Effects of Induced Metabolic Alkalosis on Prolonged Intermittent-Sprint Performance

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ABSTRACT

BISHOP, D., and B. CLAUDIUS. Effects of Induced Metabolic Alkalosis on Prolonged Intermittent-Sprint Performance. Med. Sci. Sports Exerc., Vol. 37, No. 5, pp. 759–767, 2005. Purpose: Previous studies have shown that induced metabolic alkalosis, via sodium bicarbonate (NaHCO₃) ingestion, can improve short-term, repeated-sprint ability. The purpose of this study was to assess the effects of NaHCO₃ ingestion on a prolonged, intermittent-sprint test (IST). Methods: Seven female team-sport athletes (mean \pm SD: age = $19 \pm 1 \text{ yr}, \dot{VO}_{2\text{peak}} = 45.3 \pm 3.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) volunteered for the study, which had received ethics clearance. The athletes ingested two doses of either $0.2 \text{ g} \cdot \text{kg}^{-1}$ of NaHCO₃ or $0.138 \text{ g} \cdot \text{kg}^{-1}$ of NaCl (placebo), in a double-blind, random, counterbalanced order, 90 and 20 min before performing the IST on a cycle ergometer (two 36-min "halves" of repeated ~2-min blocks: all-out 4-s sprint, 100 s of active recovery at 35% VO_{2peak}, and 20 s of rest). Capillary blood samples were drawn from the ear lobe before ingestion, and before, during, and after each half of the IST. VO2 was also recorded at regular intervals throughout the IST. Results: Resting plasma bicarbonate concentration ([HCO₃⁻]) averaged 22.6 \pm 0.9 mmol·L⁻¹, and at 90 min postingestion was 21.4 \pm 1.5 and 28.9 \pm 2.8 mmol·L⁻¹ for the placebo and NaHCO₃ conditions, respectively (P < 0.05). Plasma [HCO₃⁻¹] during the NaHCO₃ condition remained significantly higher throughout the IST compared with both placebo and preingestion. There was a trend toward improved total work in the second (P = 0.08), but not first, half of the IST after the ingestion of NaHCO₃. Furthermore, subjects completed significantly more work in 7 of 18 second-half, 4-s sprints after NaHCO₃ ingestion. Conclusions: The results of this study suggest that NaHCO₃ ingestion can improve intermittent-sprint performance and may be a useful supplement for team-sport athletes. Key Words: BLOOD LACTATE, BUFFER CAPACITY, CYCLING, INTERMITTENT EXERCISE, PEAK POWER, pH

ost team sports (e.g., field hockey and the various football codes) require participants to repeatedly produce high-intensity efforts (interspersed with short breaks) over the duration of a game. The ability to recover and to reproduce a maximal effort in subsequent sprints is, therefore, likely to be an important determinant of the outcome of team-sport games. However, further research is required to determine factors that limit intermittent-sprint ability, and to determine training and ergogenic strategies to improve team-sport performance.

Maximal sprint exercise requires a high skeletal muscle adenosine triphosphate (ATP) turnover rate. As intramuscular ATP storage is able to sustain muscular activity for only 1-2 s, ATP must continually be resynthesized for activity to continue. It is now generally accepted that the majority of the energy required to resynthesize ATP for short-duration, maximal exercise is provided by phospho-

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0195-9131/05/3705-0759 MEDICINE & SCIENCE IN SPORTS & EXERCISE_® Copyright © 2005 by the American College of Sports Medicine DOI: 10.1249/01.MSS.0000161803.44656.3C creatine (PCr) degradation and anaerobic glycolysis (9). Anaerobic glycolysis is associated with the intracellular accumulation of hydrogen ions (H^+), which have been implicated as a cause of muscular fatigue (24). Thus, the ability to reproduce maximal sprint efforts is likely to depend, in part, on the ability to resist H^+ accumulation in the muscle.

The accumulation of H⁺ depends on both the production and removal of H⁺. Various intracellular and extracellular buffer mechanisms operate to buffer the H⁺ released during high-intensity exercise, and may therefore be important in maintaining repeated-sprint performance. Indeed, we have recently reported a significant relationship between repeated-sprint ability (RSA) and both change in blood pH (3) and in vivo muscle buffer capacity $(\beta m_{in vivo})$ (2). The intracellular accumulation of H⁺ will also depend on the extracellular H⁺ concentration. H⁺ efflux out of the muscle cell has been reported to be inhibited by extracellular acidosis (12) and enhanced by a greater extracellular buffer concentration (16). It may, therefore, be hypothesized that increases in the extracellular buffer concentration, via the ingestion of an alkaline solution such as sodium bicarbonate (NaHCO₃), may improve H⁺ efflux out of the muscle cell and improve repeated-sprint performance.

The effects of NaHCO₃ ingestion on short-term, repeatedsprint protocols and intermittent protocols proposed to simulate the playing requirements of team-sport games have been investigated. It was recently reported that NaHCO₃ ingestion is ergogenic for work completed and power output during sprints 3, 4, and 5 of a repeated-sprint protocol (5 ×

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6-s all-out sprints every 30 s) (1). Similarly, Price et al. (20) reported greater power output during a long-term repeatedsprint test. The 30-min intermittent cycling protocol consisted of 10 \times 3-min blocks of 90 s at 40% \dot{VO}_{2peak} , 60 s at 60% VO_{2peak}, a 14-s maximal sprint, and 16 s of rest. However, the duration of a team-sport game is much greater than the duration of the repeated-sprint test employed in these two studies. In addition, the sprint duration in the intermittent protocol used by Price et al. (20) was 14 s, which is considerably longer than the short-duration sprints (<6 s) characteristic of most team-sport games (18,23). To date, no studies have investigated the effects of NaHCO₃ ingestion on short-duration (4 s) repeated-sprint activity over a prolonged period of time (70 min) to replicate the average sprint profile of a typical team-sport game. This is surprising because in most countries the most popular sports, and those with the highest participation levels, are team games (which require athletes to sprint intermittently throughout a match).

The purpose of the present study, therefore, was to investigate the effects of NaHCO₃ ingestion on an intermittent-sprint test (IST) designed to replicate the average sprint profile of a typical team-sport game. The IST was based on motion analysis of international field hockey and consisted of short-duration sprints interspersed with short periods of active and passive recovery (23). It was hypothesized that NaHCO₃ ingestion would enhance the performance of the prolonged IST.

METHODS

Subjects. Seven female team-sport athletes were recruited to participate in this study (mean \pm SD: age 19 \pm 1 yr, mass 58.0 \pm 1.6 kg, \dot{VO}_{2peak} 45.3 \pm 3.0 mL·kg⁻¹·min⁻¹). None of the subjects were involved in any form of nutritional supplementation that may have compromised the administration of the NaHCO₃. Subjects were informed of the study requirements, benefits, and risks before giving written informed consent. Approval for the study's procedures was granted by the research ethics committee of the University of Western Australia.

Experimental overview. In addition to a familiarization session for all tests, the main experiment required the subjects to be tested on three separate occasions. On day 1, subjects performed a graded exercise test (GXT) to determine VO_{2peak}. At least 48 h later, in a random, counterbalanced order, subjects then performed the IST, after the ingestion of either sodium bicarbonate (NaHCO₃) or a placebo substance (NaCl). A week separated the two IST sessions, and both tests were conducted at the same time of day (between 9:00 a.m. and 12:00 p.m.) to control for diurnal effects. One week was considered a sufficient washout period to remove any ergogenic effects of NaHCO₃. Capillary blood was sampled before and during each IST. Expired air was also collected during the GXT and during parts of the IST to determine VO₂. A heart rate monitor (Polar Vantage NV, Finland) was used to monitor and store heart rate during both the GXT and the IST. Subjects were

asked to maintain their normal diet and training throughout the study. Subjects were required to consume no food or beverages (other than water) 2 h before testing, and were asked not to consume alcohol or perform vigorous exercise in the 24 h before testing (this was verified via a 24-h dietary and activity recall).

Ergometers. Air-braked cycle ergometers were used to conduct all cycle tests. These ergometers were interfaced with an IBM-compatible computer system to allow for the collection of data for the calculation of work and power generated during each flywheel revolution (Cyclemax, The University of Western Australia, Perth, Australia). These ergometers require subjects to pedal against air resistance caused by rectangular vanes attached perpendicular to the axis of rotation of the flywheel. The power output of the air-braked cycle ergometer is proportional to the cube of the flywheel velocity. An optical sensor monitored the velocity of the flywheel at a sampling rate of 80 pulses per pedal revolution. Before testing, each ergometer was dynamically calibrated on a mechanical rig (Western Australian Institute of Sport, Perth, Australia) across a range of power outputs (100-2000 W).

Graded exercise test. The GXT was performed on an air-braked track-cycle ergometer (Evolution Pty. Ltd., Adelaide, Australia) and consisted of graded exercise steps (3-min stages), using an intermittent protocol (1-min break between stages). The test commenced at 40 W, and, thereafter, intensity was increased by 30 W every 3 min until volitional exhaustion. Subjects were required to maintain the set power output, which was displayed on a computer screen in front of them. The test was stopped when the subject could no longer maintain the required power output. Strong verbal encouragement was provided to each subject as they came to the end of the test.

Intermittent-sprint test (IST). Based on a motion analysis study of international field hockey (23), the IST was designed to mimic the average sprint profile of a typical team-sport game, and consisted of two 36-min "halves" of intermittent-sprint exercise (Fig. 1). The protocol was divided into \sim 2-min blocks of sprinting, active recovery, and passive rest. Each block started with an all-out 4-s sprint, immediately followed by 100 s of active recovery. The active recovery required the subject to maintain a constant power output of 35% of their predetermined power output at \dot{VO}_{2peak} . The 2-min block was then completed by 20 s of passive rest. Our motion analysis study (23) also identified that there were, on average, approximately "two repeatedsprint bouts" (defined as a minimum of three sprints, with mean recovery duration between sprints < 21 s) per playing position during an international field hockey game, with a mean sprint number of 4 ± 1 sprints per bout. In addition, on average, 95% of the recovery during the repeated-sprint bouts was of an active nature. Therefore, in an effort to more closely mimic the average sprint profile of a typical teamsport game, on two occasions during each 36-min half (after sprints 8 and 16), a repeated-sprint bout (RSB), comprising 5×2 -s sprints departing every 20 s with active recovery between subsequent 2-s sprints, replaced the 2 min of active

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FIGURE 1—Schematic representation of the first half of the repeated-sprint test (top section). Each \sim 2-min block comprised a 4-s maximal sprint, 100 s at 35% \dot{VO}_{2peak} , and 20 s of passive rest (bottom section). There were also two repeated-sprint bouts, which comprised 5 × 2-s sprints separated by \sim 20 s at 35% \dot{VO}_{2peak} . WUP, warm-up; B, blood sample; EG, expired gas sample (60 s).

and passive recovery. The subjects were given a 10-min passive recovery between "halves."

Twelve minutes before the beginning of the IST, the subjects completed a 10-min warm-up on the front-access ergometer (Model Ex-10, Repco, Australia). The warm-up required the subjects to cycle for 5 min at 50% of their predetermined power output at $\dot{V}O_{2peak}$, followed by two blocks of 30 s at 70% of the power output at VO_{2peak}, followed by 30 s of rest. The subjects then performed a practice 2-min block of the IST protocol, followed by 1 min at 35% of the power output at VO_{2peak}. The subjects then rested, and the test started 2 min after the completion of the warm-up. Although the IST was performed on the front-access cycle ergometer, it has been reported that repeated-sprint cycling performance on the front-access cycle ergometer is strongly correlated with repeated-sprint running performance (4). To further enhance the relevance of this study, all sprints were performed in the standing position on the front-access cycle ergometer. In our laboratory, the coefficient of variation for individual sprints is 1.8 and 2.5% for peak power output and mean power output, respectively. The subjects were provided with standardized amounts of water (3 \times 150 mL) and carbohydrate solution (3 \times 150 mL) at alternate intervals (approximately every 15 min) during the IST to ensure they were adequately hydrated and to better simulate match demands.

Calculation of test scores (IST). The work done (J) and peak power achieved (W) were recorded for each 4-s sprint of the IST. The mean work and peak power achieved during the repeated-sprint bouts of each half of the IST were also calculated by averaging the work done and peak power achieved in the 4-s sprints preceding (sprints 8 and 16) and proceeding (sprints 9 and 17) the two repeated-sprint bouts.

Supplement ingestion. The NaHCO₃ was administered in 2×0.2 -g·kg⁻¹ doses taken 90 and 20 min before the start of the IST. This ingestion protocol was chosen in an attempt to maintain elevated [HCO₃⁻] throughout the IST. Furthermore, pilot work indicated that this protocol did not

result in any adverse affects, whereas larger doses (e.g., $2 \times$ 0.3 $g \cdot kg^{-1}$) greatly increased the risk of gastrointestinal disturbances. Based on previous results (7), $0.2 \text{ g} \cdot \text{kg}^{-1}$ body mass is sufficient to induce alkalosis and produce a significant increase in blood buffering capacity. Similarly, the placebo substance (NaCl) was administered in 2×0.138 $g \cdot kg^{-1}$ dosages taken 90 and 20 min before the start of the IST. The dosage of 0.138 $g \cdot kg^{-1}$ body weight of NaCl was used as a placebo substance because its sodium content contains an equimolar amount of salt to the NaHCO₃ dosage. The subjects were given 30 min to ingest the NaHCO₃; the first dosage was ingested 110 to 90 min before the test, and the second dosage was ingested 50 to 20 min before the test. The order of supplementation (NaHCO₃ and NaCl) was double blind and randomized. The supplements were administered orally via gelatin capsules (approximately 15-30 capsules) and were consumed with as much water as required. All subjects coped well with this supplement protocol, and there were no reported adverse effects (e.g., gastrointestinal discomfort, nausea).

Gas analysis (GXT). During the GXT, expired air was continuously analyzed for O_2 and CO_2 concentrations using Ametek gas analyzers (Applied Electrochemistry, SOV S-3A11 and COV CD-3A, Pittsburgh, PA). Ventilation was recorded every 15 s using a turbine ventilometer (Morgan, 225A, Kent, UK). The gas analyzers were calibrated immediately before and verified after each test using three certified gravimetric beta-grade gas mixtures (BOC Gases, Chatswood, Australia). The ventilometer was calibrated preexercise using a 1-L syringe in accordance with the manufacturer's instructions. The ventilometer and gas analyzers were connected to an IBM PC that measured and displayed variables every 15 s. The sum of the four highest consecutive 15-s values was recorded as the subject's \dot{VO}_{2peak} .

Gas analysis (IST). During the recovery period after sprints 2 and 14 of each half of the IST, expired gas was collected with Douglas bags. Expired gas was analyzed for volume and concentration of O_2 and CO_2 (Ametek gas analyzers SOV S-3A and COV CD3A, respectively, Pittsburgh, PA). The gas analyzers were calibrated immediately before and verified after each test using three certified gravimetric beta-grade gas mixtures (BOC Gases). From the Douglas bag, the expired gas was passed through a Tissottank, and the volume of expired gas was calculated.

Capillary blood sampling and analysis. Glass capillary tubes were used to collect 35 μ L of blood during the GXT (D957G-70-35, Clinitubes, Radiometer Copenhagen) and 125 μ L of blood during the IST (D957G-70-125, Clinitubes). Capillary blood samples were taken at rest and immediately after each 3-min stage of the GXT. Capillary blood samples were also taken before the ingestion of the supplement, before and after the warm-up, and before and after each half of the IST. In addition, blood was sampled during the active recovery after the 9th and 17th 4-s sprint (after the 5 \times 2-s repeated-sprint bout) of the IST (Fig. 1). Capillary blood was analyzed for pH, lactate concentration, and bicarbonate concentration. The blood-gas analyzer (ABL 625, Radiometer Copenhagen) was regularly calibrated using precision standards, and routinely assessed by external quality controls.

Statistical analysis. All values are reported as mean \pm SEM. Two-way ANOVA (2 treatments × 18 sprints) with repeated measures were used to determine whether there were any performance differences between each half of the IST. Similarly, two-way ANOVA (2 treatments × 10 measurements) with repeated measures were used to determine whether the blood data collected over the duration of each IST test differed across conditions. Where appropriate, *post hoc* comparisons were employed (Student–Newman–Keuls test). Paired sample *t*-tests were used to determine whether the volume of oxygen consumption (\dot{VO}_2) and respiratory exchange ratio (RER) were different between each half of the IST for the two conditions. Statistical significance was accepted at *P* < 0.05 unless otherwise stated.

RESULTS

Blood. Plasma $[HCO_3^-]$, pH, and $[La^-]$ for both conditions across all time points is summarized in Figure 2. There was no significant difference in the preingestion concentration of any blood variable between the placebo and NaHCO₃ conditions. Plasma $[HCO_3^-]$ and pH were significantly higher at all postingestion measures in the NaHCO₃ condition compared with the placebo condition. Furthermore, all postingestion measures for plasma $[HCO_3^-]$ and pH in the NaHCO₃ condition were significantly higher than the "baseline" preingestion level (P < 0.05). There was no significant difference in $[La^-]$ during either half of the IST between the placebo and NaHCO₃ conditions. However, posttest plasma $[La^-]$ was significantly higher in the NaHCO₃ condition compared with the placebo conditions.

Performance data. There was no significant difference in the total work completed between the placebo and NaHCO₃ conditions during the first half (34,554.2 \pm 5,827.4 vs 34,836.4 \pm 6,133.3 J, respectively; *P* = 0.751) or

second half (34,269.8 \pm 5,138.6 vs 35,563.2 \pm 5,941.3 J, respectively; P = 0.08) of the IST. There was also no significant difference in work performed between "halves" for either condition, and no significant order effect. The average amount of work performed by participants during individual sprints in each half of the IST is summarized in Figure 3. Although there were no significant differences during the first half, the work completed during 7 of the 18 second-half sprints was found to be significantly greater in the NaHCO₃ condition compared with the placebo condition (P < 0.003).

There was also no significant difference in the mean peak power achieved between the placebo and NaHCO₃ conditions during the first half (745.2 \pm 116.9 vs 757.3 \pm 128.8 W, respectively; P > 0.05) and second half (727.2 \pm 107.5 vs 763.7 \pm 136.1 W, respectively; P > 0.05) of the IST. Again, there was no significant order effect. The average peak power achieved by participants during individual sprints of each half of the IST is summarized in Figure 4. Although there were no significant differences during the first half, the peak power achieved during 8 of the 18 second-half sprints was significantly greater in the NaHCO₃ condition compared with the placebo condition. There was no significant difference in the mean work completed or peak power achieved in the sprints preceding and proceeding repeated-sprint bouts 1 (sprints 8 and 9) and 2 (sprint 16 and 17) of each half of the IST in both conditions (Table 1).

Gas analysis data. The mean rate of oxygen consumption ($\dot{V}O_2$) and respiratory exchange ratio (RER) during each half of the IST test is summarized in Figure 5. No differences were observed between the placebo and NaHCO₃ conditions for oxygen consumption ($\dot{V}O_2$; P > 0.05). In addition, RER values were not significantly different between the trials.

Heart rate. There were no significant differences in the average heart rate during each half of the IST (P > 0.05). The average heart rate during the first half of the IST was 137 ± 13 and 142 ± 18 beats·min⁻¹ in the NaHCO₃ and placebo conditions, respectively. The average heart rate of the second half of the IST was 140 ± 12 and 147 ± 16 beats·min⁻¹ in the NaHCO₃ and placebo conditions, respectively.

DISCUSSION

The purpose of this study was to investigate the effects of NaHCO₃ ingestion on the performance of an IST designed to replicate the average sprint profile of a typical team-sport game. Blood bicarbonate concentration, lactate concentration, and pH levels were measured to elucidate possible mechanisms underlying any performance differences between the two trials. The main finding of this study was that preexercise ingestion of NaHCO₃ was effective in enhancing the extracellular [HCO₃⁻] and pH level both before and during the IST. The second major finding was that the ingestion of NaHCO₃ was ergogenic at selected time points during the second half of the IST. Although the total work completed and average peak power were not significantly different between conditions in either half of the IST, there



FIGURE 2—Plasma bicarbonate concentration ([HCO₃⁻]) (A), pH (B), and lactate concentration ([La⁻]) (C) for the placebo and NaHCO₃ conditions. Values are mean \pm SEM (N = 7). * Indicates a significant difference from placebo condition (P < 0.005). I, ingestion; wu, warm-up; RSB, repeated-sprint bout; 1/2, first "half" of IST.

was a trend toward improved performance in the second half of the IST after the ingestion of NaHCO₃. Furthermore, performance of a number of second-half sprints was significantly greater after NaHCO₃ ingestion.

Efficacy of NaHCO₃ ingestion. The ingestion protocol for the NaHCO₃ condition consisted of 2×0.2 -g·kg⁻¹ doses of NaHCO₃, ingested 90 and 20 min before the start of the IST. Based on pilot work, this ingestion protocol was chosen in an attempt to counter the decrease in pH and [HCO₃⁻] that is typically reported during prolonged exercise trials (20,25). In particular, we wanted to ensure that subjects began the second half of the IST with an elevated [HCO₃⁻]. To the authors' knowledge, this is the first time that NaHCO₃ has been administered in two doses preexercise. The results are therefore difficult to compare with previous studies that have employed singledose ingestion protocols.

Ninety minutes after ingestion of the first 0.2-g·kg⁻¹ dose of NaHCO₃, plasma [HCO₃⁻] was increased by 5.5 mmol·L⁻¹ (22.6–28.9 mmol·L⁻¹). This increase is of similar magnitude to previous NaHCO₃ studies. Costill et al. (7) reported an increase in plasma [HCO₃⁻] of 3.5 mmol·L⁻¹ (27.5–31.0 mmol·L⁻¹) 60 min after the ingestion of a single 0.2-g·kg⁻¹ dose of NaHCO₃, whereas Horswill et al. (14) reported a similar increase in plasma [HCO₃⁻] of 4.8 mmol·L⁻¹ (26.1–30.9 mmol·L⁻¹) 60 min after the ingestion of 0.2 g·kg⁻¹ of NaHCO₃. In a meta-analysis, Matson and Tran (17) reported that the average increase in plasma [HCO₃⁻] after the ingestion of 0.3 g·kg⁻¹ of NaHCO₃ was 5.3 mmol·L⁻¹. It therefore appears that two doses of 0.2

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FIGURE 3—Work completed (J) during individual sprints in the first half (A) and second half (B) of the IST for the placebo and NaHCO₃ conditions. Values are mean \pm SEM (N = 7). * Indicates a significant difference from placebo condition (P < 0.003). \uparrow , repeated-sprint bout.

 $g \cdot kg^{-1}$ taken 90 and 20 min before exercise is as effective as a single NaHCO₃ dose (0.2–0.3 $g \cdot kg^{-1}$) in increasing plasma [HCO₃⁻]. Furthermore, and consistent with our hypothesis, with this ingestion protocol plasma [HCO₃⁻] and pH remained significantly elevated throughout the IST in the NaHCO₃ condition compared with both the placebo condition and preingestion values.

In the present study, the plasma $[HCO_3^-]$ peaked at 30.0 $\text{mmol}\cdot\text{L}^{-1}$ immediately before the second half of the IST. The timing of the peak in [HCO₃⁻] in the NaHCO₃ condition was approximately 90 min after the second ingestion of NaHCO₃. It is possible that the timing of the peak in [HCO₃⁻] is associated with the second NaHCO₃ ingestion, taken 20 min before the IST. However, the timing of the peak in $[HCO_3^{-}]$ may also be an effect of the half-time rest period, because plasma [HCO₃⁻] also appeared to increase in the placebo condition. The increase in $[HCO_3^{-}]$ does not appear to be due to the release of stored $CO_2(6)$ as there was not a concomitant decrease in pH (i.e., an increase in $[H^+]$). Although further research is required, the ingestion of multiple doses of NaHCO₃ may have implications for sustaining an elevated plasma [HCO3⁻] during other types of prolonged exercise.

As a consequence of the NaHCO₃ ingestion, blood pH level was also significantly higher at all postingestion measurements, including post-IST. The increase in blood pH of 0.06 (7.43-7.49) after NaHCO₃ ingestion in the present study was similar to that reported in previous studies. Using a single

0.2-g·kg⁻¹ dose, Costill et al. (7) and Horswill et al. (14) both reported an increase of 0.07 pH units 60 min postingestion. An increase of 0.5–0.6 pH units has also been reported 60 min after the ingestion of a single 0.3-g·kg⁻¹ dose of NaHCO₃ (8,20). Consistent with previous research, there was no change in resting blood lactate concentration (7,8,19,25).

Effects of metabolic alkalosis on exercise lactate concentration. There was also no significant difference in plasma [La⁻] during the IST (Fig. 2). However, posttest [La⁻] was significantly higher (26%) in the NaHCO₃ condition $(3.4 \text{ mmol} \cdot \text{L}^{-1})$ compared with the placebo condition $(2.7 \text{ mmol}\cdot\text{L}^{-1})$. Numerous other studies (1,5,8,10) have also reported greater blood [La⁻] after high-intensity exercise after NaHCO3 ingestion. There are a number of mechanisms that may explain the greater postexercise [La⁻]. The higher posttest blood [La⁻] may be related to enhanced efflux of H⁺ and lactate from the contracting muscle due to the activity of the lactate/H⁺ transporter, which becomes more active as the intracellular/extracellular H⁺ gradient increases (22). However, the higher postexercise blood [La⁻] after NaHCO₃ ingestion also could be due to a reduction in blood lactate clearance by inactive tissues, rather than an increased lactate efflux from the contracting muscle. Granier et al. (11) have previously reported that arteriovenous difference across the inactive forearm was reduced during repeated-sprint cycling exercise (6-s sprints with 5 min of recovery) after NaHCO₃ infusion, suggesting less lactate removal. The higher postexercise [La⁻] in the



FIGURE 4—Peak power achieved (W) during individual sprints in the first half (A) and second half (B) of the IST for the placebo and NaHCO₃ conditions. Values are mean \pm SEM (N = 7). * Indicates a significant difference from placebo condition (P < 0.003). \uparrow , repeated-sprint bout.

 $NaHCO_3$ condition may also be associated with a higher anaerobic energy contribution and greater glycogenolytic flux due to the upregulation of both phosphorylase and phosphofructokinase activity (13). This may have contributed to the improved performance in many of the secondhalf sprints, especially the final sprint, after NaHCO₃ ingestion. There is, however, controversy as to whether the

TABLE 1. Mean work completed (A) and peak power achieved (B) in the sprints preceding and proceeding repeated-sprint bouts 1 (sprints 8 and 9) and 2 (sprint 16 and 17) of each half of the repeated-sprint test in both conditions; values are mean \pm SEM.

	Mean (Sprint 8 and 9)	Mean (Sprint 16 and 17)
A		
Work (J)		
Placebo—first half	1906.1 ± 331.3	1923.8 ± 281.4
Bicarbonate—first half	1995.5 ± 291.7	1861.3 ± 425.8
Placebo—second half	1885.5 ± 222.7	1930.4 ± 309.3
Bicarbonate—second half	1879.5 ± 300.8	2003.3 ± 429.1
В		
Peak Power (W)		
Placebo-first half	713.0 ± 121.9	727.8 ± 101.3
Bicarbonate—first half	769.2 ± 118.3	743.8 ± 158.0
Placebo—second half	749.0 ± 95.2	712.0 ± 113.7
Bicarbonate—second half	725.0 ± 118.8	774.0 ± 160.7

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increase in postexercise plasma [La⁻] after NaHCO₃ ingestion is due to an increase in the rate of glycogenolysis, an increase in the rate of La⁻ release from the muscle, or decreased clearance from inactive tissues. As neither muscle metabolites nor lactate kinetics were measured in the present study, the mechanism(s) responsible for the greater posttest plasma [La⁻] after NaHCO₃ ingestion in the present study require further investigation.

The effect of NaHCO₃ **ingestion on performance.** In the present study, the preexercise ingestion on NaHCO₃ was ergogenic during the second half of the IST. Subjects performed significantly more work and achieved a higher peak power in almost half of the second-half sprints. Furthermore, the difference between conditions for total work completed during the second half of the IST approached significance (P = 0.08). The results of this study are consistent with the findings of previous studies that have reported significant performance improvements for repeated-sprint exercise after the ingestion of NaHCO₃. Bishop et al. (1) found NaHCO₃ ingestion to be ergogenic for work completed and power output during sprints 3, 4, and 5 of a repeated-sprint protocol (5 × 6-s sprints every 30 s), and Price et al. (20) reported significantly greater power output



FIGURE 5—Mean oxygen consumption ($\dot{V}O_2$) during each half of the IST for the placebo and NaHCO₃ conditions. Values are mean ± SEM (N = 7).

during a long-term repeated-sprint test. The 30-min intermittent cycling protocol consisted of 10×3 -min blocks of 90 s at 40% $\dot{V}O_{2peak}$, 60 s at 60% $\dot{V}O_{2peak}$, a 14-s maximal sprint, and 16 s of rest. These exercise protocols were proposed to simulate the playing requirements of a typical team-sport game. However, the duration of a typical teamsport game is much greater than the duration of the repeatedsprint test employed by Bishop et al. (1). In addition, the sprint duration in the intermittent protocol used by Price et al. (20) was 14 s, which is considerably longer than the short-duration sprints (<6 s) characteristic of most teamsport games (23). Therefore, this study is the first to show that NaHCO₃ ingestion can improve performance during a prolonged IST typical of a team-sport game (especially in the second half).

The blood pH and [HCO₃⁻] during the IST in the present study was relatively high, and the blood [La⁻] considerably smaller, in both conditions compared with previous studies that have reported improved performance after the ingestion of NaHCO₃. This may partially explain why, in contrast to our previous research (1), we did not observe improved performance after NaHCO₃ ingestion during the early stages of the present study. Studies that have shown an ergogenic benefit of NaHCO₃ to exercise performance have typically reported a decline in blood $[HCO_3^-]$ in the range of 10–15 $\text{mmol}\cdot\text{L}^{-1}$, a decline in pH to below 7.2, and posttest [La⁻] greater than 10 mmol· L^{-1} (5,7). In the present study, however, the posttest pH in the placebo and bicarbonate conditions were 7.38 and 7.50, respectively, and the posthalf blood $[HCO_3^{-}]$ in the placebo and NaHCO₃ conditions was 19.3 and 29.0 mmol· L^{-1} , respectively. The decline in $[HCO_3^{-}]$ between the postingestion measure and posttest measure in the placebo and NaHCO₃ conditions was 3.1 and $0.2 \text{ mmol}\cdot\text{L}^{-1}$, respectively. The blood [La⁻] in both halves of the IST was also relatively low, and peaked at 3.2 and 4.0 $\text{mmol}\cdot\text{L}^{-1}$ for the placebo and bicarbonate conditions, respectively. These relatively small changes can possibly be attributed to the use of female subjects, the relatively long recovery between sprints, and/or the relatively short duration of the sprints. Although Matson et al. (17) have suggested that NaHCO₃ ingestion is ergogenic to exercise protocols that cause a large accumulation of H⁺, the results of the present study suggest that $NaHCO_3$ ingestion also can improve the performance of repeated-sprint exercise that produces a relatively small change in acid–base balance.

As the cell membranes are relatively impermeable to HCO_3^- (15,21), the intake of NaHCO₃ does not appear to alter the intracellular buffering capacity (1). Instead, the ergogenic benefit of NaHCO₃ ingestion has been attributed to the increased buffering of H⁺ in the blood and better pH maintenance during exercise. It is believed that the increased buffering potential of the blood enhances the efflux of H⁺ from the contracting muscle into the blood (7,10), reducing the intracellular accumulation of H⁺, which has been implicated as a cause of muscular fatigue (24).

In the present study, however, the relatively high posttest plasma pH levels in both conditions (placebo: 7.38; bicarbonate: 7.50), the small decline in $[HCO_3^{-}]$, and the absence of a significant decline in performance in either condition indicate that performance decrements due to H⁺ accumulation were unlikely. An alternate hypothesis is that the induced alkalosis may have increased glycogenolytic/ glycolytic flux via the allosteric upregulation of both glycogen phosphorylase (GP) and phosphofructokinase (PFK) (13). In the NaHCO₃ condition, plasma $[HCO_3^-]$ was higher in the second half of the IST compared with the first half. Therefore, the improved second-half performance in the NaHCO₃ trial may have been due to enhanced glycogenolytic/glycolytic flux and glycogen utilization as a result of the greater blood $[HCO_3^-]$ in the second half of the IST compared with the first-half and the placebo conditions. Further research is required to determine if NaHCO₃ ingestion is able to enhance glycogenolytic/glycolytic flux during prolonged intermittent exercise.

It is difficult to explain why significant ergogenic effects were only observed in selected second-half sprints, typically at the beginning and toward the end of this half. It is interesting to note, however, that the greatest difference in plasma [HCO₃⁻] between the two conditions occurred at these two time points (9.5 and 9.4 mmol·L⁻¹, respectively). Furthermore, although the pattern of improvement may seem random, in those sprints at the beginning and toward the end of the second half that were not significantly different, there was a large variability in the sprint scores that reduced the power of the ANOVA to detect change.

In contrast to the second-half results, there was no significant difference in performance of any of the sprints during the first half of the IST between conditions. The improved performance in the second half of the IST, and not the first half, may have been related to the ingestion protocol. The greatest increase in plasma $[HCO_3^-]$ and pH level in the bicarbonate condition was seen in the second half. Therefore, the increase in extracellular alkalosis may not have been great enough in the first half to increase performance by the mechanisms already mentioned. The NaHCO₃ was administered in two dosages taken 90 and 20 min before the start of the IST. It is possible that increases in $[HCO_3^-]$ in the second half of the IST were due to the second dose or to an additive effect of the two dosages on blood $[HCO_3^-]$. Further research is required to determine whether first-half

performance of the IST would have been improved if the two bicarbonate dosages had been ingested earlier (e.g., 120 and 90 min before the IST).

SUMMARY

It was hypothesized that the ingestion of NaHCO₃ would enhance the extracellular $[HCO_3^-]$ and aid the performance of an IST designed to simulate the playing requirements of a team-sport game. The preexercise ingestion of NaHCO₃ affected a significant increase in the extracellular $[HCO_3^-]$ and improved the performance of the IST. Compared with

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the placebo condition, the ingestion of NaHCO₃ significantly increased blood pH during the IST and resulted in a significantly higher posttest blood [La⁻]. The ingestion of NaHCO₃ was ergogenic during the second-half performance of the IST, with subjects completing significantly more work in 7 of 18 second-half sprints. However, the total work completed and mean peak power achieved were not significantly different between conditions in either half of the IST. The relatively high posttest pH and [HCO₃⁻] in both conditions was an indication that the accumulation of H⁺ in the extracellular compartments did not exceed the extracellular buffer capacity.

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