Enhancement on Wingate Anaerobic Test Performance With Hyperventilation

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Relatively long-lasting metabolic alkalizing procedures such as bicarbonate ingestion have potential for improving performance in long-sprint to middle-distance events. Within a few minutes, hyperventilation can induce respiratory alkalosis. However, corresponding performance effects are missing or equivocal at best. **Purpose:** To test a potential performance-enhancing effect of respiratory alkalosis in a 30-s Wingate Anaerobic Test (WAnT). **Methods:** 10 men (mean ± SD age 26.6 ± 4.9 y, height 184.4 ± 6.1 cm, body-mass test 1 80.7 ± 7.7 kg, body-mass test 2 80.4 ± 7.2 kg, peak oxygen uptake 3.95 ± 0.43 L/min) performed 2 WAnTs, 1 with and 1 without a standardized 15-min hyperventilation program pre-WAnT in randomized order separated by 1 wk. **Results:** Compared with the control condition, hyperventilation reduced (all *P* < .01) pCO2 (40.5 ± 2.8 vs 22.5 ± 1.6 mm Hg) and HCO3− (25.5 ± 1.7 vs 22.7 ± 1.6 mmol/L) and increased (all *P* < .01) pH (7.41 ± 0.01 vs 7.61 ± 0.03) and actual base excess (1.4 ± 1.4 vs 3.2 ± 1.6 mmol/L) pre-WAnT with an ergogenic effect on WAnT average power (681 ± 41 vs 714 ± 44 W) and total metabolic energy (138 ± 12 vs 144 ± 13 kJ) based on an increase in glycolytic energy (81 ± 13 vs 88 ± 13 kJ). **Conclusion:** Hyperventilation-induced respiratory alkalosis can enhance WAnT cycling sprint performance well in the magnitude of what is seen after successful bicarbonate ingestion.

**Keywords:** respiratory alkalosis, lactate, sprint

There is convincing evidence that alkalizing procedures such as bicarbonate ingestion have potential for improving performance in long-sprint to middle-distance events. The physiological background of this phenomenon is that alkalosis can increase glycolytic rate by law of mass action, enhance activity of selected glycolytic enzymes, and facilitate dephosphorylation of ATP as a proton-generating process. Equivocal findings or even a negative impact of bicarbonate ingestion on performance seem to be related to side effects. Studies discriminate between responders and nonresponders based on the incidence of bicarbonate-induced intestinal symptoms. Other potential reasons for equivocal alkalizing effects may be linked to the exercise modes, with some possibly not significantly limited by hydrogen cation accumulation. Another reason might have been a relative high within-subject variability of the ergogenic effects of bicarbonate ingestion. The prevention of intestinal symptoms requires individual, partly rather time-consuming application programs lasting up to days. However, long-lasting ingestion programs combine the increase of the bicarbonate-buffer capacity with successive combined respiratory and metabolic compensation of the bicarbonate effect. This resulted in a relative mild alkalosis and a moderate base-excess increase in the magnitude of 5 to 7 mmol/L 40 to 120 minutes postingestion. Hyperventilation can induce alkalosis more rapidly by respiratory reduction of the partial pressure of CO2 (pCO2). Based on rare and equivocal findings, it is unknown whether respiratory alkalosis has potential in terms of performance enhancement similar to that observed in successful experiments using bicarbonate for the induction of a metabolic alkalosis. In a single 45-second-sprint experiment, a 15-minute hyperventilation procedure maintaining end-tidal CO2 at about 2% reduced pCO2 to approximately 28 mm Hg and increased pH from 7.44 to 7.65. However, the latter experiment showed effects on neither blood lactate concentration (BLC) nor on performance, while exercise-induced changes in base excess were highly interrelated with corresponding changes in BLC. Another more recent experiment on repeated 10-second-sprint performance with 60-second breaks, which included hyperventilation between the 30th and 60th seconds of each break, induced decreases in pCO2 of 1.2 to 8.4 mm Hg combined with pH elevations of 0.03 to 0.07 between sprints. In spite of these minute acid-base effects, the results suggested positive hyperventilation effects on sprint performance as indicated by condition-by-time interactions for peak power and average power.

One potential reason for the still-inconclusive performance effects of respiratory alkalosis may be the previously chosen sprint durations of 10 and 45 seconds. In theory, physical-exercise modes that are significantly limited by hydrogen cation accumulation should reflect dominant reliance on anaerobic-glycolytic energy. It is possible that 45-second sprinting may generate highest post-sprint BLC levels. However, in cycling the corresponding lactate appearance rate of approximately 0.3 mmol/L is roughly 25% lower than that of 30-second sprints and 67% lower than that of 10-second sprints. During maximum sprints lasting up to 10 seconds, peak rates of adenosine-triphosphate (ATP) synthesis from phosphocreatine (PCr) and glycolysis may approach rates of 6 to 9 mmol ATP · kg dry mass−1 · s−1. These sum up to approximately 15 mmol ATP · kg dry mass−1 · s−1 with glycolysis being maximally activated not earlier than after 5 seconds. At longer-lasting all-out sprints, the glycolytic rate decreases below the rate of aerobic ATP synthesis within the subsequent 20 to 30 seconds of maximum glycolytic activation. The latter suggests that in 10-second...
sprints with approximately 96% reliance on anaerobic energy ATP synthesis, PCr reflects the dominant performance-limiting source of metabolic energy.25 In all-out 30-second sprints with an overall contribution of anaerobic energy of 70% to 84%,25 approximately 30% ATP synthesis reflects PCr hydrolysis, 50% glycolytic ATP synthesis, and 20% aerobic ATP synthesis.19,26 Consequently, in an all-out 30-second sprint the relative contribution of glycolytic ATP synthesis alone approaches a magnitude of the combined effect of PCr plus glycolysis in 45-second sprinting.25

Therefore, the aim of this study was to test the effect of a preexercise hyperventilation procedure on acid-base status and subsequent alterations of energy metabolism with a potential performance-enhancing effect in a 30-second all-out Wingate Anaerobic Test (WAnT) cycling sprint. The experiment was based on the hypothesis that anaerobic-glycolytic ATP synthesis reflects the dominant source of metabolic energy of the WAnT, and the preexercise hyperventilation procedure induces a sufficient level of respiratory alkalosis to facilitate anaerobic-glycolytic ATP synthesis and utilization, which potentially enhances WAnT performance.

**Methods**

**Subjects**

One week before the first main experiment, 10 men (mean ± SD age 26.6 ± 4.9 y, height 184.4 ± 6.1 cm, body-mass test 1 80.7 ± 7.7 kg, body-mass test 2 80.4 ± 7.2 kg, peak oxygen uptake 3.95 ± 0.43 L/min) were familiarized with all experimental procedures in an initial laboratory session. Then each subject performed 2 WAnTs, 1 with and 1 without a standardized hyperventilation program pre-WAnT in randomized order separated by 1 week. Based on pilot hyperventilation tests, conducted when establishing the hyperventilation procedure used in this experiment with differences in pH of 0.18 ± 0.04 combined with acute base excess (ABE) effects of 0.8 ± 0.5 mmol/L, an a priori power calculation revealed the necessity of a sample size of n = 6 to achieve a power of 80% at a significance level of P ≤ .05 for securing hyperventilation-induced acid-base effects against the control condition pre-WAnT. If the expected ABE effect can be converted into an increase of the BLC response based on a linear interrelationship of changes in ABE and BLC, previously seen BLC responses16-18,20 could be elevated by approximately 7% ± 4%. Assuming that WAnT metabolism reflects approximately 50% glycolytic energy,19,26 the expected gain in performance would be in the magnitude of about 3% ± 2%. The latter hypothesis would also require a performance-based sample size of n = 6. All participants were healthy and nonsmoking. None was under pharmacological or special dietetic treatment. Informed consent was obtained after explanation of the nature and risks involved in participation in the experiments, which conformed to internationally accepted policy statements regarding the use of human subjects approved by the local ethics committee.

**Procedures**

Each WAnT session started with a standardized warm-up of 5 minutes cycling at 50 W on an electromagnetically braked cycle ergometer (Excalibur Sport, Lode). The warm-up included 2 sprints lasting 3 seconds performed at the end of the third and the fourth minutes to prepare the participants for the sprint-like WAnT. The warm-up was followed by a resting period of 15 minutes with or without hyperventilation program. During this resting period, in both control and hyperventilation conditions, the subject sat on the mechanically braked cycle ergometer (834 E, Monark) that was used for the subsequent WAnTs. The 15-minute hyperventilation procedure had a set breathing frequency of 25 breaths/min (metronome) with individually adjusted tidal volume to lower the end-expiratory partial pressure of carbon dioxide (PetCO2) to approximate 20 mm Hg within 3 minutes. pCO2 was then kept at this level for the remaining 12 minutes of hyperventilation. After that the WAnT started and the subjects were instructed to accelerate the pedaling rate maximally. A resistance corresponding to 7.5% of individual body mass was applied after an acceleration phase of 3 seconds. The subjects were verbally encouraged to maintain as high a pedaling rate as possible throughout the 30-second WAnT. After test termination subjects were supervised during a 30-minute recovery in a seated position. Subsequently, they were asked whether they had experienced any episodes of lightheadedness, tunnel vision, or other feelings of orthostatic instability at any time during the complete testing procedure. All tests were performed at similar times in the morning at least 2 hours after a light breakfast. Subjects were instructed not to engage in strenuous activity during the day before an exercise test.

**Measures**

Mechanical power was measured throughout the 30-second WAnT and sampled at 5-second averages. Oxygen uptake (VO2) and carbon dioxide production were measured breath by breath with a spirometric system (Oxycon gamma, Mijnhard) during the total warm-up, testing, and recovery session. Before each testing, the device was calibrated according to the manufacturer’s instructions. The BLC was determined from 20-μL capillary blood samples drawn from the hyperemic ear lobe before the warm-up, immediately before and directly after the WAnT, minute by minute up to the tenth minute and then every second minute up to the 30th minute post-WAnT using the enzymatic amperometric method (Ebio Plus, Eppendorf). Blood gases were analyzed from 85-μL capillary blood samples drawn from the other hyperemic ear lobe also before warm-up, immediately before and directly after the WAnT, and at minutes 3, 5, 7, 9, 12, 16, 20, 25, and 30 post-WAnT, pH and pCO2 were determined using potentiometry and hemoglobin concentration ([Hb]) by spectrophotometry (ABL 700, Radiometer).

**WAnT Performance Indices**

WAnT peak and minimum power were defined as the highest and lowest mechanical power elicited from the test taken as the average power over a 5-second period. Average power is the mean power output sustained throughout the six 5-second segments. Power drop is the difference between peak power and minimum power. Fatigue index is the degree of power drop during the test expressed as percentage of peak power.27 Mechanical energy was estimated as average power times WAnT duration.

**Acid-Base Status**

Bicarbonate concentration ([HCO3-]) and ABE were calculated using pH, pCO2, and [Hb] (ABL Reference Handbook, Radiometer).

**BLC Kinetics**

The kinetics of the BLC response to the WAnT were analyzed based on a 3-parameter model20:
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BLC(t) = \frac{A \cdot k_1}{k_2 - k_1} \cdot (e^{-k_2 \cdot t} - e^{-k_1 \cdot t}) + (BLC_0 - BLC_{\text{rest}}) \cdot e^{-k_2 \cdot t} + BLC_{\text{rest}}

where A is the extravascular increase of lactate generated during sprint exercise, k_1 is the invasion constant, k_2 is the evasion constant of lactate into and out of the blood compartment, BLC_{\text{rest}} = BLC before warm-up, and BLC_0 = the corresponding value immediately pre-WAnT.

Based on an assumed water space of 60% of body mass\textsuperscript{26,28} the O_2-lactate equivalent (OLE) is \sim 3.0 mL^{-1} \cdot mmol^{-1} \cdot L^{-1} \cdot kg^{-1} \textsuperscript{26,29,30} The caloric equivalent of oxygen (CE) for a respiratory-exchange ratio above 1.0 is 21.131 J/mL.\textsuperscript{31} Consequently, knowledge of A enables one to calculate the energy derived from anaerobic glycolysis (W_{BLC}) during the WAnT:

\[ W_{BLC} = A \times OLE \times CE \]

Oxygen Uptake

The oxygen used during the WAnT was determined as the integral of all breaths detected during the 30 seconds. Post-WAnT VO_2(t) was analyzed based on a biexponential 4-parameter model:

\[ \dot{V}O_2(t) = A_{Ox} e^{-r_{Ox} t} + B_{Ox} e^{-r_{Ox} t} + \dot{V}O_20 \]

where A_{Ox} and B_{Ox} are the amplitudes of a fast and a slow component, r_{Ox} and r_{Ox} are the corresponding time constants, and \dot{V}O_20 = the asymptotic resting VO_2 at time \rightarrow \infty.

The fast component of the post-WAnT VO_2 is considered to reliably represent (r = .89)\textsuperscript{22} the amount of oxygen required for PCR resynthesis immediately postexercise.\textsuperscript{26,32,33} Therefore, integration of the corresponding term and multiplication with CE gives the anaerobic alactic energy (W_{PCr}) required for the WAnT. The aerobic energy used during the WAnT (W_{Aer}) was calculated based on the integral of the VO_2 during the WAnT and the CE. W_{Ttot} is the sum of W_{PCr}, W_{BLC}, and W_{Aer}. Biomechanical efficiency was estimated as mechanical energy of the (WAnT/W_{Ttot}) \times 100.

Statistics

Descriptive results are reported as mean ± SD. Non-linear-regression analyses were used to test whether the biexponential 3- and 4-parameter models sufficiently describe the behavior of the BLC and VO_2 over time and to determine the constants A, k_1, k_2, A_{Ox}, B_{Ox}, r_{Ox}, and k_{Bmax}. Interrelationships between selected variables were tested with linear-regression analysis. Main effects of hyperventilation versus control condition, sampling time, and interaction between the latter factors were tested using repeated-measures ANOVA with hyperventilation versus control condition as within and sampling time as between factors. Significant interactions and main effects were further analyzed using 1-way ANOVA and paired-samples t tests as appropriate. Within-subject differences of singular measures were tested using paired-samples t tests. For all statistics the significance level was set at P < .05. Effect sizes in the form of omega squared (\omega^2) were calculated where 0.01 < \omega^2 < 0.06 indicates a small, 0.06 < \omega^2 < 0.14 a moderate, and 0.14 < \omega^2 a large effect.

Results

PetCO_2

Pre-warm-up PetCO_2 was independent of control or hyperventilation (P = .724, \omega^2 = 0.000). After hyperventilation, PetCO_2 was lower immediately pre-WAnT than pre-warm-up (20.5 ± 1.2 vs 37.6 ± 2.1 mm Hg, P = .000, \omega^2 = 0.996), while no corresponding differences were found under control conditions (38.3 ± 1.8 vs 37.9 ± 2.1 mm Hg, P = .433, \omega^2 = 0.000). No subject expressed any episodes of lightheadedness, tunnel vision, or other feelings of orthostatic instability.

Acid-Base Status

pCO_2, pH, HCO_3^-, and ABE showed main effects of hyperventilation (P < .000, \omega^2 = 0.309, P = .000, \omega^2 = 0.033, P = .000, \omega^2 = 0.076 and P = .002, \omega^2 = 0.014) and sampling time (P = .000, \omega^2 = 0.725, P = .000, \omega^2 = 0.907, P = .000, \omega^2 = 0.930 and P = .000, \omega^2 = 0.929) with interaction between hyperventilation and sampling time in pCO_2 (P = .000, \omega^2 = 0.417), pH (P = .000, \omega^2 = 0.336) and ABE (P = .042, \omega^2 = 0.016). Post hoc analyses revealed that compared with the control condition, hyperventilation resulted in lower pCO_2 pre-WAnT and up to the sixteenth minute post-WAnT (all P < .05, \omega^2 = 0.291; Figure 1(A)), higher pH immediately pre-WAnT and post-WAnT (P = .000, \omega^2 = 0.976 and P = .026, \omega^2 = 0.379; Figure 1(B)), lower HCO_3^- pre-WAnT and up to the twelfth minute post-WAnT (all P < .02, \omega^2 > 0.439; Figure 1(C)), and higher ABE immediately pre-WAnT (P = .004, \omega^2 = 0.583; Figure 1(D)) combined with lower ABE between the third and sixteenth minutes post-WAnT (all P < .05, \omega^2 > 0.296; Figure 1(D)). None of these acid-base measures returned to pretest level within 30 minutes of recovery (all P < .001, \omega^2 > 0.544).

BLC and VO_2 Kinetics

The applied 3-parameter model in control and hyperventilation conditions explained 97.5% ± 1.4% and 95.6% ± 1.7% (all P < .001) of the variance of the BLC response, respectively. A was higher (P = .001, \omega^2 = 0.703) and k_1 lower (P = .033, \omega^2 = 0.349) after hyperventilation with no effect on k_2 (P = .910, \omega^2 = 0.000; Table 1), reflecting a lower BLC immediately post-WAnT (P = .026; \omega^2 = 0.463) but higher BLC values between the 4th and 28th minutes post-WAnT (all P < .05; \omega^2 > 0.256) after hyperventilation (Figure 2). Irrespective of testing condition, BLC did not return to pretest level within the 30-minute recovery period (both P < .001, \omega^2 > 0.840).

During the WAnT the used oxygen was lower (877.4 ± 179.0 vs 940.8 ± 183.9 mL, P = .003, \omega^2 = 0.608) after hyperventilation without any effect on post-WAnT VO_2 kinetics (Table 1). The explanation of the variance of the post-WAnT VO_2 by the chosen 4-parameter model response was 88.8% ± 4.4% and 84.9% ± 5.3% in control and hyperventilation conditions, respectively.

The fast component of the post-WAnT VO_2 reflected 1748 ± 243 and 1759 ± 203 mL (P = .868, \omega^2 = 0.000) and showed no difference in oxygen used for PCR replenishment in both conditions.

ΔBLC, ΔA, and ΔABE

The close interrelationship between ΔBLC and ΔABE related to BLC and ABE pre-WAnT remained unaffected (Figure 3). The hyperventilation-related change in ABE pre-WAnT (ΔABE_{CNI}) was interrelated with the corresponding effect on A (ΔA_{CNI}) (Figure 4).

Metabolic Energy

W_{Ttot} increased by 4% ± 4% or 6 ± 5 kJ (P = .007, \omega^2 = 0.528) as a result of a 7 ± 4 kJ (P = .001, \omega^2 = 0.684) increase in W_{BLC}
(A) Partial pressure of CO₂ (pCO₂), (B) pH, (C) bicarbonate concentration (HCO₃⁻), and (D) acute base excess (ABE) during the pre-WAnT period (-15 to 0 min), WAnT performance (0-0.5 min), and passive recovery (0.5-30.5 min). Black circles, control condition; gray triangles, hyperventilation pre-WAnT. Abbreviations: WAnT, Wingate Anaerobic Test; HYP, hyperventilation; CON, control.

Table 1 Kinetics of Blood Lactate Concentration and Post-WAnT Oxygen Uptake

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Hyperventilation</th>
<th>t</th>
<th>P</th>
<th>$\alpha^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A$ (mmol/L)</td>
<td>15.9 ± 1.1</td>
<td>17.3 ± 2.5</td>
<td>-4.963</td>
<td>.001</td>
<td>0.703</td>
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<tr>
<td>$k_1$ (1/min)</td>
<td>0.46 ± 0.11</td>
<td>0.39 ± 0.07</td>
<td>2.520</td>
<td>.033</td>
<td>0.349</td>
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<tr>
<td>$k_2$ (1/min)</td>
<td>$3.04 \times 10^2 ± 7.97 \times 10^3$</td>
<td>$3.06 \times 10^2 ± 7.42 \times 10^3$</td>
<td>-0.116</td>
<td>.910</td>
<td>0.000</td>
</tr>
<tr>
<td>$A_{ox}$ (mL/min)</td>
<td>2381 ± 399</td>
<td>2240 ± 696</td>
<td>1.059</td>
<td>.317</td>
<td>0.120</td>
</tr>
<tr>
<td>$\tau_{ox}$ (min)</td>
<td>0.74 ± 0.09</td>
<td>0.83 ± 0.17</td>
<td>-2.000</td>
<td>.077</td>
<td>0.231</td>
</tr>
<tr>
<td>$B_{ox}$ (mL/min)</td>
<td>737 ± 179</td>
<td>838 ± 162</td>
<td>-1.694</td>
<td>.125</td>
<td>0.158</td>
</tr>
<tr>
<td>$\tau_{sox}$ (min)</td>
<td>9.01 ± 3.25</td>
<td>7.92 ± 1.87</td>
<td>1.600</td>
<td>.144</td>
<td>0.135</td>
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Abbreviations: WAnT, Wingate Anaerobic Test; $A$, extravascular increase of lactate; $k_1$, invasion constant (blood compartment); $k_2$, evasion constant (blood compartment); $A_{ox}$, amplitude of the fast component of the post-WAnT oxygen uptake; $\tau_{ox}$, time constant of the fast component of the post-WAnT oxygen uptake; $B_{ox}$, amplitude of the slow component of the post-WAnT oxygen uptake; $\tau_{sox}$, time constant of the slow component of the post-WAnT oxygen uptake.
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Figure 2 — Blood lactate concentration (BLC) during the pre-WAnT period (-15 to 0 min), WAnT performance (0-0.5 min), and passive recovery (0.5-30.5 minute). Black circles, control condition; gray triangles, hyperventilation pre-WAnT. Abbreviations: WAnT, Wingate Anaerobic Test; HYP, hyperventilation; CON, control.

Figure 3 — Interrelationship between difference (pre-Wingate Anaerobic Test vs each recovery value) in acute base excess (ABE) and blood lactate concentration BLC (ΔBLC). Black circles, control condition; gray triangles, hyperventilation.

combined with a $1 \pm 1$-kJ ($P = .003, \omega^2 = 0.608$) decrease in $W_{\text{Aer}}$ and no significant effect on $W_{\text{PCr}}$ (Table 2).

Mechanical Power

Main effects indicated that WAnT power increased ($P = .000, \omega^2 = 0.046$) with hyperventilation and decreased ($P = .000, \omega^2 = 0.908$) over WAnT duration. The hyperventilation effect reflected power differences of 5-second segments ending at 10 ($P = .007, \omega^2 = 0.550$), 15 ($P = .033, \omega^2 = 0.347$), and 20 seconds ($P = .042, \omega^2 = 0.316$) but no effects at the initial ($P = .077, \omega^2 = 0.231$) and final two 5-second segments ($P = .05, \omega^2 = 0.290$) (Figure 5). After hyperventilation the average power increased by

5% ± 5% ($P = .017, \omega^2 = 0.430$) without any effect on the other WAnT-performance indices (Table 3).

Biomechanical Efficiency

Biomechanical efficiency remained unaffected (14.9% ± 1.1% vs 15.0% ± 1.0%; $P = .645, \omega^2 = 0.000$) by hyperventilation.

Discussion

Hyperventilation enhanced 30-second cycling sprint performance without negative side effects as have been reported previously in conjunction with a hypocapnic-related decrease in cerebral blood pressure after comparable interventions. The decrease of $p$CO₂ resulted in a corresponding increase in $p$H of approximately 0.2 (Figure 1[A, B]). This was comparable with a previous pre-long-sprint hyperventilation experiment and substantially higher than that seen after successful bicarbonate ingestion with changes in $p$H of less than 0.1. Hyperventilation reduced
CO₂ body stores by approximately 3 L, which may be seen as a kind of preliminary respiratory compensation and the possibility to store more CO₂ produced during exercise. The hyperventilation procedure went along with a decrease in HCO₃⁻ of approximately 3 mmol/L (Figure 1[C]), also as seen previously. In combination, all effects resulted in an increase in ABE slightly lower than that observed after bicarbonate ingestion of 0.2 and 0.3 g/kg.⁴¹,²⁸

After hyperventilation, average power was enhanced by 5%, or 33 W (Table 3). This increase was higher than most effects seen after successful bicarbonate ingestion. The relative increase in average power is almost identical to the relative increase in average power over Wingate Anaerobic Test (PP) (W) 975 ± 78 1003 ± 78 -1.988 .078 0.228

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The current study demonstrated that 15 minutes of hyperventilation with a target PetCO₂ close to 20 mm Hg is well tolerable and has the potential to enhance sprint performance if the energetic profile of the subsequent sprint is dominantly anaerobic-glycolytic. This reflects sprint durations of approximately 20 to not more than 30 seconds, for example, 200-m track and field or 500-m speed skating. However, at real events time tables may vary. In addition, 10-second sprints rely much more on ATP synthesis from PCr rather than glycolysis. In the present experiment, ATP synthesis from PCr remained unchanged.

**Practical Applications**

The current study demonstrated that 15 minutes of hyperventilation with a target PetCO₂ close to 20 mm Hg is well tolerable and has the potential to enhance sprint performance if the energetic profile of the subsequent sprint is dominantly anaerobic-glycolytic. This reflects sprint durations of approximately 20 to not more than 30 seconds, for example, 200-m track and field or 500-m speed skating. However, at real events time tables may vary. In addition, PetCO₂ and blood-gas control is no routine procedure under competitive field conditions. Nevertheless, the hyperventilation procedure is quite a stringent process. It uses a strict breathing routine. Like other preparatory routines it appears feasible that hyperventilation can be trained and individually adapted to each athlete's needs. Training and fine-tuning can happen under carefully controlled conditions using portable spirometric systems and blood-gas analyses during competition simulations in the field. Consequently, comparable to other features like, for example, psychoregulatory techniques or music, which are already applied concerning lactate water space, oxygen lactate equivalent, caloric equivalent, and a muscle mass of around 40% of body mass, the present findings reflect an increase of glycolytic ATP provision from approximately 5.9 to 6.6 mmol ATP · kg dry mass⁻¹ · s⁻¹. This is a substantial increase but well within physiological limits.

The glycolysis-related performance-enhancing effect in terms of ΔAccH of hyperventilation is highly interrelated with the change in ABE over pre-WAT (Figure 4). In confirmation of previous findings this did not affect the interrelationship between changes in BLC and ABE during WAT and recovery (ΔABE over ΔBLC) (Figure 3). With and without hyperventilation the regression of ΔABE over ΔBLC was almost identical to previously published results. The regression coefficient of approximately 1.5 seems to question that even during and after maximum cycling-sprint condition, changes in BLC as a predominant acid product of exercise metabolism reflect the exercise-induced added acid in the blood. However, ΔABE is an overall measure of entrance of acid into the blood. Not only entrance of lactic acid into the blood but also losses of the buffer bicarbonate to the interstitium decrease ABE. Therefore for our experimental conditions ΔABE is up to 1.5 (control) and 3.5 (hyperventilation) mmol/L larger than ΔBLC using a nomogram in Böning. A previous hyperventilation 45-second cycle-ergometer-sprint experiment did not find any performance effect. This discrepancy with our results cannot be attributed to differences in the hyperventilation procedure because they were almost identical. However, the present results showed average power effects based on performance enhancement between the fifth and twentieth seconds only. The latter suggests that the previously used 45-second-spurt test combined with a possibly insufficient statistical power of 5 subjects made it difficult to identify any positive effect of hyperventilation on performance. Also, the limited evidence of a more recent experiment suggesting positive hyperventilation effects on repeated 10-second-spurt performance appears to have suffered from the testing modality. During short recovery periods between subsequent 10-second sprints ventilation is already increased due to compensation of oxygen deficit and respiratory compensation of a sprint-induced metabolic acidosis. As observed in that experiment, effective hyperventilation (excess ventilation on top of compensatory demand) may become a challenge with only small effects on pCO₂ and pH. In addition, 10-second sprints rely much more on ATP synthesis from PCr rather than glycolysis. In the present experiment, ATP synthesis from PCr remained unchanged.

Figure 5 — Power in 5-second segments over Wingate Anaerobic Test progression. Black circles, control condition; gray triangles, hyperventilation. Abbreviations: HYP, hyperventilation; CON, control.

<table>
<thead>
<tr>
<th>Table 3 Wingate Anaerobic Test Performance Indices</th>
<th>Control</th>
<th>Hyperventilation</th>
<th>t</th>
<th>P</th>
<th>(\phi^2)</th>
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<tr>
<td>PP (W)</td>
<td>975 ± 78</td>
<td>1003 ± 78</td>
<td>-1.988</td>
<td>.078</td>
<td>0.228</td>
</tr>
<tr>
<td>MP (W)</td>
<td>496 ± 32</td>
<td>521 ± 44</td>
<td>-2.238</td>
<td>.052</td>
<td>0.286</td>
</tr>
<tr>
<td>AP (W)</td>
<td>681 ± 41</td>
<td>714 ± 44</td>
<td>-2.928</td>
<td>.017</td>
<td>0.430</td>
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<tr>
<td>PD (W)</td>
<td>479 ± 90</td>
<td>482 ± 85</td>
<td>-0.304</td>
<td>.768</td>
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</tbody>
</table>

Abbreviations: PP, peak power; MP, minimum power; AP, average power; PD, power drop; FI, fatigue index.
in competitive settings, hyperventilation may also have potential as an ergogenic preparatory routine for sprinters.

Conclusions
Hyperventilation-induced respiratory alkalosis had a performance-enhancing effect in a 30-second all-out WAnT cycling sprint. Such effects require that a main performance-limiting factor be the rate of ATP generation via glycolysis. Under these conditions the effect is well in the magnitude of possibly optimized bicarbonate ingestion. Hyperventilation induced an approximately 3-L reduction of the CO₂ stores of the body, which corresponds to approximately 130 mmol of acid. In addition it may offer a short-term functional facilitation of the respiratory component of buffering during exercise itself.

References


