Title

Effect of oral administration of sodium bicarbonate on surface EMG activity during repeated cycling sprints

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Abstract: The purpose of this study was to determine the effect of oral administration of sodium bicarbonate ($NaHCO_3$) on surface electromyogram (SEMG) activity from the vastus lateralis (VL) during repeated cycling sprints (RCS). Subjects performed two RCS tests (ten 10-s sprints) interspersed with both 30-s and 360-s recovery periods 1 h after oral administration of either $NaHCO_3$ (RCS_{Alk}) or $CaCO_3$ (RCS_{Pla}) in a random counterbalanced order. Recovery periods of 360 s were set before the 5th and 9th sprints. The rate of decrease in plasma HCO_3^- concentration during RCS was significantly greater in RCS_{Alk} than in RCS_{Pla}, but the rates of decline in blood pH during the two RCS tests were similar. There was no difference between change in plasma lactate concentration in RCS_{Alk} and that in RCS_{Pla}. Performance during RCS_{Alk} was similar to that during RCS_{Pla}. There were no differences in oxygen uptake immediately before each cycling sprint ($pre\dot{V}O_2$) and in SEMG activity between RCS_{Alk} and RCS_{Pla}. In conclusion, oral administration of $NaHCO_3$ did not affect SEMG activity from the VL. This suggests that the muscle recruitment strategy during RCS is not determined by only intramuscular pH.

Key words: Metabolic alkalosis, Surface electromyogram, Sprint exercise, Muscle fatigue

Introduction

In most of the popular team sports (e.g., soccer, tennis and basketball), a short-term sprint is repeated with passive recovery and/or low-intensity exercise. When such an intermittent sprint exercise is performed, exercise-induced muscle fatigue results in a progressive decline in power output.

Gaitanos et al. (1993) assumed that a decline in power output during intermittent sprint exercise resulted from depleted energy substrate or accumulated metabolites. However, in their experiment, phosphocreatine (PCr), muscle glycogen or ATP level did not decrease sufficiently to be regarded as limiting for muscle contraction (St Clair Gibson et al. 2001). Alternatively, some researchers have suggested that depleted energy substrate or accumulated metabolites contribute to muscle fatigue as an afferent signaler to the central nervous system (CNS) (Gandevia 1998; Lambert et al. 2005; St Clair Gibson and Noakes 2004; St Clair Gibson et al. 2001). They have argued that the afferent signal to the CNS indicates that this level of intensity repeated again would damage the muscle issue or lead to failure of homeostasis and that subsequently the efferent motor command and power output are inhibited. It is not clear whether such a down-regulation of the efferent motor command results from a feed-forward process as previous experience and knowledge or from a feedback process as response to the afferent signal from peripheral muscles.

Since glycolysis contributes to the energy supply during a short-term (6-10 s)

cycling sprint (Gaitanos et al. 1993; Nevill et al. 1996), and in repeated cycling sprints (RCS), lactic acid accumulates within muscle, resulting in an increase in hydrogen ions (H^+) within muscle. The reduced intramuscular pH due to intramuscular accumulation of H^+ had been reported to decrease power output through impairment of the muscle contractile apparatus (Cooke et al. 1988; Godt and Nosek 1989). However, a recent study using mammalian skinned muscle fibers (Pate et al. 1995) has shown that the reduced intramuscular pH does not adversely affect the muscle contractile apparatus at physiological temperatures. Furthermore, Bruton et al. (1998) have suggested that accumulated lactate and H^+ delay the onset of fatigue. Thus, recent studies have shown a positive effect of lactate and H^+. Nevertheless, since these studies showing a positive effect of lactate and H^+ were *in vitro* studies, it seems to be difficult to extrapolate these findings to an *in vivo* condition (Lamb et al. 2006). In addition, it has been suggested that lowered pH in an *in vivo* condition down-regulates the activation of pH-sensitive glycolytic enzymes (Fitts 1994). Therefore, it is likely that the decline in intramuscular pH during RCS detrimentally influences muscle tissue and homeostasis.

Thus, since it is possible that a decline in intramuscular pH during RCS is involved in the afferent signal that induces inhibition of the efferent motor command, it is likely that manipulation of intramuscular pH during RCS alters the efferent motor command and affects the muscle recruitment strategy. Many studies have shown that ingestion of sodium bicarbonate ($NaHCO_3$) delays the decline in intramuscular pH

during exercise (Bishop et al. 2004; Bouissou et al. 1989; Gaitanos et al. 1991; Stephens et al. 2002). Therefore, we hypothesized that oral administration of $NaHCO_3$ affects the muscle recruitment strategy during RCS. Since Enoka and Stuart (1992) have suggested that a shift to lower frequencies of an electromyogram (EMG) power spectrum during maximal voluntary contraction (MVC) is due to the selective recruitment of type I muscle fibers, recordings of surface EMG (SEMG) activity during each sprint in RCS would allow investigation of the muscle recruitment strategy during RCS.

The purpose of this study was to determine the effect of oral administration of $NaHCO_3$ on SEMG activity during RCS. To the best of our knowledge, there have been no studies in which the effect of oral administration of $NaHCO_3$ on SEMG activity during RCS was examined. The RCS used in this study included both 30-s and 360-s recovery periods. It takes approximately 360 s to resynthesize PCr completely after high-intensity exercise (Arsac et al. 2004; Harris et al. 1976; McCann et al. 1995). Therefore, since it is likely that SEMG activity during a cycling sprint immediately after a 360-s recovery period was hardly influenced by the degree of resynthesis of PCr, the use of a 360-s recovery period in RCS would reveal the relationship between delay of decline in intramuscular pH induced by oral administration of $NaHCO_3$ and SEMG activity. Furthermore, the use of a 360-s recovery period at the last stage of RCS would enable determination of the relationship between delay of decline in intramuscular pH

induced by oral administration of $NaHCO_3$ and SEMG activity in a more acidic condition.

Methods

Subjects

Eight healthy male undergraduate students participated in the present study. The subjects' mean age, height and body weight were 20.8 ± 2.1 (SD) yr, 173.4 ± 10.0 cm and 66.0 ± 9.2 kg, respectively. They had no neuromuscular disorders and were participating in regular training programs. Subjects were non-smokers. Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for three tests. The time interval between two consecutive tests was at least 2 days, and all tests were completed within 2 weeks. On the first test day, the subjects' body characteristics were measured and each subject performed the 1st-4th cycling sprints of the experimental protocol described below to become familiarized with RCS. Body mass (BM) was used to determine the loads of the cycling sprint. Each subject was instructed to refrain from intense physical exercise,

drinking, and taking caffeine for 24 h prior to each visit.

Experimental protocol

Each subject performed two RCS tests with oral administration of either $NaHCO_3$ (alkalosis condition: RCS_{Alk}) or $CaCO_3$ (placebo condition: RCS_{Pla}) in a randomized, counterbalanced order on separate days. Each subject came to the laboratory 3 h before the start of the test. First, the subjects ingested $NaHCO_3$ (RCS_{Alk}) or $CaCO_3$ (RCS_{Pla}) equivalent to a total dose of 0.3 g·kg BM^{-1}. This total dose was equally divided into 6 parts and each of the 6 parts was ingested with 350 mL of water at intervals of 10 minutes (Jones et al. 1977). The order in experimental conditions remained unknown to the subjects (single-blind method). Since $NaHCO_3$ and $CaCO_3$ were wrapped in wafers, the subjects did not know which they were ingesting. Experimental instruments were fitted to each subject 1 h after this administration procedure had been completed. Then, after resting for 3 min on the bicycle seat, each subject started an RCS test. The test consisted of ten 10-s cycling sprints with eight 30-s passive recovery periods immediately before the 2nd, 3rd, 4th, 6th, 7th, 8th, 10th sprints, respectively, and two 360-s passive recovery periods immediately before the 5th and 9th sprints, respectively. All cycling sprints were performed with a resistive load [N] of $0.075 \cdot BM \cdot 9.81^{-1}$ (Ayalon et al. 1974). Subjects were instructed to pedal as many revolutions as possible during cycling sprints. The number of sprints in RCS was

not announced in advance to prevent the subjects from making an unconscious plan for whole peak power output. Subjects were instructed that the number of sprints in RCS was not necessarily same in the two treatments (RCS_{Alk} and RCS_{Pla}). For all tests, subjects were in the seated position during exercise and recovery. All sprints started from a stationary position.

Repeated cycling sprints

All exercise tests were carried out on a bicycle ergometer (POWERMAX-V_{II}, Combi, Tokyo, Japan). The duration and load were adjusted by a built-in computer. The computer also calculated peak rpm (Rpm_{peak}) for a given exercise and displayed the results. Since the screen on the bicycle ergometer displayed Rpm_{peak}, the screen was covered. Time series behavior in rpm during each cycling sprint was recorded by an online computer at a rate of 10 Hz. Each subject's feet were strapped to the pedals to prevent them from slipping. The seat height was adjusted so that there was a slight bend in the knee joint when the foot pedal was at its lowest position. Peak power output (PPO) and mean power output (MPO) during each cycling sprint were calculated by methods of Lakomy (1986). It was assumed that the external mechanical work performed on this type of cycle ergometer was equal to the work dissipated against braking forces (i.e., inertia of the flywheel, rolling resistance of the flywheel, and freewheel mechanism) plus the kinetic energy of the flywheel. Thus, "effective

load" on the cycle ergometer was determined. This is the load required to prevent acceleration of the flywheel and consisted of not only resistive load that was applied to the flywheel but also "excess" load that would be required to stop the subject from accelerating the flywheel at any instant (Gaitanos et al. 1993; Lakomy 1986).

The results of rpm were averaged over 1-s time intervals and acceleration was calculated from the averaged rpm. The "excess load" was calculated from the acceleration. The instantaneous product of average 1-s rpm and effective load (resistive load + excess load) was used to determine corrected power output throughout RCS:

$$\text{Power output [W]} = \text{rpm} \cdot 6 \cdot \text{effective load [N]} \cdot 0.624^{-1},$$

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll [m], and 0.624 is the value for transforming Nm units to W units [$\text{Nm} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$]. The maximum product of rpm and effective load, during each sprint, is referred to as PPO. The average 1-s product of rpm and effective load, over the total time period of each cycling sprint (i.e., 10 s), is referred to as MPO. To reduce variation due to the difference in body characteristics of subjects, PPO and MPO divided by BM ($\text{PPO} \cdot \text{BM}^{-1}$ and $\text{MPO} \cdot \text{BM}^{-1}$, respectively) were used.

Plasma lactate concentration and blood gas

Blood samples (125 or 25 μL) were collected from fingertips using capillary tubes. The 25-μL samples were analyzed using a lactate analyzer (YSI-1500 sport, YSI,

Tokyo, Japan) to measure plasma lactate concentration ([La^-]). The lactate analyzer was calibrated by a standard lactate solution of 5 mmol·L^{-1} before each test. The 100-μL samples were analyzed using a blood gas analyzer (i-STAT, i-STAT Corporation, United States) to measure oxygen partial pressure (PO_2), plasma sodium concentration ([Na^+]), blood pH and plasma HCO_3^- concentration ([HCO_3^-]). The blood gas analyzer was calibrated by known calibration liquid (pH: 7.43, PCO_2: 30 torr, PO_2: 160 torr, [Na^+]: 140 mEq·L^{-1}, [K^+]: 4 mEq·L^{-1}) before each test. Blood was sampled before the administration of either $NaHCO_3$ or $CaCO_3$ (Pre-Ad: 125μL), 1 h after the administration (Post-Ad: 125 μL), 30 s before the 5th and 9th sprints (Pre5 and Pre9, respectively: 25 μL), and immediately after the end of RCS (Post-Ex: 125 μL). In the present study, only blood gas data in which PO_2 was not lower than 70 torr were analyzed.

Oxygen uptake

Data on oxygen uptake ($\dot{V}O_2$) were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation ($\dot{V}E$) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 L). O_2 and CO_2 concentrations were measured by a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O_2: 15.17%, CO_2: 4.92%). $\dot{V}O_2$ was measured continuously

during rest, exercise, and recovery periods. For each 10-s interval, the average of $\dot{V}O_2$ was calculated.

Surface electromyogram analysis

An SEMG was recorded from the left vastus lateralis (VL) at a rate of 1000 Hz during each of the ten cycling sprints. Before attachment of the surface electrodes, the skin was shaved, abraded, and cleaned with alcohol in order to reduce skin impedance. A bipolar surface EMG sensor (SX230, Biometrics Ltd, United Kingdom; inter-electrode distance of 20 mm) was placed on the lateral side of the crural area five-fingers proximal from the patella of the belly of the VL in the main direction of muscle fibers. The ground electrode was placed over the styloid process of the right wrist. The SEMG signals were amplified using an amplifier imbedded in the EMG sensor (bandwidth = 20-450 Hz; common mode rejection ratio, CMRR > 96 dB; input impedance > 10 TΩ; gain = 1000) and converted into digital signals using an analog-digital converter (MacLab/8s, AD Instruments, Australia). Then SEMG data were processed offline by using analysis software (Acknowledge, BIOPAC Systems, United States). Raw data were filtered using a band-pass Finite Impulse Response filter with cut-off frequencies of 5 and 500 Hz. The SEMG activity during RCS was determined by measuring the mean value of the root mean square (RMS) and mean power frequency (MPF, described below) of the signal between the onset and the end

of the burst (Racinais et al. 2007). Burst onsets and offsets were determined using a constant electric threshold of ± 0.2 mV (Billaut et al. 2005; Racinais et al. 2007). The electric signal of each burst was processed with fast Fourier transform (FFT) to obtain each frequency power spectrum. The frequency spectrum analysis was restricted to frequencies in the range 5-500 Hz (Kay et al. 2001; St Clair Gibson et al. 2001). The MPF was defined as the ratio between spectral moments of orders one and zero (Moritani et al. 1982). Because peak power was attained in 3.2 ± 1.0 s, during the sixth pedal revolution, in the present study, values of RMS and MPF for the fifth and the sixth pedal revolutions were averaged to obtain one value for the peak power development phase of each cycling sprint (Racinais et al. 2007). The position of the electrodes for SEMG detection was similar in the two conditions because reference points were marked on the skin. The RMS and MPF were normalized as a percentage of the 1st sprint value.

Statistical analysis

Results are presented as means ± standard deviations (SD). Two-way ANOVA for repeated measures on both factors (time × treatment) was used. When main effects were found, the means were compared by using Tukey-Kramer's post-hoc test. If a significant interactive effect was indicated, one-way ANOVA for repeated measures was used to examine the time effect, and a paired t-test was used to examine the

treatment effect. A value of $P < 0.05$ was regarded as statistically significant.

Results

Peak power output and mean power output

Fig. 1 shows the PPO·BM^{-1} and MPO·BM^{-1} data for RCS_{Alk} and RCS_{Pla}. No significant interaction ($P > 0.1$) was found in both PPO·BM^{-1} and MPO·BM^{-1}, and no significant treatment effect ($P > 0.1$) was found in both PPO·BM^{-1} and MPO·BM^{-1}. The values of PPO·BM^{-1} were significantly lower in the 3rd, 4th, 7th, 8th and 9th sprints than in the 1st sprint in both treatments, and the values of MPO·BM^{-1} were significantly lower in the 3rd, 4th, 6th, 7th, 8th, 9th and 10th sprints than in the 1st sprint in both treatments.

Blood

No significant interaction ($P > 0.1$) was found in plasma [La^-], and no significant treatment effect ($P > 0.1$) was found in plasma [La^-]. In both treatments, the values of plasma [La^-] were significantly higher in Pre5, Pre9 and Post-Ex than in Post-Ad, and the values of plasma [La^-] in Pre9 and Post-Ex were significantly higher than the value in Pre5 (Table 1).

PO_2 values in all blood gas data were not lower than 70 torr (range: 70-108 torr). In blood pH, the interactive effect was tendency to be significant ($P = 0.07$). Therefore,

one-way repeated ANOVA and paired t-test were used. Blood pH was significantly increased at 1 h after the administration of $NaHCO_3$ but was not increased at 1 h after the administration of $CaCO_3$ (Table 1). In both RCS_{Alk} and RCS_{Pla}, blood pH was significantly decreased at the end of RCS compared to the values in Pre-Ad and Post-Ad. Blood pH values in Post-Ad and Post-Ex were significantly higher in RCS_{Alk} than in RCS_{Pla} (Table 1). The difference between blood pH values in Post-Ad and Post-Ex (Δblood pH) during RCS_{Alk} was similar to that during RCS_{Pla} ($P > 0.1$, Fig. 2).

In plasma $[HCO_3^-]$, the interactive effect was significant. Plasma $[HCO_3^-]$ was significantly increased at 1 h after the administration of either $NaHCO_3$ or $CaCO_3$ (Table 1). In both RCS_{Alk} and RCS_{Pla}, the value of plasma $[HCO_3^-]$ at the end of RCS was significantly lower than the values in Pre-Ad and Post-Ad. The values of plasma $[HCO_3^-]$ in Post-Ad and Post-Ex during RCS_{Alk} were significantly higher than those during RCS_{Pla} (Table 1). The difference between plasma $[HCO_3^-]$ in Post-Ad and Post-Ex (Δplasma $[HCO_3^-]$) during RCS_{Alk} was significantly higher than that during RCS_{Pla} ($P < 0.05$, Fig. 2). In plasma $[Na^+]$, the interactive effect was significant. In RCS_{Alk}, plasma $[Na^+]$ was significantly higher in Post-Ad and Post-Ex than in Pre-Ad, and plasma $[Na^+]$ in Post-Ex was significantly higher than that in Post-Ad (Table 1). In RCS_{Pla}, the values of plasma $[Na^+]$ in Post-Ad and Post-Ex were significantly lower and higher, respectively, than the value in Pre-Ad and plasma $[Na^+]$ in Post-Ex was significantly higher than that in Post-Ad (Table 1). The values of plasma $[Na^+]$ in

Post-Ad and Post-Ex during RCS_{Alk} were significantly higher than those during RCS_{Pla}.

Oxygen uptake immediately before each cycling sprint

Changes in $\dot{V}O_2$ immediately before each cycling sprint ($pre\dot{V}O_2$) during the two RCS tests are shown in Fig. 3. In $pre\dot{V}O_2$, no significant interaction ($P > 0.1$) or significant treatment effect ($P > 0.1$) was found. In both treatments, $pre\dot{V}O_2$ values before the 2nd, 3rd, 4th, 6th, 7th, 8th and 10th sprints were significantly higher than the value before the 1st sprint and there were no significant differences between $pre\dot{V}O_2$ values before the 5th and 9th sprints and that before the 1st sprint.

Surface electromyogram

There were no differences between the two RCS tests in absolute RMS value (VL: $P = 0.29$) and absolute MPF value (VL: $P = 0.67$) in the 1st sprint. Changes in RMS and MPF in the two RCS tests are shown in Fig. 4. In both RMS and MPF, no significant interaction ($P > 0.1$) was found and no significant treatment effect ($P > 0.1$) was found. In both treatments, MPFs significantly decreased in the 3rd and 7th sprints compared to those in the 1st sprint.

Discussion

The main finding in the present study was that oral administration of $NaHCO_3$ did not affect SEMG activity during RCS.

Induction of metabolic alkalosis by oral administration of $NaHCO_3$

Oral administration of $NaHCO_3$ raised blood pH and plasma [HCO_3^-] in Post-Ad. The magnitude of increase in plasma [HCO_3^-] was slightly greater than that in previous studies (Costill et al. 1984; Gaitanos et al. 1991; Stephens et al. 2002). The magnitude of increase in blood pH was similar to that in a study by Bishop et al. (2004). Consistent with the results of previous studies (Costill et al. 1984; Stephens et al. 2002), oral administration of $NaHCO_3$ did not change plasma [La^-] in Post-Ad. Oral administration of $CaCO_3$ also increased plasma [HCO_3^-] in Post-Ad but did not increase blood pH in Post-Ad. Therefore, it seems that in only RCS_{Alk} metabolic alkalosis was induced, allowing an increase in blood pH.

Delay of decline of intramuscular pH by oral administration of $NaHCO_3$

During exercise, induced metabolic alkalosis increases the rate of muscle H^+ efflux into blood, resulting in a delay of decline in intramuscular pH (Bishop et al. 2004). The possible mechanisms responsible for the delay include increased skeletal muscle lactate/H^+ cotransporter activity, increased Na^+/H^+ exchanger, and/or increased strong ion difference (SID) (Stephens et al. 2002). If induced metabolic alkalosis

enhanced muscle lactate/H^+ cotranspoter activity in the present study, higher plasma [La^-] in RCS_{Alk} would be expected. However, plasma [La^-] was the same in the two treatments. Therefore, it is unlikely that muscle lactate/H^+ cotransporter activity was enhanced in RCS_{Alk}. Since plasma [Na^+] significantly increased after oral administration of $NaHCO_3$, if this was associated with increased muscle [Na^+] with alkalosis, it would be expected that there would be a corresponding removal of H^+ from the muscle by the muscle Na^+/H^+ exchanger (Juel et al. 1998; Stephen et al. 2002). Furthermore, it has reported that an increase in muscle [Na^+] after oral administration of $NaHCO_3$ would induce an increase in SID, resulting in a decrease in muscle H^+ concentration (Heigenhauser and Johnson 1991; Johnson et al. 1996). Therefore, it is thought that in RCS_{Alk} more H^+ was released from the muscle to blood due to increased Na^+/H^+ exchanger or increased SID. However, this speculation does not necessarily mean that oral administration of $NaHCO_3$ induced a delay in decline of intramuscular pH because it is possible that the difference between the rates of H^+ generation in the two conditions results in similar intramuscular pH in both conditions. In the present study, there were no differences in both performance (PPO and MPO) and plasma [La^-] between the treatments, suggesting that contribution of glycolysis was similar in RCS_{Alk} and RCS_{Pla}. It is inferred from these results that there was no difference between the rates of H^+ generation in the treatments. Thus, it is presumed that a delay in decline of intramuscular pH was induced in RCS after oral

administration of $NaHCO_3$.

The magnitudes of Δblood pH in RCS_{Alk} and RCS_{Pla} were the same, and the magnitude of Δplasma [HCO_3^-] was greater in RCS_{Alk} than in RCS_{Pla}. These results indicate that in RCS_{Alk} greater plasma H^+ was buffered by plasma HCO_3^-. Most of the greater buffered plasma H^+ during RCS_{Alk} would be the H^+ released from the muscle to blood. Therefore, it is thought that difference in intramuscular pH at the end of RCS between the treatments was similar to the difference in blood pH in Post-Ex.

Effect of 360-s recovery period during RCS on oxygen uptake

To eliminate effects of resynthesis of PCr on SEMG activity wherever possible, in the present study, a 360-s recovery period, which has been reported to be sufficient for complete resynthesis of PCr (Arsac et al. 2004; Harris et al. 1976; McCann 1995), was set immediately before the 5th and 9th sprints. Since Rossiter et al. (2002) reported that restoration of PCr after high-intensity exercise is accompanied by a decrease in $\dot{V}O_2$ during the recovery period, pre$\dot{V}O_2$ measured in the present study roughly predicts the degree of resynthesis of PCr. Because there was no difference in pre$\dot{V}O_2$ in Pre5 and Pre9 between the treatments, it seems plausible that the degree of PCr resynthesis over the 360-s recovery periods in RCS_{Alk} was similar to that in RCS_{Pla}. In addition, in both RCS_{Alk} and RCS_{Pla}, since there was no difference between pre$\dot{V}O_2$ before the 1st sprint (at rest) and the values before the 5th and 9th sprints, it is thought

that PCr would have been almost completely restored in the 360-s recovery periods. This idea is supported by the results of a study by Sahlin et al. (1997) showing that muscle biopsy PCr contents in mixed muscle fibers recovered 5 min after exhaustion.

Effect of oral administration of $NaHCO_3$ on SEMG activity during RCS

Oral administration of $NaHCO_3$ did not affect SEMG activity from the VL during RCS. This result indicates that the delay in decline of intramuscular pH does not affect SEMG activity during RCS. An EMG power spectrum has been reported to shift to lower frequencies due to a decline in intramuscular pH (Hagberg 1981; Komi and Tesch 1979), but Bouissou et al. (1989) have shown that there is no correlation between a decline in intramuscular pH and a decrease in MPF. Furthermore, many authors have reported that a slowdown of muscle fiber action potential conduction velocity (MFCV) prolongs the muscle fiber action potential wave form (Lindström et al. 1970), which in turn brings about a decrease in MPF (Moritani et al. 1986; Stulen and De Luca 1981). In view of the results of a previous study showing that reduced intramuscular pH does not directly have an adverse effect on E-C coupling at physiological temperatures (Pate et al. 1995), it is unlikely that a slowdown of MFCV results from reduced intramuscular pH. Therefore, it is likely that the changes in MPF during exercise are indicative of difference in the MFCV characteristic (i.e., fiber type) of recruited muscle fibers (Matsuura et al. 2006). Thus, the results in the present study

suggest that delay in decline of intramuscular pH did not affect fiber type recruited during RCS.

The RMS calculated from SEMG is indicative of the number of motor units (MUs) recruited and/or the rate of discharge of the recruited MUs in exercising muscle (Lind and Petrofsky 1979; Moritani et al. 1982). Since there was no difference in RMS from the VL between the two conditions, there would also be no difference in both recruitment and the rate of discharge of MUs between the treatments. In both RCS_{Alk} and RCS_{Pla}, MPF values in the 3rd and 7th sprints significantly decreased compared to that in the 1st sprint, suggesting that muscle fibers with slower MFCV were recruited in the 3rd and 7th sprints. RMS in the 3rd and 7th sprints should have decreased because the rate of discharge of muscle fibers with slower MFCV is lower. The shift in the EMG power spectrum to lower frequencies might cause recordings of more EMG signal since muscle tissue and skin act as low-pass filters (Bouissou et al. 1989; Lindström et al. 1977). It is inferred from these results of SEMG activity that delay in decline of intramuscular pH did not affect the muscle recruitment strategy in a feedback manner during RCS.

Afferent signal from peripheral muscles and feedback mechanism

Indeed, it has been suggested that efferent motor command is also inhibited in a feed-forward manner during exercise. Several authors (St Clair Gibson and Noakes

2004; Ulmer 1996) have argued that during exercise an individual subconsciously sets power output to maintain homeostasis before the onset of the exercise on the basis of subconscious mental calculations for energy reserve and predicted energy cost of the exercise. Therefore, the afferent signal for intramuscular pH may contribute to subconscious setting of power output before the onset of exercise as information for previously experienced exercise. However, it has also been suggested that power output set previously in a feed-forward manner is modified by afferent signals for changes in muscle metabolites and/or changes in cardiovascular function during exercise (Lambert et al. 2005; Noakes et al. 2005; St Clair Gibson et al. 2001). These ideas are not consistent with the results of the present study. This discrepancy may be explained as follows.

In the present study, the number of sprints in RCS was not announced over the experimental period. If the number of sprints in RCS was announced, subjects might have adjusted power output so as not to induce failure of homeostasis before RCS is completed and to reach the limit of maintenance of homeostasis at the end of RCS. In that case, information for the critical level of intramuscular pH given by the afferent signal for intramuscular pH would have be beneficial to maintain homeostasis. However, if the number of sprints in RCS is not announced, since subjects do not subconsciously adjust power output to reach the limit of maintenance of homeostasis at the end of RCS, it seems that the importance of information for the critical level of

intramuscular pH is reduced.

Effect of oral administration of $NaHCO_3$ on relationship between performance and SEMG activity during RCS

In both RCS_{Alk} and RCS_{Pla}, despite the fact that SEMG activity in the 9th sprint was similar to that in the 1st sprint, PPO and MPO significantly decrease in the 9th sprint. This suggests that a decline in intramuscular pH accompanied by progress of RCS peripherally depressed performance during RCS through an inhibitory effect on glycolysis. However, the manipulation of intramuscular pH by oral administration of $NaHCO_3$ did not affect performance. This disagreement may be explained by exercise duration of sprints used in the present study. In fact, for MPO, in which contribution of glycolysis is greater, 6 of the 8 subjects generated greater power in RCS_{Alk} than in RCS_{Pla} in the 9th sprint. Therefore, in a longer sprint duration, oral administration of $NaHCO_3$ might affect performance.

Conclusion

Oral administration of $NaHCO_3$ did not affect SEMG activity during RCS. It is thought that the muscle recruitment strategy during RCS is not determined by only intramuscular pH. Furthermore, it is possible that performance during RCS is affected by both peripheral factors and muscle recruitment strategy.

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Table

Table 1 Plasma lactate concentration ($[La^-]$), blood pH, plasma bicarbonate concentration ($[HCO_3^-]$), and plasma sodium concentration ($[Na^+]$) at rest (Pre-Ad), post-administration (Post-Ad), before the 5th and 9th sprints (Pre5 and Pre9) during repeated cycling sprints (RCS), and at the end of RCS after the administration of $NaHCO_3$ (RCS_{Alk}) or $CaCO_3$ (RCS_{Pla}).

				RCS		
	Trial	Pre-Ad	Post-Ad	Pre5	Pre9	Post-Ex
plasma $[La^-]$ (mmol/L)	RCS_{Alk}	1.2 (0.3)	0.9 (0.2)	13.2 (2.6)^{b}	16.1 (3.3)^{b,c}	16.5 (3.0)^{b,c}
	RCS_{Pla}	1.2 (0.3)	0.9 (0.2)	12.1 (2.6)^{b}	14.1 (2.4)^{b,c}	14.1 (2.7)^{b,c}
blood pH	RCS_{Alk}	7.44 (0.01)	7.52 (0.03)^{a,*}			7.28 (0.07)^{a,b,*}
	RCS_{Pla}	7.43 (0.02)	7.45 (0.03)			7.21 (0.06)^{a,b}
plasma $[HCO_3^-]$ (mmol/L)	RCS_{Alk}	26.8 (1.4)	35.3 (1.8)^{a,*}			14.6 (3.4)^{a,b,*}
	RCS_{Pla}	26.3 (1.2)	28.1 (1.5)^{a}			11.5 (2.1)^{a,b}
plasma $[Na^+]$ (mmol/L)	RCS_{Alk}	139.1 (1.8)	141.0 (1.9)^{a,*}			143.1 (2.9)^{a,b,*}
	RCS_{Pla}	139.3 (1.8)	137.5 (1.8)^{a}			140.6 (2.3)^{a,b}

Values are means (SD); $n = 8$. [a]: significantly different from Pre-Ad ($P < 0.05$). [b]: significantly diffrent from Post-Ad ($P < 0.05$). [c]: significantly different from Pre5 ($P < 0.05$). [*]: significantly different from RCS_{Pla} ($P < 0.05$).

Figures

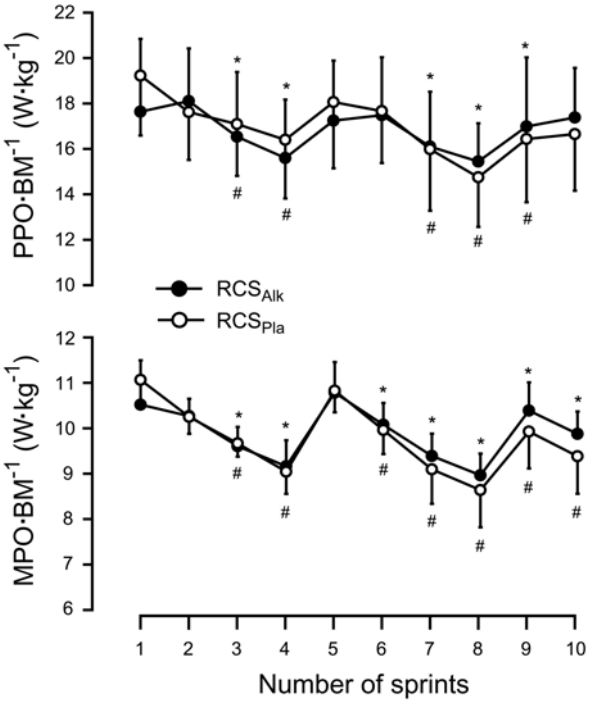

Figure 1

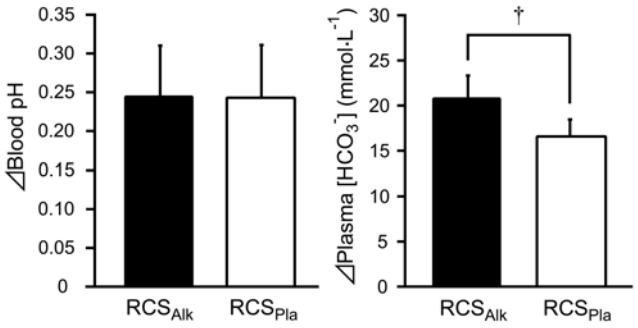
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0
⊿Blood pH
RCS$_{Alk}$
RCS$_{Pla}$
30
25
20
15
10
5
0
⊿Plasma [HCO_3^-] (mmol·L^{-1})
†
RCS$_{Alk}$
RCS$_{Pla}$

Figure 2

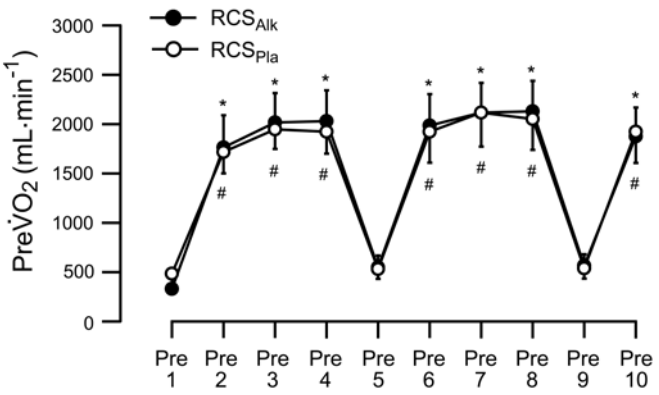
RCS$_{Alk}$
RCS$_{Pla}$
3000
2500
2000
1500
1000
500
0
Pre$\dot{V}O_2$ (mL·min^{-1})
Pre 1
Pre 2
Pre 3
Pre 4
Pre 5
Pre 6
Pre 7
Pre 8
Pre 9
Pre 10

Figure 3

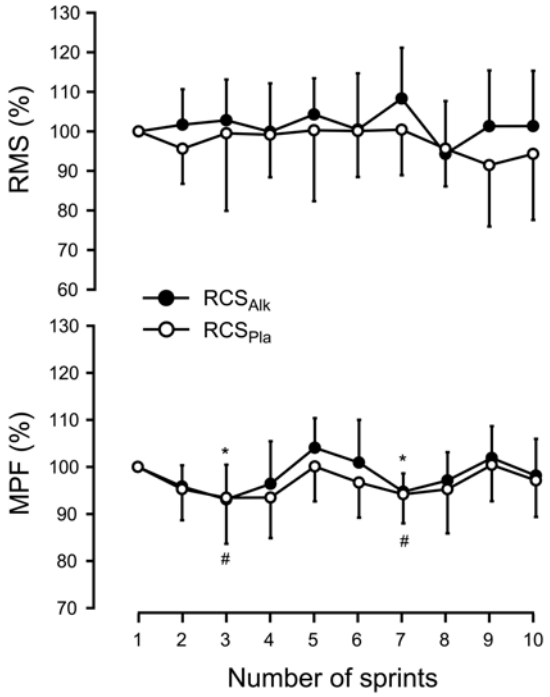

Figure 4

Figure legends

Fig. 1. Changes in peak power output (PPO) divided by body mass (BM) in each of the subjects ($PPO \cdot BM^{-1}$) and mean power output (MPO) divided by BM in each of the subjects ($MPO \cdot BM^{-1}$) during two repeated cycling sprints (RCS) tests with oral administration of either $NaHCO_3$ (RCS_{Alk}: ●) or placebo ($CaCO_3$) (RCS_{Pla}: ○). *: significantly different ($P < 0.05$) from the value in the 1st sprint in RCS_{Alk}. #: significantly different ($P < 0.05$) from the value in the 1st sprint in RCS_{Pla}.

Fig. 2. Difference between blood pH 1 h after the oral administration and that at the end of RCS (left: Δblood pH) and difference between plasma bicarbonate concentration ($[HCO_3^-]$) 1 h after the oral administration and that at the end of RCS (right: Δplasma $[HCO_3^-]$) during two RCS tests with oral administration of either $NaHCO_3$ (RCS_{Alk}: black bars) or placebo ($CaCO_3$) (RCS_{Pla}: blank bars). †: significant difference ($P < 0.05$) between the value in RCS_{Alk} and that in RCS_{Pla}.

Fig. 3. Changes in oxygen uptake ($\dot{V}O_2$) divided by body mass (BM) in each of the subjects for 10 s immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) during two repeated cycling sprints (RCS) tests with oral administration of either $NaHCO_3$ (RCS_{Alk}: ●) or placebo ($CaCO_3$) (RCS_{Pla}: ○). *: significantly different ($P < 0.05$)

from the value immediately before the 1st sprint (Pre1) in RCS_{Alk}. #: significantly different ($P < 0.05$) from Pre1 in RCS_{Pla}.

Fig. 4. Changes in RMS from the left vastus lateralis (top) and mean power frequency (MPF) from the left VL normalized by the 1st sprint value during two repeated cycling sprints (RCS) tests with oral administration of either $NaHCO_3$ (RCS_{Alk}: ●) or placebo ($CaCO_3$) (RCS_{Pla}: ○). *: significantly different ($P < 0.05$) from the value in the 1st sprint in RCS_{Alk}. #: significantly different ($P < 0.05$) from the value in the 1st sprint in RCS_{Pla}.