affected but endurance time might be. Also measurements were not made of muscle pH or lactate concentration. It is difficult to make inferences about occurrences in the muscle from extracellular measurements. Finally the possibility exists that the amount of bicarbonate infusion was not enough to affect a change in performance. This might be possible since the change in $\text{H}^+$ concentration after the bicarbonate infusion was only 1nmol/liter or a rise in pH of .01 pH unit.

Kinderman, Keul and Huber (35) investigated the effects of bicarbonate and Tris-buffer infusions on performance times of 400 meter runs. Run time, maximal lactate concentration and heart rates of ten males who ran 400 meters on two occasions. Subjects were infused with 130 mls of Tris-buffer or 8.4% sodium bicarbonate. After buffer infusions pH, $\text{PCO}_2$, base excess and standard bicarbonate increased. Results showed that a reduction in work-related acidosis by infusion of buffers caused no improvement in performance, or any difference in maximal lactate concentrations between infusion and control values.

This study although open to criticism because of its design, does raise an interesting question on the
effectiveness of extra and intracellular buffering on short term high intensity exercise. Tris buffer reportedly penetrates the muscular space rapidly (35) and so the presence of excess buffer only in the extracellular space cannot account for the lack of improved performance. However since no measures of muscle pH or lactate were made, it is difficult to determine this definitely. Due to the nature and purpose of this investigation the design and thus the conclusions would be strengthened if the control session had been run with an infusion of sodium chloride. This would have allowed for a blind or even double blind study which is better suited for this type of study, and the elimination of subject and investigator bias. It is also possible that the choice of work test was too short. The 400 meter run although supposedly 80% glycolytic in nature (36), still involves a significant contribution by phosphagens. As stated previously the degradation of CP stores absorbs H+ ions. It is possible that phosphagens contribute enough of the metabolic energy demand to offset the excess buffer present, however this seems highly unlikely. Another possibility might be the fact that at this work intensity, CP stores are essentially exhausted, and glycolysis cannot provide the energy required at this supra-maximal workload, and fatigue at the neuro-muscular junction occurs. If this is the case, then the increased buffering capacity may not be effective.
In a recent study by Jones, Sutton, Taylor and Toews (37), the effects of bicarbonate ingestion and ammonium chloride ingestion on exercise performance was investigated. This double blind study involved five male subjects who during the experimental sessions ingested sodium bicarbonate and ammonium chloride. On the third occasion calcium carbonate was ingested as a control. The dose amounted to .3 gram per kilogram of body weight, over a three hour period. The work protocol began with a workload corresponding to 33% of subjects VO_2\text{max} for a period of 20 minutes. This was followed by 20 minutes at 66% VO_2\text{max}, and finally 95% until exhaustion occurred. Results of this study showed a definite separation in pH values following the ingestion period, with bicarbonate causing a higher pH compared with control, and ammonium chloride serving to acidify the blood. The endurance time at the 95% workload was significantly higher after bicarbonate ingestion and lower after ammonium chloride ingestion. This amounted to approximately a doubling of the time to exhaustion with bicarbonate compared to the ammonium chloride ingestion. Cardiovascular parameters (including cardiac output), and gas exchange parameters were not significantly different among the conditions. The lactate concentration was significantly higher after bicarbonate ingestion and lowest after ammonium chloride ingestion. The authors suggest that when alkalotic conditions prevail, increased times to exhaustion and higher
lactates were found most likely due to delayed inhibition of glycolysis by decreased pH (the reverse being true then for acidic conditions).

This investigation from a design standpoint is much easier to draw conclusions from due to the double blind nature in which it was conducted. Possible subject and investigator bias was eliminated in this manner. However as in previous investigations no measure of muscle lactate or pH were made, and the authors suggest these to be a possible cause of their results.

The discrepancy between this and other studies quoted on acid-base balance and exercise performance probably stems from a number of factors. First the type and duration of exercise performed varied between studies, also the method of inducing the acid-base change differed, that is whether the dose was ingested or infused, and finally the designs themselves varied, that is some subjects and investigators knew what was being given and others did not. Of particular interest is the fact that studies involving high intensity short duration exercises fail to show improved performances, while relatively longer duration exercises followed by high intensity exercise to exhaustion show improved performances after ingestion of bicarbonate. This phenomenon remains unexplained and open to further investigation.

In a paper by Cerretelli (38) it has been suggested that a limiting factor in anaerobic energy release may be
blood pH. Hermansen (39) tested this hypothesis by measuring lactate and pH in blood after 5 work periods. Results showed that a marked increase in blood lactate with an accompanying decrease in pH after each work bout. Since the subjects worked to exhaustion with each bout and blood pH continued to decrease the idea that the blood pH was the limiting factor in exercise performance is questionable. Hermansen suggested that the intracellular pH may be the limiting factor.

Diamant, Karlson and Saltin (40) found a range in muscle lactate concentration of 3.0mm at rest to 19.1 mmoles per kilogram of wet weight tissue after maximal exercise on a cycle ergometer. These authors found a gradient for lactate from muscle to blood at rest and especially after exercise to exhaustion. The authors also suggest that the real intracellular lactate concentration is even higher since the needle biopsy method introduced by Bergstrom (41) contains 25% extracellular water.

Sahlin, Harris, Nylins, and Hultman (42) studied lactate content and pH in muscle after exercise. These authors found that muscle pH decreased from 7.08 to 6.6 at exhaustion. The decrease in muscle pH was linearly related to muscle content of lactate + pyruvate. Lactate content was very high in muscle after exercise (2-3 times that of blood).
Sahlin, Alvestrand, Brandt and Hultman (43) have shown that after exhaustive bicycle exercise, lactate continues to be released from the working muscle well into the recovery period. It was also found that the base deficit in arterial blood increased by the same amount as lactate + pyruvate indicating that H⁺ ions accompany the transport of lactate into the blood. The authors concluded that the increase in H⁺ ions in blood is equivalent to the accumulation of lactate plus pyruvate during exercise, but during early recovery it is possible that H⁺ are released at a faster rate than lactate from exhausted muscle. It was also suggested that during early recovery a rapid resynthesis of CP occurs, which is associated with a release of H⁺ ions (2). This could cause a change in the membrane permeability which could account for the faster efflux of H⁺ ions than lactate.

It is interesting to note that in the studies that found increased performances after ingestion of bicarbonate (18, 19, 20, 21), no changes in oxygen consumption were found. In a study by Patterson and Sullivan (44) the determinants of oxygen uptake during bicarbonate infusion were studied in dogs. Previously it had been shown that passive hyperventilation increases the total body VO₂ in proportion to the rise in pH (45). In this study respiratory and metabolic alkalosis and their effects on VO₂ were studied in dogs. Results showed that an increase in PaCO₂
sufficient to suppress the increase in VO$_2$ that would be expected from the rise in pH occurred. It is possible that this is what occurs in humans during ingestion, but is still open to investigation.

Hermansen and Osnes (46) investigated blood and muscle pH after maximal exercise in 13 subjects. The type of exercise used was both continuous and intermittent on a cycle ergometer and treadmill. Needle biopsy specimens of the quadriceps muscle and capillary blood samples were taken. Results showed that the capillary blood pH decreased to 7.11 and 6.94 after continuous and intermittent exercise respectively, while muscle pH decreased to 6.4 essentially the same for both types of exercise. The authors conclude that muscle pH may be the limiting factor in exercise to exhaustion.

In summary it is clear that little is known about the cause or causes of muscular fatigue. It has been suggested that pH of the exercising muscle may be the limiting factor during relatively high intensity exercise. It has also been suggested that alterations of extracellular acid-base balance may elicit a corresponding change in work performance. Since most of these studies have not looked at acid-base changes in the working muscle it is difficult to interpret their results and shall form the basis for this investigation.
CHAPTER 3

METHODS AND PROCEDURES

It was the purpose of this investigation to study the effects of sodium bicarbonate ingestion on muscle pH and exercise performance during maximal exercise on a stationary ergometer.

Subject Selection

The subjects in this investigation were four male volunteers from The Ohio State University. Subjects were relatively untrained but active participants in various physical education activities. Subject characteristics are given in Table 1. From those subjects expressing interest in the study four were chosen on the basis of their ability to tolerate the blood and biopsy procedures.

Subjects completed a medical health history which was reviewed by a physician who served as Medical Monitor for this investigation. Subjects also underwent a preliminary physical examination prior to participation. The entire protocol was approved by and conducted under the supervision of the Human Subject Review Committee at The Ohio State University. The subjects were informed of the nature and
purpose of this study and were aware of the potential risks and side effects when the informed consent document was given.

Procedures

Subjects reported to the laboratory in a post-absorptive state of four hours. The laboratory was located at 129 Larkins Hall at The Ohio State University and was controlled at a temperature of approximately 23 degrees C. After giving informed consent subjects began the ingestion of .3 gram/kg of body weight of either sodium bicarbonate or lactose in capsule form over a three hour period. The order in which the subjects took the dose was randomly assigned between the subjects. Neither the subjects nor the investigators were aware of which substance was being ingested on the two testing sessions. The particular dose used was chosen based on previous work by other investigators showing this to be the highest tolerable dose that could be taken without producing symptoms of nausea and diarrhea (37). Prior to the start of the ingestion period a plastic catheter was inserted into a prominent hand vein and a resting blood sample taken. All blood samples were analyzed for lactate, pH, PO₂, PCO₂, Base Excess or Deficit, and Standard Bicarbonate. Blood samples were taken at various times during the ingestion period, exercise and during recovery as shown in Figure 1.
Gas Exchange and Cardiovascular Measures. Minute ventilation (VE), oxygen consumption (VO2), carbon dioxide output (VCO2), mixed oxygen (PEO2) and carbon dioxide (FECO2) were measured with a Beckman LB2 and OM 11 gas analysis system. Inspired ventilation was measured using a Parkinson-Cowan CD4 dry gas meter with output signals to a Tracewell Digital Ventilation meter. Subjects breathed through a low resistance Rudolph valve. Exhaled air moved through a 5 liter mixing chamber before sampling. All ventilations were corrected to BTPS conditions while VO2 and VCO2 was corrected to STPD conditions. Heart rates were measured on a Hewlett-Packard model 1500A electocardiograph.

Biopsy Procedure. Following the three hour ingestion period a resting skeletal needle biopsy was taken from the vastus lateralis muscle according to a modification of the procedure described by Bergstrom (41). Another biopsy specimen was taken immediately following the termination of the exercise test. All biopsies were immediately frozen (within 5 seconds) in liquid nitrogen while still in the needle. Muscle biopsies after removal from the needle in a cold room at -20C were stored in liquid nitrogen or in a -60C freezer until subsequent analysis.

Ergometer Protocol. A Fitron cycle ergometer was used in this investigation allowing the subject to pedal at a constant revolution (60 rpm) at each workload. After the
initial biopsy was taken subjects pedalled at 66% VO\textsubscript{2}max for 20 minutes. This was followed by increasing to 95% VO\textsubscript{2}max until exhaustion. The workloads were determined from a previous work test approximating the time of the experimental protocol.

**Blood Analysis.** Arterialized venous blood samples were obtained by warming the hand in water at approximately 45C prior to obtaining the specimen. The first 3ml sample was immediately deproteinized in 8% perchloric acid and stored at 4C. A second 2ml sample was taken anaerobically into heparinized syringes and stored on ice at 0-4C until analysis for pH and blood gasses (within one hour) on a Corning model 168 blood gas analyzer. PO2 values were used only to indicate adequacy of arterialization.

Blood lactate was performed according to Sigma Technical Bulletin 826-UV (47). This procedure consisted of the spectrophotometric determination of NADH levels at 340 nmeters and comparing to a blank cuvette. The change in absorbance was divided by the coefficient of extinction in order to determine the mM concentration of lactate in the cuvette. This was in turn multiplied by the appropriate dilution factors to obtain the concentration in blood expressed as millimoles (mmoles) per liter. All assays were performed in duplicate.
Biopsy Analysis. The biopsy specimens were weighed in a specially designed cryostat at -20°C on a Perkin Elmer model AD1 autobalance after being sectioned for duplicate assays for pH.

Muscle pH was determined according to the method described by Hermansen and Osnes (46) and modified by Sahlin (49). The sample was homogenized at 0°C in a 20 ml/gram medium containing 5mM of iodoacetate, 145mM KCl, and 10mM NaCl. Approximately 50 μl of this homogenate was rapidly sucked into the electrode of a Radiometer Model BMS MK3 Blood Gas System with Microelectrode. It should be noted here that the pH obtained is not a true measure of intracellular pH due to the content of both intra and extracellular water, but is assumed to be an accurate reflection of the pH of the muscle as a whole (47). The reliability of this method was studied by Hermansen and Osnes (47) and found to have an error of .03 ± .004 pH units for 48 paired analysis of the same homogenate and .03 ± .01 pH units for 11 paired specimens. The muscle pH method used in this investigation has been shown to provide stable pH values by the addition of IAA to the homogenate, while the procedures of Hermansen and Osnes (47) showed a decline in pH during the measurement time with all samples except those taken immediately after exhaustive exercise (most acidic samples). Sahlin investigated the pH values in two specimens using both techniques (with and without IAA) and found no
differences in pH values, and a prevention of the decline in pH during the measurement period.

Data Analysis

Statistical analysis for significant differences between means was performed using a paired t test for pH, peak lactate in blood, gas exchange parameters, heart rate max, and time to exhaustion at the highest workload. The .05 level of confidence was used in all tests.

Specifically tests were made for end ingestion pH, pH at the onset of the 95% VO2max workload, and end exercise pH. The test for peak blood lactate was made using the highest value obtained after exercise. Muscle pH was compared between conditions at rest and immediately post exercise.
CHAPTER 4

RESULTS AND DISCUSSIONS

The purpose of this investigation was to study the effects of altered acid-base balance, specifically the ingestion of .3 gram per kilogram of body weight of NaHCO3 on exercise performance. Four college males were studied on two occasions, once with NaHCO3 and the other with lactose which served as control. Various parameters were measured in order to study the reaction to the NaHCO3 treatment including time to exhaustion at the highest workload, blood pH and lactate, standard bicarbonate and base deficit, in addition muscle biopsy specimens were taken and analyzed for pH.

Cardiovascular Measures

Results showed that there was no significant difference in maximum oxygen consumption between the lactose and NaHCO3 sessions (3.998 ± 0.466 L/min and 3.905 ± 0.499 L/min respectively). In addition no significant difference was found in maximum heart rate between the sessions (180 ± 6.65 bpm and 183 ± 4.05 bpm).
Maximum minute ventilation was not significantly different between the lactose and NaHCO₃ sessions (5.74 L/min and 6.77 L/min).

**Blood Parameters**

Figures 1, 2, and 3 show the changes in blood pH, base deficit and standard bicarbonate respectively. It is clear that the NaHCO₃ treatment resulted in a definite separation from control values. Table 2 shows the difference (Δ) between the NaHCO₃ and control sessions for blood pH, base deficit and standard bicarbonate. The mean Δ value was computed from time 0 (end ingestion) through recovery 30. The ΔpH was .084 ± .009. The Δbase deficit was 5.47 ± .55 and the ΔHCO₃⁻ was 3.78 ± .56.

A significant difference (p < 05) was found between the control and NaHCO₃ sessions in blood pH at the end of the ingestion period (7.338 ± .023 and 7.424 ± .013 respectively). A significant difference (p < 05) was found between sessions in standard bicarbonate at the end of the ingestion period (25.5 ± 1.08 mEq/L for the control and NaHCO₃ sessions respectively). A significant difference (p < 05) was also found between sessions in base deficit at the end of the ingestion period (-.75 ± .18 and 5.725 ± .38 mEq/L). Base excess at the onset of the 95% workload was significantly different (p < .05) between the sessions (-6.625 ± 1.15 and .825 ± 1.24 mEq/L) as was standard bicarbonate (19.25 ± 1.44 and 24.25 ± 1.11 mEq/L).
When base deficit was plotted against lactate concentration during the exercise test a high correlation was found (−.994 for control and −.994 for NaHCO₃) while no significant difference was found in the slopes of these graphs (−1.242 for control and −1.187 for NaHCO₃, see Figure 5).

When standard bicarbonate was plotted against lactate concentrations during the exercise test a high correlation was also found (−.970 for control and −.990 for NaHCO₃, see Figure 6) while no difference was found in the slopes (−1.26 for NaHCO₃ and −1.02 for control).

A significant difference (p < 05) was found between sessions for peak lactate concentration (13.18 ± .623 and 14.15 ± .592 mmoles/L for control and NaHCO₃ respectively, see Figure 4). The error in double determinations of peak lactate concentrations for both conditions was .261 ± .07 mmole/Liter.

**Time To Exhaustion**

It was found that there was a significant difference (p < 05) in the time to exhaustion at the highest workload of 95% V0₂max between the control and NaHCO₃ treatments (214 ± 46.6s and 287 ± 61.98s respectively, see Table 2).

**Muscle pH**

Resting muscle pH for both treatments was found to be 6.92 ± .02. The error in double determinations of
the same homogenate for both sessions was .047 ± .007. The error for split sample pH determinations was .082 ± .015. Exercised muscle pH determinations was 6.706 ± .021. There was no significant difference for resting pH between the sessions (6.918 ± .016 for control and 6.92 ± .05 for NaHCO₃). In addition there was no significant difference for end exercise pH between the sessions (6.667 ± .05 for control and 6.733 ± .068 for NaHCO₃).

**DISCUSSION**

Figures 1, 2, 3 show the changes in blood pH, base deficit and standard bicarbonate, respectively. It is clear that the NaHCO₃ treatment resulted in a definite separation from control values at rest.

From Figures 1, 2 and 3 it is clear that the bicarbonate ingestion had an effect on pH, base deficit and bicarbonate concentration. Table 2 shows that the reaction to the two treatments was similar since the standard errors of the mean were so low. Thus the bicarbonate treatment simply raised blood pH, base excess and bicarbonate concentration by the means shown. This difference was maintained throughout the exercise and recovery period. It is also clear that blood pH is not a limiting factor in high intensity work since the subjects worked longer with the bicarbonate treatment, yet during the control session blood pH was significantly lower at the end of exercise.
There is little doubt therefore that the ability of the blood to buffer $\text{H}^+$ ions was improved as a result of the NaHCO$_3$ ingestion. It is also clear from Figures 1, 2, 3 that pH, base excess and standard bicarbonate are at higher levels just prior to the onset of the 95% VO$_{2\text{max}}$ workload compared to the control value. However these results must be considered cautiously since it has been well documented that blood pH is not a limiting factor in high intensity exercise (46). Thus just because the buffer capacity of blood is higher does not necessarily mean that intracellular buffer capacity has been increased.

The significantly higher blood lactate concentration shown in Figure 4 is in agreement with the study by Jones and coworkers (37). This increase in blood lactate could be accounted for by a number of mechanisms. The blood lactate concentration seen in blood at any given time is a reflection of the rate of production in working muscle, rate of release from working muscle, rate of uptake by liver and other organs, and fluid shifts which can occur but are usually limited to exercises of longer duration than used in this investigation. Thus blood lactate values must also be considered with reservation, however it seems reasonable to assume that whatever the case may be, changes in extracellular pH are at least a part of the cause in the difference seen.
Figures 5 and 6 show the relationship between base deficit and standard bicarbonate and the increase in lactate concentration in blood during exercise. It is clear from these strong negative correlations that the bicarbonate ion concentration is decreasing in direct relationship with increasing lactate concentration in blood. In should also be noted here that lactate is not the only acid that is produced during heavy exercise, pyruvate will also tend to increase but this amount is negligible since the equilibrium constant lies far in favor of the reduction to lactate.

The increase in time to exhaustion at the highest workload is also in agreement with the study by Jones and coworkers (37). Due to the design of this study it can be assumed that the increases were not by chance and that the experimental variable is involved in the difference seen.

It is interesting to note that the cardiovascular variables measured were not different between the sessions. This is in agreement with other studies (35, 37). Studies in vitro have shown an increase in resting VO2 when the pH was raised. Karetsky and Cain (50) demonstrated in man that pH changes induced by hyperventilation or CO2 breathing caused an increase in resting VO2 in a linear and inverse relationship to PCO2. Several mechanisms could explain the lack of a consistent change in VO2 between the two conditions in this study. First, in the previous studies the
rise in resting VO2 was associated with very low PCO2 values which did not occur in this investigation. It is possible that the alkalosis induced was not severe enough to elicit this change. It is also possible that an increase in arterial PCO2 occurs as a result of the dissociation of the HCO$_3^-$ ion which could suppress the increase in VO2. This was shown in dogs by Patterson and Sullivan (51) who found that during the infusion of NaHCO3 in passively ventilated dogs that an increase in arterial PCO2 tension occurred most likely due to the dissociation of the HCO$_3^-$ ion and suppressed the increase in VO2 that would have been expected from the rise in pH. How this situation might apply to exercising humans is not clear, as no significant difference was found in PCO$_2$ between conditions in this investigation.

The next question that needs to be addressed is what effect did the increase in blood pH have on resting muscle pH. It has been suggested (37) that the increase in endurance time and blood lactate is a result of a delay in the inhibition of PFK activity in the muscle. It is well documented (52) that PFK activity is severely inhibited at a pH of 6.4. It is logical to hypothesize that an increase in the buffer capacity of muscle would enable the muscle to contract longer before some critical pH was reached. Results of this investigation show no change in resting muscle pH as a result of NaHCO3 ingestion. This was an interesting result. It is known that the bicarbonate
ion is not readily permeable to the muscle primarily due to its charged nature. However it was postulated that the rise in pH in extracellular fluid might cause a larger gradient for translocation of the H\(^+\) out of the muscle. Since there is no mechanism to distinguish between an inward flux of HCO\(_3^-\) or outward flux of H\(^+\) this is an area requiring further study. It remains that since no consistent change in resting muscle pH was found, the theory that an increase in extracellular buffer capacity results in an increase in the muscle buffering capacity must be questioned.

In contrast to these findings, results also showed that the pH declined in muscle to essentially the same values for control and HCO\(_3^-\) sessions. As stated previously blood pH does not seem to be limiting in high intensity exercise, because repeated bouts of exercise demonstrated that the muscle pH reached a limiting value with each bout (46), while blood pH continued to decline.

A number of investigators have shown in vitro that by creating an alkaline environment around the muscle cell it is possible to increase the rate of lactate efflux from the cell (2, 53, 54, 55). It seems quite possible in light of the findings of this investigation that the bicarbonate treatment increased the permeability of the muscle cell for lactate and thus delayed the onset of fatigue. Since the production of lactate in the muscle also releases an
equivalent amount of H\(^{+}\) ions, it seems reasonable to question whether the rate of lactate transport out of muscle is equal to the amount of hydrogen ion transport from muscle. Sahlin (2) determined that during exercise lactate efflux is accompanied by an equivalent amount of H\(^{+}\). The exception to this seems to be during the early phases of recovery where H\(^{+}\) are transported out of the muscle in excess of lactate.

Thus it seems reasonable to explain the lack of change in muscle pH at rest with bicarbonate treatment as being due to a low permeability of the muscle to the HCO\(_3\)\(^{-}\) ion. However the alkaline environment created around the muscle may increase the permeability for lactate and thus the H\(^{+}\) during exercise, which could delay the onset of muscular fatigue due to inhibition of muscle glycolysis.

It must be recognized that PFK activity is not the only mechanism by which a lowered pH may inhibit muscle contraction. It is generally accepted that muscle contraction is initiated by a release of calcium from the sarcoplasmic reticulum. This in turn activates ATPase which will cause the breakdown of ATP and allow the formation of the cross-bridge linkage. Schadler (56) showed that the activity of ATPase decreases approximately 25% when the pH decreases from 7.0 to 6.5. Portzehl and coworkers (57) showed that under these conditions it required a greater amount of calcium to achieve the activation under low pH conditions. It has also been shown in vitro by Nakamaru and Schwartz (58)
that the protein binding of calcium in the sarcoplasmic reticulum increased when the pH decreased to 6.5.

It has even been shown that the amount of energy liberated when one mole of ATP is hydrolyzed is affected by pH among other factors. Sahlin (2) has suggested that the minimum energy required to break and construct a new bond may under low pH conditions be too high to be met by ATP hydrolysis due to the effect of low pH on the amount of energy liberated.

As a result of this study it is clear that under these conditions there is an alteration in work performance, specifically the time to exhaustion at a relatively high workload preceded by a relatively low workload with the ingestion of .3 grams per Kg of body weight of NaHCO3. It must be made clear at this time that the purpose of this investigation was to study a proposed cause of muscular fatigue, namely a decline in muscle pH. It was not the purpose of this study to test NaHCO3 as an ergogenic aid. The effects of NaHCO3 ingestion under controlled conditions by unqualified individuals are not known and are potentially dangerous.

The results of this investigation should not be interpreted by the reader as an endorsement by the authors of the use of NaHCO3 or any other ergogenic aid whose sole purpose is to artificially improve the work performance of athletes. As stated by so many well informed coaches, exercise
scientists and even athletes themselves, "There is no substitute for hard work and determination."
CHAPTER 5

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to study the effects of .3 grams NaHCO₃ ingestion on exercise performance. Four college age healthy males volunteered as subjects and underwent an initial graded exercise test to determine VO₂max. Subjects then reported to the laboratory on two occasions in a post absorptive state of 4 hours and began to ingest either NaHCO₃ or lactose which served as control in capsule form. Neither the subject nor the investigator knew which substance was being ingested.

Resting blood samples were taken from an indwelling venous catheter placed in a prominent hand vein and pre-warmed in order to arterialize the blood. Samples were also taken at various times during the exercise test and during recovery and analyzed for lactate, blood gases, base deficit and standard bicarbonate. Two needle biopsies were also taken, one at the end of the three hour ingestion period and one immediately after exercise and analyzed for pH.
The work protocol consisted of 20 minutes of cycling at 66% VO$_{2\text{max}}$ followed by 95% VO$_{2\text{max}}$ until volitional exhaustion.

CONCLUSIONS

As a result of this investigation it was concluded that:

1. Bicarbonate ingestion under these conditions results in a significant increase in work time to exhaustion.

2. Bicarbonate ingestion under these conditions does not alter resting muscle pH or increase muscle buffer capacity.

3. Muscle pH is at least one important limiting factor in high intensity work as the muscle pH declined to the same values with each treatment.

4. Bicarbonate ingestion does not affect central cardiovascular measures (heart rate and VO2) for reasons still not clear, but possibly due to the lack of extreme alkalotic conditions.

RECOMMENDATIONS FOR FURTHER RESEARCH

As a result of this investigation and others there are still many questions left to be answered. It was suggested that bicarbonate ingestion does not increase the muscle buffer capacity. This was inferred from the fact that
resting muscle pH was not altered as a result of bicarbonate ingestion. It is possible to measure muscle buffer capacity directly by titration with HCl, where the buffer capacity is equal to the slope of the titration curve at a given pH. A study of this nature would help clarify the mechanism of the increase in performance time.

It was also suggested in this and other studies that ingestion of bicarbonate may increase the permeability of the muscle membrane for lactate and H⁺. A study involving the use of a progressive exercise test to exhaustion after bicarbonate ingestion is needed. Specifically the onset of blood lactate or the so-called "Anaerobic Threshold" (AT) needs to be studied. If the bicarbonate ingestion results in an AT occurring earlier this would support the changed permeability theory.

In any study involving the use of some substance, it is important to keep the design of the study in mind. It should be double blind in nature in order to control for subject and investigator bias. This necessitates the use of capsules or infusion of a control substance like sodium chloride. In addition the order of testing should be randomized so that any Hawthorne effect is negated.
### TABLE 1

**SUBJECT CHARACTERISTICS**

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age</th>
<th>Weight</th>
<th>VO\textsubscript{2}\textsubscript{max} (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25</td>
<td>67.04</td>
<td>3.087</td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>75.91</td>
<td>4.116</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>66.70</td>
<td>3.074</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>81.82</td>
<td>4.456</td>
</tr>
</tbody>
</table>

| X           | 25.75 | 72.87 | 3.68 |
| SE          | 1.11  | 3.67  | .355 |
### TABLE 2

Δ VALUES (NaHCO₃ - CONTROL)

<table>
<thead>
<tr>
<th>Rest</th>
<th>Blood pH</th>
<th>Base Deficit</th>
<th>HCO₃⁻</th>
</tr>
</thead>
<tbody>
<tr>
<td>-120</td>
<td>.033</td>
<td>.917</td>
<td>.5</td>
</tr>
<tr>
<td>-60</td>
<td>.062</td>
<td>2.605</td>
<td>2.75</td>
</tr>
<tr>
<td>0</td>
<td>.086</td>
<td>6.475</td>
<td>5.5</td>
</tr>
<tr>
<td>Ex 5</td>
<td>.093</td>
<td>6.35</td>
<td>4.75</td>
</tr>
<tr>
<td>Ex 10</td>
<td>.089</td>
<td>7.39</td>
<td>6.25</td>
</tr>
<tr>
<td>Ex 20</td>
<td>.114</td>
<td>7.42</td>
<td>5.0</td>
</tr>
<tr>
<td>Ex 21</td>
<td>.087</td>
<td>6.29</td>
<td>4.75</td>
</tr>
<tr>
<td>End Ex</td>
<td>.083</td>
<td>4.52</td>
<td>1.58</td>
</tr>
<tr>
<td>Rec 1</td>
<td>.009</td>
<td>1.07</td>
<td>0</td>
</tr>
<tr>
<td>Rec 3</td>
<td>.071</td>
<td>4.17</td>
<td>2.75</td>
</tr>
<tr>
<td>Rec 5</td>
<td>.072</td>
<td>4.58</td>
<td>3.0</td>
</tr>
<tr>
<td>Rec 15</td>
<td>.114</td>
<td>5.69</td>
<td>3.25</td>
</tr>
<tr>
<td>Rec 30</td>
<td>.103</td>
<td>6.2</td>
<td>4.75</td>
</tr>
</tbody>
</table>

\[ \bar{x} = .084 \pm .009 \quad \bar{x} = 5.47 \pm .55 \quad \bar{x} = 3.78 \pm .56 \]
TABLE 3
TIME TO EXHAUSTION AT 95% VO$_{2\text{max}}$

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Control(s)</th>
<th>NaHCO$_3$(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>142</td>
<td>164</td>
</tr>
<tr>
<td>2</td>
<td>183</td>
<td>233</td>
</tr>
<tr>
<td>3</td>
<td>180</td>
<td>300</td>
</tr>
<tr>
<td>4</td>
<td>351</td>
<td>454</td>
</tr>
</tbody>
</table>

$\bar{X} = 214 \pm 47$ \hspace{1cm} $\bar{X} = 288 \pm 62$
FIGURE 1: Blood pH Response to Treatments
FIGURE 2: Base Deficit Response to Treatments
FIGURE 3: Standard Bicarbonate in Response to Treatments
APPENDIX D

- Control
- NaHCO₃

FIGURE 4: Blood Lactate Response to Treatments
Figure 5: Base Deficit and Lactate Concentration
Figure 6: Standard Bicarbonate and Lactate Concentration
BIBLIOGRAPHY


