Hyperventilation as a Strategy for Improved Repeated Sprint Performance

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Abstract

Sakamoto, A, Naito, H, and Chow, C-M. Hyperventilation as a strategy for improved repeated sprint performance. *J Strength Cond Res* 28(4): 1119–1126, 2014–Repeated high-intensity sprints incur substantial anaerobic metabolic challenges and create an acidic muscle milieu that is unfavorable for subsequent performance. Hyperventilation, resulting in respiratory alkalosis, acts as a compensatory mechanism for metabolic acidosis. This study tested the hypothesis that hyperventilation performed during recovery intervals would attenuate performance decrement in repeated sprint pedaling. Thirteen male university athletes performed 10 sets of 10-second maximal pedaling on a cycle ergometer with a 60-second recovery between sets under control (spontaneous breathing) and hyperventilation conditions in a crossover counter-balanced manner. Pedaling load was set at 0.075 × body mass. Peak and mean power outputs were documented for each set to compare performance decrements for 10 sets between conditions. Hyperventilation (60 breaths per minute and end-tidal partial pressure of CO_2 maintained at 20–25 mm Hg) was performed 30 seconds before each sprint set. This intervention successfully increased blood pH by 0.03–0.07 but lowered P_{CO_2} by 1.2–8.4 mm Hg throughout exercise ($p < 0.001$). The peak and mean power outputs, and blood [La^-] accumulation were not significantly different between the conditions. However, a significant condition × time interaction existed for peak power ($p = 0.035$) and mean power ($p = 0.023$), demonstrating an attenuation in power decrement in later sprint sets with hyperventilation. In conclusion, hyperventilation implemented during recovery intervals of repeated sprint pedaling attenuated performance decrements in later exercise bouts that was associated with substantial metabolic acidosis. The practical implication is that hyperventilation may have a strategic role for enhancing training effectiveness and may give an edge in performance outcomes.

Key Words respiratory alkalosis, hypocapnia, power, fatigue, recovery

Introduction

The body acid-base status is usually maintained within the survival range by metabolic buffers of bicarbonate (HCO_3^-), protein (or hemoglobin), and phosphate buffers and the respiratory and renal systems (19,29). Training at high exercise intensities, however, creates a temporary acidic muscle milieu. The accumulation of hydrogen ions (H^+) and resulting fall in pH are associated with reduced activities of glycolytic enzymes (e.g., phosphofructokinase) with compromised glycolytic energy supply (22,29) and impaired excitation/contraction mechanisms (11,13,26,28). Therefore, these changes are unfavorable for performance. Training under shorter recoveries with minimal performance decrement may play crucial roles in maximizing training efficiency. This concept should also hold for better performance and game outcomes during intermittent sport competitions. A useful mechanism to reduce performance decrement and/or facilitate recovery for further sprint bouts is to alleviate or promptly reverse the acidic muscle environment. Proposed ergogenic strategies to date include sodium bicarbonate ingestion ($NaHCO_3$, metabolic alkalosis) (2,15,34) and hyperventilation (respiratory alkalosis) (10,25) according to the equilibrium reaction (19,29):

$$H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2 \qquad (1)$$

Ingestion of $NaHCO_3$ elevates plasma [HCO_3^-], rather than within muscles (3,12). The primary role of the elevated plasma HCO_3^- is to promote the uptake of H^+ in the circulation and produce a greater transmembrane H^+ gradient for acceleration of H^+ efflux out of the active muscles (12,15,30). By contrast, hyperventilation raises blood pH without the need for exogenous interventions (7,10,16,33). Hyperventilation-induced respiratory alkalosis establishes hypocapnia (through excretion of CO_2), which favors buffering of H^+ ions as per Equation 1. Respiratory

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alkalosis may alter both intra- and extracellular pH, thus pH may be more directly elevated or prevented from falling (5,7,25). Regardless of the technique used, theoretically, both may attenuate the unfavorable pH fall and enhance performance.

Nevertheless, induced alkalosis may be detrimental to endurance performance because it is associated with reduced blood flow and O_2 supply (alkalosis prevents vasodilatation of the active musculature); decreased O_2 availability for diffusion (alkalosis causes a left-shift of the oxyhemoglobin dissociation curve); and slowing of O_2 kinetics (with attenuated rate of activation of the oxidative enzyme, pyruvate dehydrogenase) (5,10). Moreover, O_2 delivery to the active muscles may be slightly compromised when hyperventilation is performed because of increased O_2 demand by the respiratory muscles (5,33). Given these detrimental effects, the ergogenic benefits of induced alkalosis could be maximized through (a) using selective exercise tasks that are intermittent, of high intensity, and of short duration (such as repeated sprints during which energy sources primarily come from phosphocreatine breakdown and glycolysis or glycogenolysis); (b) the practicable implementation of voluntary hyperventilation during the recovery period between exercise bouts; and (c) performance decrement should be largely accounted for by the accumulation of H^+ ions and pH fall, rather than a deficiency in aerobic energy supply (5,10).

In fact, $NaHCO_3$ ingestion studies that used varying exercise modes and intensities had demonstrated inconsistent and inconclusive results regarding ergogenic effects (2,3,12,14,15,30,34). However, the studies that most consistently confirmed these effects were when the exercise task primarily incurred a substantial whole-body anaerobic metabolic challenge consisting of intensive repeated bouts in highly fit individuals (2,3,15). Despite this, $NaHCO_3$ ingestion remains debatable given the adverse gastrointestinal effect (14) and controversies over optimal dosage and timing (24,34).

Hyperventilation, being a voluntary act, can be instantly initiated or immediately terminated to avoid long-standing body alkalosis. Based on such flexible utility, the use of hyperventilation may prove useful as an alternative ergogenic aid for intense intermittent sports. However, studies on hyperventilation have been limited to long duration or single bout exercises (7,25,33), which were either aerobically demanding or unsuitable for maximizing the buffering effects. Furthermore, these studies showed no ergogenic effects but decreased exercise time. No studies, to our knowledge, have investigated the effect of respiratory alkalosis by hyperventilation on performance during repeated bouts of intensive short sprints.

This study aimed to explore the effects of hyperventilation-induced alkalosis on sprint performance in highly power-trained athletes with dependent variables of interest being peak and mean power outputs, and secondary measures of blood pH, P_{CO_2}, and lactate concentration. It was hypothesized that hyperventilation implemented during the recovery intervals between sprint sets would attenuate decrements in sprint performance, expressed in peak and mean power, through a diminished fall in blood pH over successive sprint sets.

Methods

Experimental Approach to the Problem

This was a randomized crossover design with highly trained subjects undergoing both the control and hyperventilation conditions separated by 48–72 hours in a counter-balanced manner. Exercise protocol consisted of 10 sets of 10 seconds maximal pedaling on a cycle ergometer with a 60 seconds recovery between sets. For the control condition, spontaneous breathing was assumed during recovery. For the hyperventilation condition, voluntary (methodological) hyperventilation (60 breaths per minute with end-tidal partial pressure of CO_2 maintained at 20–25 mm Hg) took place during the last 30 seconds of each 60 seconds recovery period. Peak and mean power outputs achieved within each sprint set were recorded to evaluate the sprint performance decrement for 10 repeated sets. Blood lactate concentration was measured to reflect glycolytic metabolism. Blood pH and P_{CO_2} were analyzed to indicate respiratory alkalosis resulting from hyperventilation. Expired gases including minute ventilation ($\dot{V}E$), respiratory rate (RR), expired tidal volume (VT), and end-tidal partial pressure of CO_2 ($P_{ET}CO_2$) were monitored breath-by-breath to control for the methodological hyperventilation, and to document the altered ventilation patterns and ventilatory demands. Breath-by-breath $\dot{V}O_2$ and $\dot{V}CO_2$ were also measured to estimate additional respiratory work and CO_2 excretion consequent of hyperventilation.

Subjects

Thirteen male university athletes (6 road cyclists, 2 track cyclists, 2 track and field sprinters, 2 soccer players, and 1 rugby player), who were familiar with intense high-power intermittent movements and assumed to be capable of completing the exhaustive exercise protocol of this study, consented to participate in this study. Their age, height, and body mass were 21.2 ± 1.9 years, 172.7 ± 4.3 cm, and 67.3 ± 7.8 kg, respectively. They had competed in their sports for 6.8 ± 3.8 years and trained 5.6 ± 0.9 times per week at the time of experiment and were experienced in repeated maximal pedaling on a cycle ergometer. All subjects reported to the laboratory on 4 separate occasions: 2 on nonexperimental days and 2 on experimental days (control or hyperventilation condition). On the first nonexperimental day, they were informed of any risks associated with the experiment, read the guidelines and received verbal instructions for the experimental procedures, and gave informed consent. On the second day, subjects practiced

the actual experimental procedures simulating the hyperventilation condition up to the third sprint set to become familiarized with the maximal pedaling and hyperventilation methods used in this study. On each of the subsequent experimental days, subjects were instructed to eat a carbohydrate-rich meal 2 hours before the exercise test to ensure fuel repletion, and encouraged to consume plenty of water. No subjects consumed alcoholic or caffeinated drinks during the experimental period. This study was approved by the Human Ethics Committee of Juntendo University, Graduate School of Health and Sports Science.

Procedures

Repeated Sprints. The exercise test consisted of a 3-minute rest on the saddle followed by 10 sets of 10 seconds standing sprints with 60 seconds recovery between sprint sets on a cycle ergometer (Powermax-V_{II}; Combi Wellness, Tokyo, Japan). The pedaling load (kp) was set at 7.5% body mass based on the standard Wingate test (20). Saddle and handle heights were selected during the familiarization session and were kept the same for subsequent experiments. Three seconds before each sprint set, subjects took a standing position and waited for the "go" signal. Strong verbal encouragement was given for each sprint to elicit maximal performance. The cycle ergometer was connected to a personal computer (PC), with power output being recorded at 10 Hz using the auxiliary software (Powermax-V_{II}; Combi Wellness). For each sprint set, peak and mean power outputs were detected to evaluate the overall performance change per condition. Peak power was defined as the highest power value attained within each sprint set, whereas mean power was calculated by taking the average of all power values recorded for each 10-second sprint set. The experiment was conducted between August and December 2011. All trials were performed in a temperature and humidity controlled room at 20° C and 64%, respectively.

Before each exercise test, subjects performed a warm-up on the ergometer for at least 10 minutes and their usual warm-up techniques if needed, for optimal performance. After completion of the warm-up exercises, all subjects performed a single set of the actual sprint pedaling as a specific practice before each experimental condition (pretest), followed by the exercise test within 10 minutes. The peak and mean power outputs recorded during this specific practice set (pretest values) were used to assess the test-retest reliability. To ensure euhydration for best performance, subjects consumed at least 500 ml of standard mineral water between the warm-up and commencement of the exercise test.

Spontaneous Breathing and Hyperventilation. For the control condition, spontaneous breathing was assumed during each recovery period, whereas for the hyperventilation condition, the methodological hyperventilation occurred during the last 30 seconds of each recovery period until the next sprint set. The first sprint set was also preceded by 30 seconds of hyperventilation to examine the "preparatory effect" of respiratory alkalosis. During both the initial rest and the recovery period, subjects were seated on the saddle with no or minimal leg movements. Sudden changes in breathing patterns due particularly to puffing or unconscious deep breaths were avoided until the burst into each sprint set, especially during the control condition. No breathing instructions were given for the sprinting duration. The 2 experiments were conducted at around the same time of day, within 2 hours of each other, for each subject.

Respiratory Monitoring. Expired gas was monitored breath-by-breath on a PC screen during both conditions using an aeromonitor (AE300s; Minato Medical Science, Osaka, Japan) through a fitted facial mask for $\dot{V}E$, RR, VT, $P_{ET}CO_2$, $\dot{V}O_2$, and $\dot{V}CO_2$. The airflow sensor was calibrated using a 2-L syringe, and O_2 and CO_2 sensors with known O_2 and CO_2 gas concentrations in accordance with the manufacturer's instructions before each experiment. When performing hyperventilation, RR was set at 60 breaths per minute using a metronome (set at 120 $b \cdot min^{-1}$: inspiration or expiration per beat) according to our pilot tests for $\dot{V}E$ and RR to necessarily achieve the desired $P_{ET}CO_2$ until the last sprint set. Expired tidal volume was always adjusted according to the $P_{ET}CO_2$ value read out by an investigator to make it fall between 20 and 25 mm Hg so as to induce respiratory alkalosis (7). For $\dot{V}E$, RR, VT, and $P_{ET}CO_2$, the values averaged for the last 5 seconds before (5-second presprint) and the first 5 seconds after each sprint set (5-second postsprint) were used for statistical analyses, whereas for $\dot{V}O_2$ and $\dot{V}CO_2$, the values were averaged for the entire intervention duration of 30 seconds before each sprint set.

Blood Samples. A small amount of blood, from hyperemized earlobes, was collected into heparinized capillary tubes to measure the concentration of blood lactate ($[La^-]$), pH, and PCO_2. For $[La^-]$, blood was collected from the right earlobe (20 μl) at rest, immediately after each sprint (10 times), and 3 minutes after the completion of the last sprint set (12 times in total). The samples were analyzed using a lactate analyzer (Biosen S-Line; EFK Diagnostics, Barleben, Germany). From the contralateral earlobe, approximately 60 μl of blood was collected at rest; 15 seconds before the first, fifth, and ninth sprint sets; and immediately after the second, sixth, and last sprint sets (7 times in total) for the measurement of blood pH and PCO_2 using a blood gas analyzer (Cobas b221 System; Roche Diagnostics, Tokyo, Japan). The number of blood sampling points for blood pH and PCO_2 was limited because of the time required by the device for self-cleaning for the subsequent measurement (about 90–120 seconds). Occasionally, the measurements were unsuccessful

because of (a) unexpectedly long time taken for the self-cleaning process resulting in clotting of the pending blood sample; (b) failure to collect blood sample with sufficient amount, especially presprint blood collection; or (c) unidentified device errors.

Statistical Analyses

Statistical tests were performed using SPSS version 17.0 software (SPSS Inc., Chicago, IL, USA). The test-retest reliability of the peak and mean power outputs were evaluated using the pretest values by means of intraclass correlation (ICC), with the values being 0.972 and 0.945 (95% confidence interval, 0.916–0.991 and 0.836–0.983), respectively. Paired *t*-tests were used to compare the pretest peak and mean powers, and resting blood pH, P_{CO_2}, and [La^-] to ensure similarity in the baseline values between the 2 conditions.

The main effects of condition (control vs. hyperventilation) and time (sprint set number) and their interaction on peak and mean power outputs were studied using 2-way repeated measures analysis of variances. The same statistical method was used for $\dot{V}E$, RR, VT, and $P_{ET}CO_2$ (separate analyses for 5-second pre- and postsprint), and $\dot{V}O_2$, $\dot{V}CO_2$ and [La^-]. For those cases violating the assumption of sphericity, significance was corrected using the Huynh-Feldt adjustment. A linear mixed model analysis was applied on the blood pH and P_{CO_2} data set with occasional missing data points to test the main effects of condition and time, and their interaction.

When a significant time effect was found, pair-wise comparisons were performed where necessary using the Bonferroni method. The $p \leq 0.05$ were considered statistically significant, and all values are presented as mean ± *SD*.

Results

The pretest baseline values between the control and hyperventilation conditions were not statistically different: peak power (833 ± 119 vs. 829 ± 107 W) and mean power (743 ± 93 vs. 739 ± 92 W), resting blood pH (7.367 ± 0.027 vs. 7.366 ± 0.021), P_{CO_2} (36.2 ± 2.5 vs. 36.5 ± 2.7 mm Hg), and [La^-] (4.5 ± 1.2 vs. 4.4 ± 1.1 mmol·L^{-1}). The resting value of [La^-] exceeded onset of blood lactate accumulation (onset of blood lactate accumulation, 4.0 mmol·L^{-1}) as a result of warm-up.

Power Outputs

Peak and mean power outputs (Figure 1) showed a gradual decrease with sprint set number (peak power: $p = 0.002$; mean power: $p < 0.001$). The condition effect was not observed for peak and mean power outputs. A significant condition × time interaction seen in both variables (peak power: $p = 0.035$, mean power: $p = 0.023$) indicated that the overall decrement in power was attenuated for the hyperventilation condition compared with control. The first-to-last sprint performance decrement (control vs. hyperventilation) was respectively −6.4% vs. −3.9% for the peak power, and −8.2% vs. −5.5% for the mean power.

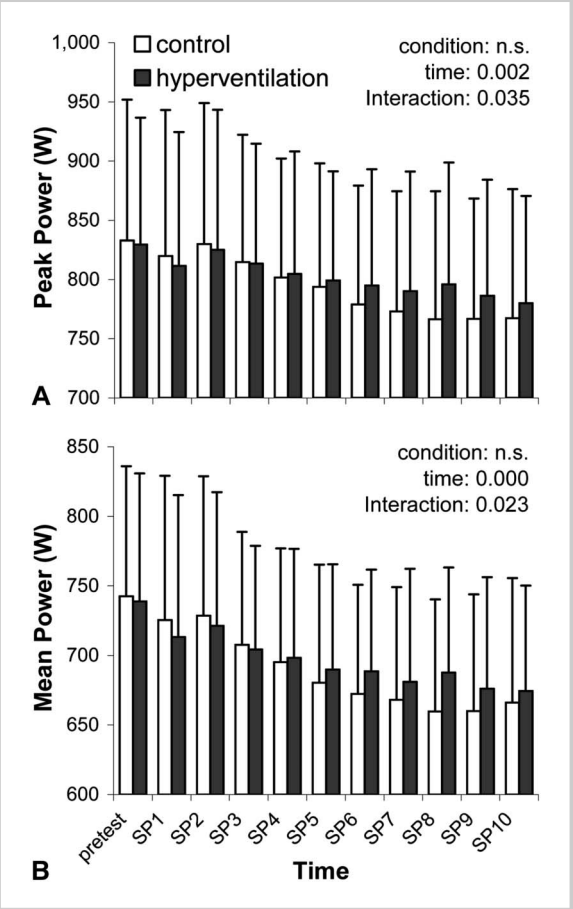

Figure 1. Peak (A) and mean (B) power outputs recorded during each sprint set ($n = 13$) for control vs. hyperventilation conditions. Pretest trial was performed after warm-up before the commencement of each experiment before exposed to the condition effect to test day-day repeatability (ICC: peak power, 0.972; mean power, 0.945). *p* values for the main effects of condition and time and their interaction are indicated in the graph when $p \leq 0.05$. n.s., non-significant where $p > 0.05$.

Blood pH, P_{CO_2}, and [La^-]. Blood samples successfully obtained for pH and P_{CO_2} analysis were 83 (91.2%) for the control and 79 (86.8%) for the hyperventilation condition of 91 (13 subjects × 7 collection time points). The missing data point occurred randomly with the number being 0–3 of 13 subjects per exercise set. Figure 2 shows a gradual fall in pH (panel A) and P_{CO_2} (panel B) as the sprint exercise progressed ($p < 0.001$). The hyperventilation intervention was successful resulting in higher blood pH ($p = 0.007$) and lower P_{CO_2} ($p < 0.001$) than the control condition (Figures 2A, B). Immediately before the first sprint set (pre-SP1), blood pH rose to 7.460 ± 0.024 for the hyperventilation condition (vs. 7.392 ± 0.032, control) and remained higher than control thereafter (Figure 2A). Significant condition × time interactions were evident for pH and P_{CO_2} ($p < 0.001$), showing that these differences between conditions were greater for the presprint than the postsprint time (Figures 2A, B). Blood

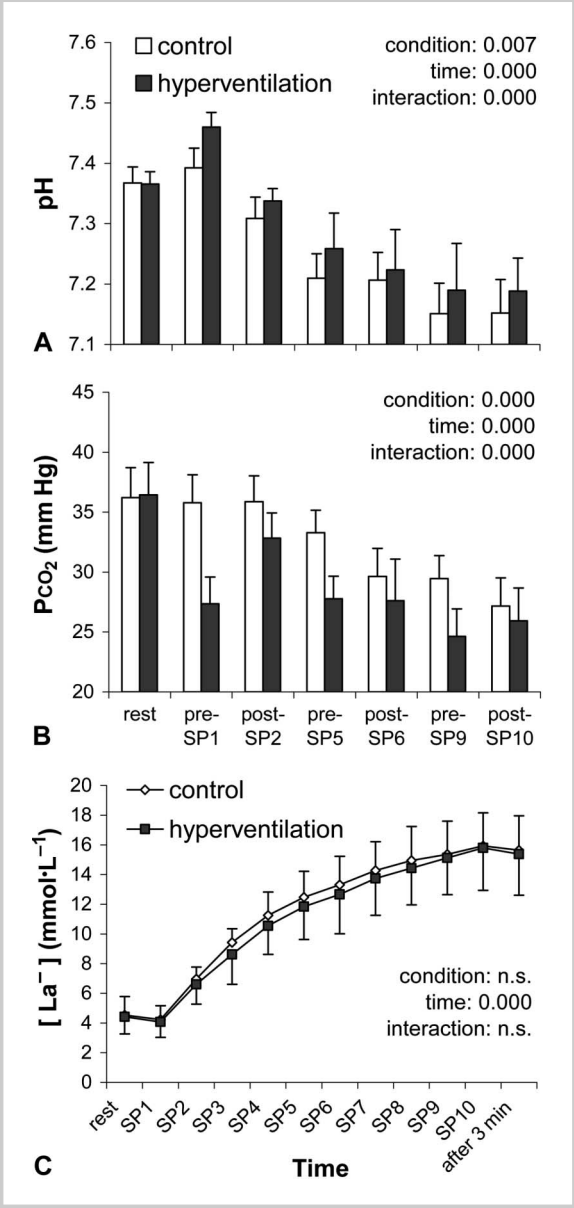

Figure 2. Blood pH (A), P_{CO_2} (B), and [La^-] (C) for control and hyperventilation conditions ($n = 13$). The pH and P_{CO_2} were measured at rest; immediately before first, fifth, and ninth sprint sets (pre-SP1, 5 and 9); and after second, sixth, and 10th sprint sets (post-SP2, 6 and 10), whereas [La^-] were measured at rest, immediately after each sprint set (SP1–SP10), and 3 minutes after the last sprint set (after 3 minutes). The p values for the main effects of condition and time, and their interaction are indicated in the graph when $p \leq 0.05$. n.s., non-significant where $p > 0.05$.

[La^-] progressively increased with sprint set number ($p < 0.001$, Figure 2C). The [La^-] was not significantly different between conditions with nonsignificant condition × time interaction (Figure 2C).

$\dot{V}E$, RR, and VT. For the hyperventilation condition, the 5-second presprint $\dot{V}E$ and RR (Figures 3A, B, left) were consistently higher ($p < 0.001$) than control. Significant time effect ($p < 0.001$) and condition × time interaction ($p < 0.001$) were observed for both the presprint $\dot{V}E$ and RR. For the presprint $\dot{V}E$, it reached a constant level after the third sprint set for the hyperventilation condition, whereas it rose steadily toward the end of exercise for the control condition (Figure 3A, left). Presprint RR were constant for the hyperventilation condition as intended (maintained close to 60 breaths per minute), whereas for the control condition, presprint RR showed a steady rise with sprint set number (Figure 3B, left). For the 5-second postsprint $\dot{V}E$ and RR (Figures 3A, B, right), the condition effect and condition × time interaction disappeared. Only the time effect remained significant ($p < 0.001$), with the values gradually increasing with sprint set number.

The presprint VT (Figure 3C, left) did not differ significantly between the 2 conditions. The time effect was significant ($p < 0.001$); however, pair-wise comparisons revealed that this effect resulted from the first few sprint sets only and the presprint TV were constant thereafter. The condition × time interaction ($p = 0.008$) was also significant, indicating that VT was more variable with time for the control condition, but again for the earlier sprint sets only (Figure 3C, left). The postsprint VT (Figure 3C, right) were slightly but significantly lower for hyperventilation ($p < 0.001$) with no condition × time interaction. The main effect of time was significant ($p < 0.001$); however, similar to the presprint VT, this effect was present only for the very early sprint sets according to the pair-wise comparisons (Figure 3C, right). The relatively consistent VT with time overall indicated that the exercise-induced rise in $\dot{V}E$ was largely accounted for by increased RR (Figures 3A–C).

End-Tidal Partial Pressure of CO_2. The presprint $P_{ET}CO_2$ (Figure 3D, left) were lower as intended for the hyperventilation condition compared with control ($p < 0.001$). Time effect and condition × time interaction were both significant ($p < 0.001$) showing that $P_{ET}CO_2$ was vastly different between conditions in earlier sprint sets but not later ones (Figure 3D, left). The condition effect and condition × time interaction both disappeared for the postsprint $P_{ET}CO_2$ (Figure 3D, right). Only the time effect remained significant, and the postsprint $P_{ET}CO_2$ steadily decreased with sprint set number (Figure 3D, right).

$\dot{V}O_2$ and $\dot{V}CO_2$. Figure 4 displays the average $\dot{V}O_2$ and $\dot{V}CO_2$ for the 30 seconds before each sprint set (intervention duration). $\dot{V}O_2$ increased sharply after the first sprint set followed by a steady rise ($p < 0.001$, Figure 4A). The condition effect was not significant; however, a significant condition × time interaction existed ($p < 0.001$), demonstrating a slightly higher $\dot{V}O_2$ with hyperventilation for the first 2 sprint sets (Figure 4A). $\dot{V}CO_2$ were higher for the hyperventilation condition ($p < 0.001$, Figure 4B). Significant time effect also existed for $\dot{V}CO_2$ ($p < 0.001$); however, pair-wise comparisons showed that this

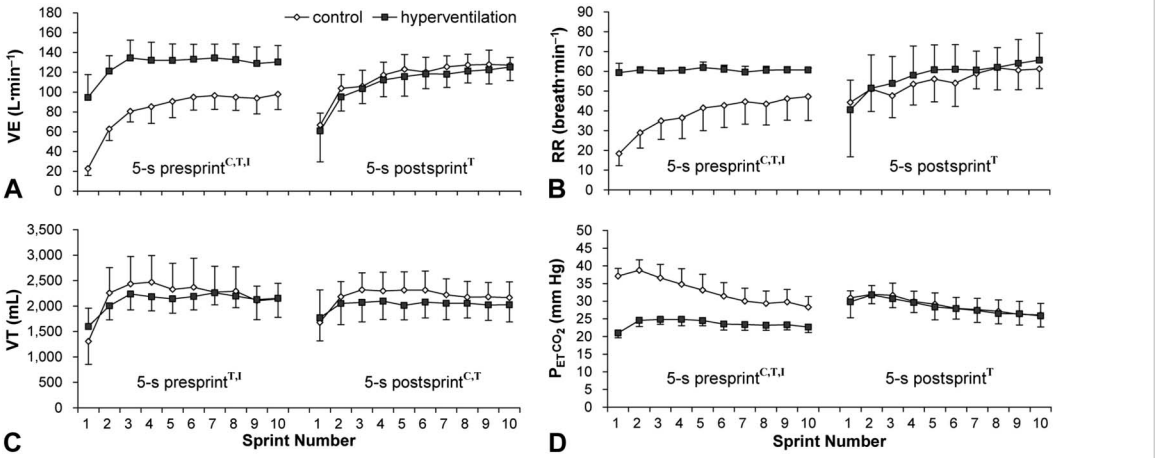

Figure 3. A) Changes in minute ventilation ($\dot{V}E$), (B) respiratory rate (RR), (C) expired tidal volume (VT), and (D) end tidal partial pressure of CO_2 ($P_{ET}CO_2$), measured by aeromonitor for control and hyperventilation conditions ($n = 13$). Breath-by-breath data were averaged for 5 seconds before (5-second presprint, left) and immediately after each sprint set (5-second postsprint, right). Two-way repeated measures analysis of variances were performed separately between the pre- and postsprint. C = significant condition effect; T = significant time effect; I = significant condition × time interaction ($p \leq 0.05$).

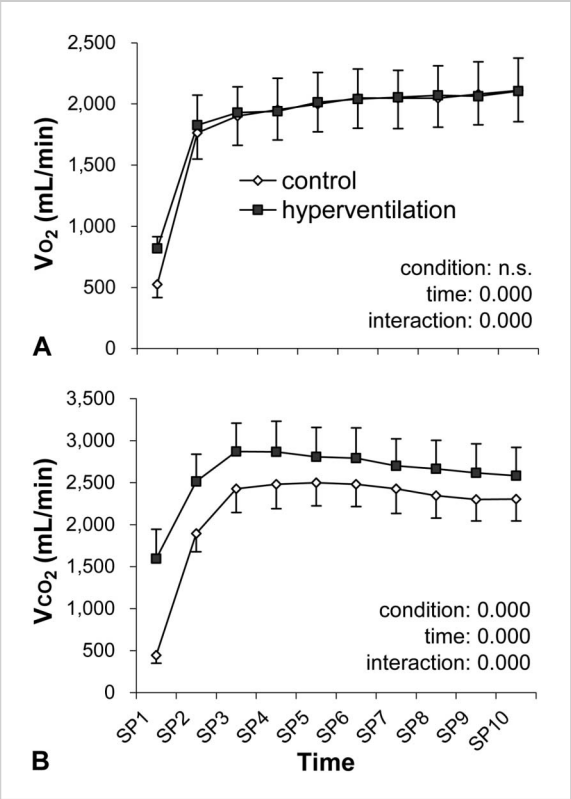

Figure 4. $\dot{V}O_2$ (A) and $\dot{V}CO_2$ (B) recorded before each sprint set for control and hyperventilation conditions ($n = 13$). Breath-by-breath data were averaged during the entire 30 seconds of intervention period before each sprint set. The p values for the main effects of condition and time, and their interaction are indicated in the graph when $p \leq 0.05$. n.s., non-significant where $p > 0.05$.

effect was mostly attributable to the early sprint sets. Condition × time interaction was also significant ($p < 0.001$) showing that the $\dot{V}CO_2$ differences were reduced in later exercise sets (Figure 4B).

Discussion

The hyperventilation method successfully attenuated performance decrements (peak power and mean power) over repeated sprint sets (Figure 1), through retarded acidosis as confirmed by a significant rise in blood pH and concomitant reduction in PCO_2 compared to the control condition (spontaneous breathing) (Figures 2A, B). The presprint RR and $P_{ET}CO_2$ were well controlled as intended for the hyperventilation condition (60 breaths per minute and maintained at 20–25 mm Hg) throughout the experiment, resulting in significantly greater $\dot{V}E$ (approximately 120–135 $L \cdot min^{-1}$, Figures 3A, B, D, left). The presprint VT were maintained at a level (approximately 2000–2500 ml) close to that achieved in the control condition to attain the target presprint $P_{ET}CO_2$ value of 25 mm Hg, despite a much higher RR (Figures 3B, C, left). The postsprint VT were found to be slightly lower for hyperventilation (Figure 3C, right) than the control condition, but this difference did not affect the postsprint $\dot{V}E$ (Figure 3A, right).

The ergogenic effects of hyperventilation may be explained by both an increased renewable fuel source and the continued supply of glycolytic energy sources in later sprint sets. With an attenuation of fall in pH, the phosphocreatine resynthesis process as well as the rate-limiting step in glycolysis (the phosphofructosekinase reaction) would be expected to be more favorable, since both reactions are dependent on intracellular pH (7,10,16,23,31,32). Furthermore,

pH attenuation may also delay the failure in the excitation/contraction process (11,13,26,28), enabling force generation to be maintained for the sprinting duration. Additionally, it is plausible that hyperventilation may serve as a diverting activity that accelerates recovery of central fatigue–a mechanism proposed as the "Setchenov phenomenon" (1,17). When volitional hyperventilation is performed during recovery, the increased activity of respiratory and trunk muscles may act as the diverting activity and may counter performance decrement associated with central fatigue through utilization of the Setchenov phenomenon.

We did not observe any "preparatory effect" of respiratory alkalosis, which if present, would have produced enhanced performance in the initial or early sprint sets. This could be because, in early exercise, phosphocreatine may not have been sufficiently depleted (22) or that there was minimal disruption to glycolytic energy supply with an initial small fall in pH (above 7.3, Figure 2A). The absence of the preparatory effect was consistent with previous results (25), demonstrating no performance change (mean power output) during a single 45-second maximal pedaling with and without performing hyperventilation before exercise. These findings imply that significant metabolic challenges and severe exercise-induced acidosis are conditions needed for eliciting ergogenic benefits of hyperventilation for performance.

Whilst systemic acidosis may be experienced during repeated sprints, marked respiratory alkalosis may be encountered within the brain. Thus, one could argue that hyperventilation may add to, rather than prevent, the development of central fatigue because respiratory alkalosis can result in reduced cerebral blood flow and reduced central motor commands (9,27). In the present study, the hyperventilation phase was however relatively short and performed intermittently with none of the subjects reporting apparent signs of reduced cerebral blood flow such as dizziness, lightheadedness, or paresthesia during the test. However, we could not deny the imposing effects of hyperventilation on performance decrement through central inhibition (27). Further studies are warranted to reveal to what extent hyperventilation may prevent or provoke factors for central fatigue during repeated intense exercise.

In the present study, blood [La^-] did not differ between the 2 conditions (Figure 2C). This finding was not expected, since sodium bicarbonate ingestion or hyperventilation results in greater [La^-] in the circulation during and/or after exercise compared to control values (5,7,18,21). Higher levels of [La^-] seen in previous studies had been explained by (a) greater lactate production resulting from sustained high rates of anaerobic glycolysis or glycogenolysis; (b) enhanced efflux of lactate either through a greater lactate gradient, or greater permeability or transporter activity for lactate; (c) reduced muscle and hepatic lactate clearance through reduced blood flow or reduced lactate reutilization via gluconeogenesis; and/or (d) reduced oxidation of pyruvate as a result of slowed activity of oxidative enzymes such as pyruvate dehydrogenase (3,5,7,8,16,25). The failure to see greater [La^-] with hyperventilation in this study could be explained by a facilitated lactate uptake or reutilization by respiratory and trunk muscles that were highly active during the hyperventilation. This suggestion may partly be supported by Chiappa et al. (4) who demonstrated an enhanced lactate clearance by increasing ventilatory work through increased inspiratory resistance.

As expected, hyperventilation increased $\dot{V}_{CO_2}$ as a result of greater CO_2 elimination than the control condition (Figure 4B). $\dot{V}_{O_2}$ were also expected to be higher for the hyperventilation condition given the greater work performed by the respiratory and trunk muscles. Indeed, the breath-by-breath raw data showed higher $\dot{V}_{O_2}$ value at the onset of every hyperventilation intervention reflecting increased respiratory work (6). However, it promptly decreased towards a value similar to the control over the remaining time in each intervention. This prompt returning in $\dot{V}_{O_2}$ may be accounted for by a left-shift of the oxyhemoglobin dissociation curve, signaling an increased hemoglobin affinity for O_2 in response to respiratory alkalosis (5,10). Alternatively, it may be explained by the reduced work of breathing resulting from bronchodilation and decreased airflow resistance with exercise activation of sympathetic drive and catecholamine secretion (6,33). These interacting effects probably yielded $\dot{V}_{O_2}$ not necessarily greater during the hyperventilation intervention despite greater activation of the respiratory and trunk muscles.

In conclusion, hyperventilation performed during recovery that separated repeated maximal sprint sets, with $P_{ET}CO_2$ maintained at 20–25 mm Hg, has an ergogenic effect on performance in highly trained athletes. The intervention achieved higher peak power and mean power outputs in later sprint sets.

Practical Applications

The observation of attenuation of power decrement suggests that using the hyperventilation strategy during a training session can facilitate sustained activity at relatively higher exercise intensity. In turn, this training strategy, incorporating hyperventilation, would be expected to enable greater adaptive changes and enhanced training efficacy. Improved performance would be expected to lead to success in sport events, when the game outcomes are appreciably influenced by repeated intensive anaerobic fitness, e.g., at the later stages of a football match or fighting sport. The present study provides the potential of hyperventilation for replacing sodium bicarbonate ingestion. However, further research is required regarding exercise modes (the type of intermittent exercise), intensity and duration, and fitness levels of athletes or sport-specific movements typically required in team sports or training. Moreover, an optimal hyperventilation method is warranted regarding the target $\dot{V}E$ (RR and VT), $P_{ET}CO_2$, duration, and/or timing. Nevertheless, hyperventilation, as a voluntary act, can be instantly initiated or

terminated at no cost, as long as dizziness and tingling are avoided. Studies assessing the efficacy, e.g., of an 8-week training program involving the hyperventilation strategy compared with one without will be valuable.

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