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The effect of induced alkalosis and acidosis on plasma lactate and work output in elite oarsmen

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Summary. In order to test the effect of artificially induced alkalosis and acidosis on the appearance of plasma lactate and work production, six well-trained oarsmen (age = 23.8 ± 2.5 years; mass = 82.0 ± 7.5 kg) were tested on three separate occasions after ingestion of 0.3 $g \cdot kg^{-1}$. NH_4Cl (acidotic), $NaHCO_3$ (alkalotic) or a placebo (control). Blood was taken from a forearm vein immediately prior to exercise for determination of pH and bicarbonate. One hour following the ingestion period, subjects rowed on a stationary ergometer at a pre-determined sub-maximal rate for 4 min, then underwent an immediate transition to a maximal effort for 2 min. Blood samples from an indwelling catheter placed in the cephalic vein were taken at rest and every 30 s during the 6 min exercise period as well as at 1, 3, 6, 9, 12, 15, 18, 21, 25 and 30 min during the passive recovery period. Pre-exercise blood values demonstrated significant differences ($p < 0.01$) in pH and bicarbonate in all three conditions. Work outputs were unchanged in the submaximal test and in the maximal test ($p > 0.05$), although a trend toward decreased production was evident in the acidotic condition. Analysis of exercise blood samples using ANOVA with repeated measures revealed that the linear increase in plasma lactate concentration during control was significantly greater than acidosis ($p < 0.01$). Although plasma lactate values during alkalosis were consistantly elevated above control there was no significant difference in the linear trend ($p > 0.05$). During recovery, there was no significant difference in the rate of lactate disappearance amongst the three conditions. It is concluded that under this protocol artificial manipulation of the acid-base status of the blood does not significantly influence work production despite significantly reduced plasma lactate concentrations during acidosis. The inability of these pH changes to alter exercise performance emphasizes the relative importance of the intracellular and the extracellular buffer systems in well trained athletes.

Key words: Alkalosis — Acidosis — Plasma lactate concentration — Performance — Bicarbonate

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Introduction

In an effort to investigate the influence of the hydrogen ion concentration (H^+) on muscle fatigue and exercise performance capacity, artificial acid-base changes of the extracellular fluids have been induced by ingestion or infusion of sodium bicarbonate ($NaHCO_3$) and ammonium chloride (NH_4Cl). Early studies (Dennig et al. 1931; Dill et al. 1932; Dorow et al. 1940) reported that artificial acidification resulted in earlier exhaustion than that encountered during normal conditions. A state of artificially induced alkalosis resulted in an increased performance capacity and higher blood lactate concentrations. Following these initial findings subsequent studies have demonstrated inconsistant results (Johnson and Black 1953; Margaria et al. 1971; Simmons and Hardt 1973). More recent investigations that use specific anaerobic exercise tasks and maximal tolerable treatment dosages have demonstrated a significant ergogenic effect with $NaHCO_3$ ingestion (Jones et al. 1977; Sutton et al. 1981; Inbar et al.

1983; Wilkes et al. 1983; Costill et al. 1984; McKenzie et al. 1986; Goldfinch et al. 1988) while others have reported that induced alkalosis had no ergogenic benefit (Kindermann et al. 1977b; Katz et al. 1984).

The ergogenic potential of $NaHCO_3$ "loading" and the examination of the limiting factors of maximal exercise in normal healthy subjects appears to be a primary focus of several research papers. Few investigators have included highly trained athletes during their sport specific exercise task. As training-induced adaptive changes are responsible for a specific enhancement in performance (Gollnick and Hermansen 1973; Parkhouse et al. 1985) inducing alkalosis and acidosis and monitoring this influence throughout exercise will allow a comparable determination of how trained subjects respond to shifts in the acid-base status of the blood. Furthermore it may be possible to identify the mechanisms responsible for the enhanced performance capacity demonstrated by well trained athletes.

Thus, the purpose of this investigation was to test the hypotheses that orally induced acidosis and alkalosis in highly trained subjects will influence the appearance of plasma lactate during intense exercise and during passive recovery, and that this acid-base disturbance will also effect exercise performance capacity.

Subjects and methods

Six oarsmen, all members of the National Rowing team (age = 23.8 ± 2.5 years; height = 1.87 ± 0.04 m; mass = 82.0 ± 7.5 kg) volunteered for the study. All subjects were very experienced with ergometric rowing; each gave written consent after they were informed of the procedures and possible risks involved in this study.

All exercise tests were performed on a rowing ergometer (Gjessing Ergorow, Bergen, Norway) which best simulates rowing in the laboratory (Hagerman et al. 1979). The test conditions were standardized; subjects performed three exercise tests in 2 weeks, with at least three days between them. They were asked not to train for 24 h before and not to eat or drink caffeine containing beverages for 6 h before the testing; 3 h before the exercise test they were weighed and given either NH_4Cl (acidotic), $NaHCO_3$ (alkalotic), or lactulose (control), each in a total dose of 0.3 $g \cdot kg^{-1}$. The gelatin capsules were taken with about 500 ml of water over a 2 h period ending one hour before exercise testing began. The order of administration was randomized; the experiment was conducted in a double blind manner.

One hour following the ingestion of the last capsule a pre-exercise blood sample was taken from a forearm vein and immediately analysed in duplicate for pH and bicarbonate determination (Corning 175 automated Blood Gas Analyser).

Then, a 20 gauge teflon catheter was inserted under sterile conditions into the cephalic vein of the left arm. Normal saline was used to keep the catheter patent. The subjects performed a 6 min exercise test (after a standardized warm-up). They rowed at a submaximal rate, calculated to represent 80% of maximal effort, for 4 min. Work rate was visually displayed in front of the subjects. Work output and stroke rate were monitored every 30 s. The subjects were always in full view of the work rate indicator and continually notified of the elapsed time. At four minutes subjects were instructed to row as hard as possible for the final 2 min.

In total, twenty-three 1.5 ml venous blood samples were taken during each experimental trial. Beginning with the resting sample, samples were drawn every 30 s during the 6 min exercise test, and at 1, 3, 6, 9, 12, 15, 18, 21, 25 and 30 min post-exercise. Each was centrifuged, the plasma separated and frozen at −20 degrees C. After all subjects had completed all three treatment conditions, the samples were analyzed enzymatically for plasma lactate concentrations according to the methods of Bergmeyer (1974) and McGrail et al. (1978).

A two-way analysis of variance with repeated measures and trend analysis was used to investigate the effect of artificially induced alkalosis and acidosis on plasma lactate appearance. This was followed by a preplanned nonorthogonal comparison to reveal specific differences between treatment groups. Exercise plasma lactate concentrations and recovery concentrations were analyzed separately; statistical significance was accepted at the $p = 0.01$ level for all tests. Work output during the 4 min submaximal test and the 2 min maximal test were also considered separately. A one-way ANOVA model was used to test significance at the 0.05 level of probability. A similar analysis was done for pre-exercise resting blood pH and bicarbonate values with statistical significance accepted at $p = 0.01$. When a significant F ratio was achieved a post hoc Scheffe test was used to ascertain where differences occurred.

Results

There was a significant acidaemia ($p < 0.01$) after taking NH_4Cl and a significant alkalaemia ($p < 0.01$) after the $NaHCO_3$, while blood pH was in the normal resting range after lactulose (Table 1). The manipulation of pH was well tolerated; several of the men complained of gas and mild nausea during the alkalotic treatment but none felt that these symptoms affected performance. The acidaemia was induced without any significant associated symptoms.

Plasma lactate concentrations immediately before the warm-up were not significantly different, and in the normal range, in the three conditions

Table 1. Pre-exercise values (Mean ± SD)

	pH	Standard HCO_3^- ($mmol \cdot l^{-1}$)
Acidosis	7.22 ± 0.04*	18.6 ± 1.6*
Alkalosis	7.40 ± 0.03*	31.5 ± 2.3*
Control	7.34 ± 0.03	24.9 ± 1.1

* Values significantly different from control ($P < 0.01$)

Table 2. Work output and power production during ergometric rowing (Mean ± SD)

	Work (kJ)		Power (W)	
Treatment	Submaximal 0–4 min	Maximal 4–6 min	Submaximal 0–4 min	Maximal 4–6 min
Acidosis	81.0 ± 4.7	38.3 ± 4.7	337.4 ± 19.6	318.8 ± 39.2
Alkalosis	83.5 ± 4.4	41.9 ± 3.7	348.0 ± 18.3	349.2 ± 30.8
Control	83.5 ± 0.6	42.0 ± 2.0	348.0 ± 19.2	350.0 ± 16.7

(1.5 ± 1.1 mmol·l^{-1} alkalotic, 0.6 ± 0.2 mmol·l^{-1} acidotic, 1.0 ± 0.4 mmol·l^{-1} control) (Gollnick et al. 1986).

There were no significant differences in work output during the four minute submaximal portions of the tests indicating that the subjects were able to repeat the pre-determined submaximal work rate during each of the three treatment conditions (Table 2). Work done during the maximal test was also not significantly altered by the treatments ($p > 0.05$). There was no significant difference in power production ($p > 0.05$) between the submaximal and maximal test in the three treatments (Table 2, Fig. 1).

At 4 mins (end of the submaximal row), although the work output was equal, the plasma lactate values for the alkalotic, control, and acidotic treatments were 14.9, 13.7, and 10.3 mmol·l^{-1} respectively. At the end of the maximal test (6 min) peak lactate concentrations were 20.2 mmol·l^{-1} alkalotic, 17.9 mmol·l^{-1} control, and 12.4 mmol·l^{-1} acidotic. Analysis of this data revealed that the linear increase in blood lactate concentration during the control treatment was

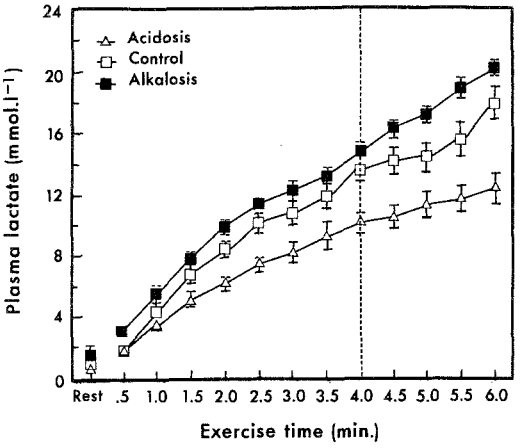

Fig. 2. Mean (± SEM) plasma lactate values during ergometric rowing in acidosis, alkalosis, and control conditions

significantly greater than during the acidotic condition ($p < 0.01$). There was no significant difference in the alkalotic and control values. Plasma lactate concentrations during the 6-min exercise test were significantly higher during alkalosis than during acidosis ($p < 0.01$).

The mean serial plasma lactate concentration of the three experimental conditions during a 30 min passive recovery period are presented in Fig. 3. There were no significant differences in the rate of fall of plasma lactate concentrations between the three treatments. Plasma lactate concentrations 30 min post-exercise were significantly elevated above resting values with mean values of 10.3, 8.6, and 4.7 mmol·l^{-1} during alkalosis, control and acidosis respectively.

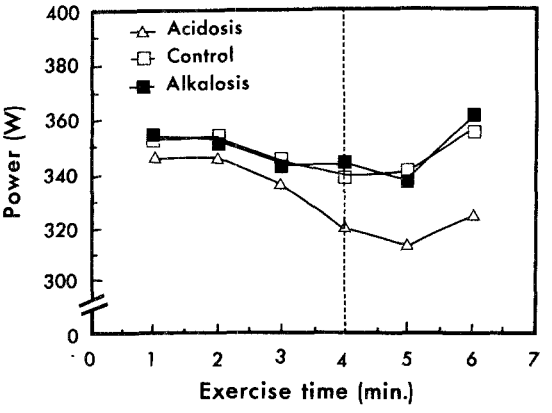

Fig. 1. Mean power production for each minute during ergometric rowing in acidosis, alkalosis and control conditions

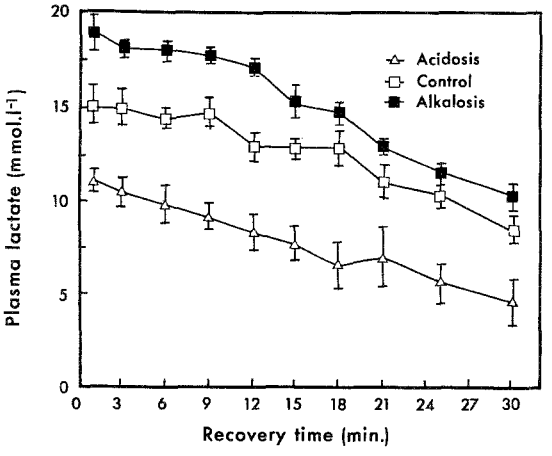

Fig. 3. Mean (± SEM) plasma lactate values during stationary recovery in acidosis, alkalosis, and control conditions

Discussion

The results from this study demonstrate that work output remained unchanged following ingestion of doses of NH_4Cl and $NaHCO_3$ during 4 mins of submaximal, immediately followed by 2 mins of maximal exercise. Although aerobic energy sources contribute significantly to the energy demands of these tests, the intensity of the task is sufficient adequately to examine the ability of the athletes to accumulate lactic acid at a high rate. Studies on elite rowers indicate that rowing provokes extremely high aerobic and anaerobic responses (Koutedakis and Sharp 1985). The trend of serial and peak plasma lactate values during control conditions in this study compare well with those of other ergometric rowing studies (Hagerman et al. 1979; McKenzie and Rhodes 1982). The peak plasma lactate of 20.2 $mmol \cdot l^{-1}$ during the alkalotic condition is comparable to the extreme levels of lactate reported in the literature (Kindermann and Keul 1977a; McCartney et al. 1986).

Even though the first 4 mins of the exercise test were planned to be submaximal and the last 2 mins maximal, work rate and power production did not change between the two parts of the test, suggesting that either the men were working close to near maximal rate for the entire 6 mins or that the acid-base perturbations affected their ability to perform to maximum.

The findings of the present study contrast with those of Wilkes et al. (1983), McKenzie et al. (1986), and Goldfinch et al. (1988) who report ergogenic benefits ($p < 0.05$) in moderately trained subjects with induced metabolic alkalosis, but their subjects were probably less well trained than our oarsmen.

While plasma lactate concentrations during acidosis were significantly decreased there was no significant change following $NaHCO_3$. Data from animal studies have shown that extracellular pH is an important determinant in the rate of lactate and H^+ efflux from muscles. Mainwood and Worsley-Brown (1975) found that efflux of lactate from isolated frog muscle is facilitated by increasing the HCO_3^- of the perfusing fluid. This was also associated with improved muscle function. Similarly Hirche et al. (1975) determined that lactate efflux was nearly three times higher during a state of induced alkalosis compared to acidosis in electrically stimulated isolated blood perfused dog gastrocenemius.

These observations from animal studies are supported by human investigations. Jones et al. (1977) and Sutton et al. (1981) reported that after $NaHCO_3$ treatment there were higher lactate levels in the blood at two submaximal work rates (66% and 95% $\dot{V}_{O_{2max}}$) than there was after NH_4Cl administration. Using needle biopsy, Sutton and colleagues (1981) directly compared several glycolytic intermediate concentrations in the three experimental conditions. They showed a reduction in glycogen utilization associated with lower blood lactate in acidosis, resulting, apparently from an inhibition of muscle glycolysis at the level of phosphofructokinase, combined with a reduction in lactate efflux. A study by Costill et al. (1984) also included muscle sampling for muscle pH determination. Following four 1 min cycling bouts at 100% $\dot{V}_{O_{2max}}$, the fifth bout to exhaustion in an alkalotic condition was 42% longer ($p < 0.01$) than control trials. A higher muscle pH immediately before the fifth cycling bout and a greater drop in plasma HCO_3^- during the repeated bouts supports the concept of a greater efflux of H^+ from the muscle, due to the increased buffer potential of the extracellular fluid. Despite the significant differences observed in blood and muscle pH and blood HCO_3^-, there was no difference between mean blood and muscle lactate values in the $NaHCO_3$ and placebo trials. This feature of blood lactate is consistant with findings in the present study, however, other studies have demonstrated increased efflux rates of both H^+ and lactate with increases in extracellular HCO_3 (Jones et al. 1977; Wilkes et al. 1983).

There is increasing evidence that intracellular pH is a limiting factor during maximal exercise (Hultman and Sahlin 1980) and the pH status of extracellular fluid plays a key role in determining intracellular pH balance (Mainwood and Renaud 1985). However, based on the results of this present study the role that induced pre-exercise acid-base changes play in performance capacity is questionnable. There are a number of factors in this experiment that may account for the unchanged performance response under the alkalotic and acidotic treatment conditions.

The major buffering components of human skeletal muscle are the bicarbonate buffer system, creatine phosphate utilization, inorganic phosphates, protein-bound histidine residues, and the histidine containing dipeptides carnosine and anserine (Hultman and Sahlin 1980). Parkhouse et al. (1985) found increased buffering in highly trained 800 meter runners and rowers, compared to marathon runners and untrained subjects. Therefore, it was concluded that the capacity of the skeletal muscle to buffer the increased hy-

drogen ions associated with maximal exercise may be enhanced, enabling these trained athletes to sustain anaerobic work for an extended time before the onset of fatigue. Results from animal lactate tracer studies have documented increased metabolic clearance rates with endurance training (Donovan and Brooks 1983). This adaptation permits lower lactate levels in trained individuals during exercise at a given work rate since lactate production is balanced by an increased rate of removal. As the primary metabolic fate of lactate and H^+ is that of oxidation, this improved removal of lactic acid will serve to suppress the fall of muscular pH during exercise. The potential for an increased intracellular buffer capacity and rate of lactate clearance, could have afforded the highly trained subjects in the present study an enhanced ability to deal with the inhibitory effects of maximal exercise. Therefore, alterations in the rate of lactate and H^+ efflux provided by pH changes in the blood are perhaps not a dominant limiting factor, as maybe the case with untrained subjects. This may explain why the significantly reduced plasma lactate values during acidosis in this study were associated with a relatively unchanged work rate while the literature reports decreased performance with decreased blood lactate concentrations in untrained subjects (Jones et al. 1977; Sutton et al. 1981). These adaptive mechanisms may also offer a plausible explanation of why $NaHCO_3$ ingestion did not produce an ergogenic effect despite slightly increased plasma lactate values.

During recovery the present data demonstrates that plasma lactate values gradually decline toward resting values. Values observed at one minute post-exercise were slightly lower (1.2–2.8 $mmol \cdot l^{-1}$) than samples at the cessation of exercise. It is reported that following heavy exercise blood lactate continues to rise reaching a peak approximately 5 min after the termination of exercise (Gollnick et al. 1986). A possible explanation is that these highly trained athletes are able to oxidize lactic acid within the skeletal muscle (Gollnick and Hermansen 1973). Although it is generally accepted that the use of active recovery following exercise results in a much higher rate of lactate reduction than complete rest (Bonen and Belcastro 1976), to standardize the protocol as much as possible, passive recovery was used in the present study.

Few human investigations have centered on the effects of pH changes in blood lactate appearance during recovery. In the present study it was noted that there were no significant differences in the rate of decrease of plasma lactate between the acidotic, alkalotic and control conditions. As blood lactate levels represent balance between lactate entry and lactate removal it could be hypothesized that the enhanced rate of lactate removal through oxidation, by these highly trained subjects may be a more important determinant in blood lactate disappearance than extracellular acid-base status.

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