Bicarbonate attenuates arterial desaturation during maximal exercise in humans

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Bicarbonate attenuates arterial desaturation during maximal exercise in humans. J Appl Physiol 93: 724–731, 2002; 10.1152/japplphysiol.00398.2000.—The contribution of pH to exercise-induced arterial O2 desaturation was established in a pilot study. The main study evaluated the influence of acidosis on SaO2 during maximal exercise in humans has not been determined. We evaluated the influence of bicarbonate on SaO2 during maximal rowing, which is associated with a marked reduction in both PaO2 and SaO2 (22, 40, 41, 47). First, the blood temperature response to maximal rowing and the dose of bicarbonate that would attenuate acidosis were established in pilot studies. The main study evaluated the effect of a high dose of bicarbonate on arterial blood-gas variables, pulmonary gas exchange, and changes in muscle oxygenation as determined by near-infrared spectrophotometry (NIRS).

METHODS

Five competitive oarsmen (Table 1) participated in the study after informed consent as approved by the Ethics Committee of Copenhagen (KF 01-280/98). No subject had any disease or injury in the 3 wk before the experiment, and they were not taking any medication. The subjects were not allowed to eat or to drink after midnight on the day of the experiment, which began at 0800.

Exercise was performed on a rowing ergometer (model C; Concept II, Morrisville, VT). First, the subjects rowed for 12 min at work rates increasing from 150 to 250 W in steps of 50 W every third minute (warm-up). Thereafter they rowed for 5

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the end of maximal rowing (Fig. 1).

bicarbonate that would attenuate acidosis during maximal increased from the onset of exercise to reach 38.9 row and for the temperature is presented as the mean over the last minute of through the basilic vein of the nondominant arm. The tem-

P

1% (41). An all-out ergometer rowing protocol elicits a maximal O₂ uptake (V\textsubscript{O₂ max}) similar to that attained during cycling (13).

In the first pilot study (n = 6), temperature in the superior vena cava was recorded with a 132F5 catheter inserted through the basilic vein of the nondominant arm. The temperature is presented as the mean over the last minute of each submaximal work rate, every 15 s during the maximal row and for the first 5 min of the recovery. The temperature increased from the onset of exercise to reach 38.9 ± 0.1°C at the end of maximal rowing (Fig. 1).

The second pilot study (n = 8) established the dose of bicarbonate that would attenuate acidosis during maximal rowing. Subjects received in a random double-blind fashion either sodium bicarbonate (1 M; 100–325 ml; Table 1) or an equal volume of isotonic saline in a crossover study design with 7 days between the two trials. However, infusion of sodium bicarbonate increased the concentration of sodium in blood to a higher level than with isotonic saline, which might affect plasma volume and in turn O₂ transport.

In the main study (n = 5), we aimed at a dose of sodium bicarbonate that was expected to eliminate acidosis during maximal exercise. Furthermore, with administration of an equal volume of 1 M saline in the placebo condition, we expected that the level of blood sodium, and in turn plasma volume, would be the same in the two trials. Treatments were applied by randomization in a double-blind fashion in a crossover study design with 7 days between trials. One subject received only 200 ml of sodium bicarbonate because of catheter failure and, accordingly, also 200 ml of saline. In both trials, the subjects experienced headache in the first minute of the recovery.

A catheter (1.0 mm, 20 gauge) was inserted in the radial artery of the nondominant arm. Infusions of bicarbonate or saline were administered through a central catheter (1.7 mm, 16 gauge) inserted in an upper arm vein. The total dose of sodium bicarbonate or saline to be infused was divided into 60-ml syringes emptied at a constant rate (4042E, SIMS) at rest and at the end of each submaximal intensity during the warm-up, every 1 minute during the maximal row, and at minutes 1, 2, 3, 4, 5, 10, 20, and 30 of the recovery. Samples were kept on ice and analyzed for blood-gas variables, Hb, glucose, sodium, calcium, potassium, and lactate in plasma by use of an ABL 615 apparatus (Radiometer, Copenhagen, Denmark) with cooximetry for determination of SaO\textsubscript{2}. Blood gases were corrected to the average blood temperature established for each time point (Fig. 1). For the subjects who participated in the temperature pilot study, the individual temperature change was used. Thus we did not take into account the extent to which infusion of sodium bicarbonate or saline would affect blood temperature. However, even with the unlikely assumption that the effect is limited to blood only, the decrease in blood temperature is estimated to be ≤0.1°C. The O₂ content in arterial blood was calculated as the sum of bound and dissolved O₂.

### Table 1. Individual variables for subjects participating in pilot studies and in the definitive study with indication of the dose of NaHCO\textsubscript{3} and saline used in the trials

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>H, cm</th>
<th>W, kg</th>
<th>V\textsubscript{O₂ max}, l/min</th>
<th>Tp study</th>
<th>NaHCO\textsubscript{3} (1 mM)</th>
<th>NaHCO\textsubscript{3} (1 mM)</th>
<th>NaCl (1 mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20</td>
<td>177</td>
<td>77</td>
<td>4.5</td>
<td>x</td>
<td>240</td>
<td>350</td>
<td>350</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>185</td>
<td>77</td>
<td>5.7</td>
<td>x</td>
<td>300</td>
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<td>350</td>
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<tr>
<td>3</td>
<td>28</td>
<td>189</td>
<td>78</td>
<td>5.4</td>
<td>x</td>
<td>300</td>
<td>350</td>
<td>350</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>183</td>
<td>80</td>
<td>5.5</td>
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<td>325</td>
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<td>200</td>
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<tr>
<td>5</td>
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<td>183</td>
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<td>170</td>
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<td>184</td>
<td>79</td>
<td>5.6</td>
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<td>200</td>
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<td>9</td>
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<td>191</td>
<td>88</td>
<td>5.9</td>
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<tr>
<td>10</td>
<td>23</td>
<td>187</td>
<td>75</td>
<td>5.2</td>
<td>x</td>
<td>200</td>
<td>350</td>
<td>350</td>
</tr>
<tr>
<td>11</td>
<td>35</td>
<td>182</td>
<td>94</td>
<td>4.1</td>
<td>x</td>
<td>330</td>
<td>330</td>
<td>330</td>
</tr>
</tbody>
</table>

H, height; Tp, temperature trial; V\textsubscript{O₂ max}, maximal O₂ uptake record for each subject; W, weight; x, subject participated in the pilot study in which temperature changes to rowing were evaluated. A dose of sodium bicarbonate (NaHCO\textsubscript{3}) indicates that this subject was included in the pilot study with infusion of bicarbonate and/or participation in the definitive study.

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**Fig. 1.** Blood temperature (vena caval) at rest, during a warm-up (W-u) at 150, 200, and 250 W, and before and during maximal ergometer rowing and 30 min into the recovery (n = 6). Values are means ± SE with the upper and lower range. ● Different from rest, P < 0.05.
Ventilatory variables were determined by use of an integrated system (MedGraphics 2001; Medical Graphics, St. Paul, MN) with values stored on a hard disk. The O₂ was determined electrochemically, and CO₂ was evaluated by an infrared analyzer. O₂ uptake (V̇O₂), ventilation (V̇E), respiratory frequency, tidal volume (V̇T), expired CO₂ (V̇CO₂), and end-tidal partial pressures for O₂ (PETO₂) and CO₂ (PETCO₂) were averaged for every 15 s and presented as the mean for the last minute of exercise. The mixed expired CO₂ fraction was also measured for calculation of the mixed expired CO₂ pressure whereby the dead space ratio (V̇D/V̇T) was estimated by ANOVA for repeated measures and by two-tail Student's t-test. A 95% confidence limit (P < 0.05).

The concentration changes of deoxygenated (ΔHb) and oxygenated Hb (ΔHbO₂) of the right vastus lateralis muscle were assessed by NIRS (3, 7, 9, 32). A continuous-wave photometer was used (NIRO500; Hamamatsu Phototonics, Hamamatsu, Japan) with light transmitted via a fiberoptic cable and reflected light delivered via a second cable to a photomultiplier operating at four wavelengths. From the measured optical densities, the chromophor concentration change in microliter per liter tissue was calculated by the measured optical densities, the chromophor concentration change in microliter per liter tissue was calculated by using computer software (ONMAIN; Hamamatsu). The algorithm is based on a modified Lambert Beer’s law: A = α·c·d·B + G, where A is the measured attenuation in optical density, α is the specific extinction coefficient of the absorbing compound (μM/cm), c is the concentration of the absorbing compound (μM), d is the distance between the optodes on the skin surface (4 cm), B is the differential pathlength factor, and G is a factor introduced to account for scattering of light in the tissue. The differential pathlength factor adopted for the leg was 4.94 (16). Data were obtained over 5 s, and variables are presented as the average during the last minute of maximal rowing. Individual and intramuscular variations in B influence the estimate of ΔHb and ΔHbO₂. However, changes were evaluated over time with each subject being his own control. Movement associated disturbances of the NIRS signal over the leg were considered to be the same in the two trials.

Data are presented as means ± SE. Results were evaluated by ANOVA for repeated measures and by two-tailed Student’s t-test for paired data. Statistical significance was set at the 95% confidence limit (P < 0.05).

RESULTS

The race time was faster and the expressed perceived exertion was lower with sodium bicarbonate than in response to saline exercise (median of 6 min 21 s (range 6 min 16 s to 6 min 58 s) vs. 6 min 28 s (6 min 23 s to 7 min 10 s) and median of 18 (range 13–19) vs. 19 (17–19), respectively; P < 0.05).

Lactate, pH, and bicarbonate. During the warm-up, the concentration of lactate in arterial blood remained below 5 mM and then increased progressively during maximal exercise with saline (Fig. 2). The level of lactate remained high in the first minutes of the recovery but then decreased toward the preexercise level in both the sodium bicarbonate and saline trials. However, in the sodium bicarbonate trial the concentration of lactate was higher from the fourth minute of exercise and throughout the 30 min of the recovery compared with the saline trial. Blood lactate increased by 10 ± 2 mM in the sodium bicarbonate compared with saline trial, and in one subject during the sodium bicarbonate trial the concentration of lactate reached 32 mM.

The concentration of blood bicarbonate was not significantly affected during the warm-up, but it became markedly reduced during maximal exercise. A further reduction was seen in the first minutes of the recovery. Thereafter, blood bicarbonate increased but remained below the preexercise level. With the infusion of sodium bicarbonate, blood bicarbonate was reduced only at the third minute of exercise and therefore remained higher than in the control trial (Fig. 2).

In response to exercise, pH decreased to reach the lowest level in the last minute of maximal exercise. After exercise, pH recovered but remained below the resting level. With infusion of sodium bicarbonate, pH was not significantly reduced and it remained higher than in the control trial.

Blood-gas variables. The PaO₂ decreased only at the last incremental stage of the warm-up, whereas SaO₂ was not significantly changed from the resting level (Fig. 2). PaO₂ was reduced from the onset of maximal exercise, and SaO₂ decreased progressively to reach a lowest value in the last minute. Resaturation was established within the first minute of the recovery. During exercise, the sodium bicarbonate trial did not significantly affect the reduction in PaO₂ compared with saline. In contrast, SaO₂ was improved during sodium bicarbonate-supplemented exercise.

During the saline trial, PaCO₂ decreased only in first minute of exercise (Fig. 2). In the last minutes of rowing with sodium bicarbonate, PaCO₂ was higher than during exercise with saline. The concentration of Hb was not changed in response to maximal exercise (Table 2). Thus CaO₂ decreased to the same extent in both trials.

The concentration of glucose and potassium increased during rowing with no significant effect of sodium bicarbonate (Table 2). In contrast, the plasma calcium concentration increased only during exercise with saline. During exercise, the increase in sodium increased to the same extent in both trials.

NIRS. Muscle oxygenation remained stable at rest, but from the onset of maximal exercise, ΔHb and ΔHbO₂ increased and decreased, respectively (Fig. 3, Table 2). These exercise-induced changes in muscle oxygenation were not significantly affected by sodium bicarbonate.

Ventilation and heart rate. Heart rate increased similarly during maximal exercise with saline and with sodium bicarbonate (Table 3). During control exercise, respiratory frequency, V̇T, V̇E, V̇CO₂, VO₂, PETO₂, and the respiratory exchange ratio increased. The PETO₂-PaO₂ difference was widened, whereas PETCO₂ decreased. Furthermore, V̇O₂/V̇T decreased in response to maximal exercise with saline (from 0.39 ± 0.03 at rest to 0.20 ± 0.02; P < 0.05).

During exercise with sodium bicarbonate, PETCO₂ increased to above the level established during exer-
cise with saline (Table 3). \( \dot{V} \text{CO}_2 \) was also higher with sodium bicarbonate than during exercise with saline. Because \( \dot{V} \text{O}_2 \) was not significantly affected by sodium bicarbonate, the respiratory exchange ratio increased. Exercise-induced change in \( \text{PETO}_2 \) and the \( \text{PETO}_2-\text{PaO}_2 \) difference were not significantly affected by sodium bicarbonate. In contrast, \( VE \) reached a lower level during exercise with bicarbonate, whereas the increased \( Vt \) and respiratory frequency during exercise were not affected by bicarbonate.

**Table 2. Blood variables and near-infrared spectrophotometry determined changes in muscle oxygenation during maximal ergometer rowing with infusion of saline or NaHCO₃**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Saline (1 mM)</th>
<th>Exercise (1 mM)</th>
<th>NaHCO₃ (1 mM)</th>
<th>Exercise (1 mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin, mmol/l</td>
<td>Rest 9.0 ± 0.3 Exercise 8.7 ± 0.2</td>
<td>Rest 8.9 ± 0.1 Exercise 9.0 ± 0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>Rest 44.2 ± 1.5 Exercise 43.1 ± 0.7</td>
<td>Rest 44.9 ± 1.1 Exercise 43.7 ± 1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \text{CaO}_2 ), ml/l</td>
<td>Rest 199 ± 6 Exercise 187 ± 4*</td>
<td>Rest 197 ± 3 Exercise 192 ± 3*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate, mmol/l</td>
<td>Rest 1.0 ± 0.2 Exercise 16.2 ± 1.2*</td>
<td>Rest 0.9 ± 0.1 Exercise 25.7 ± 2.1†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>Rest 5.5 ± 0.2 Exercise 5.8 ± 0.4*</td>
<td>Rest 5.3 ± 0.15 Exercise 5.8 ± 0.2*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potassium, mmol/l</td>
<td>Rest 4.0 ± 0.2 Exercise 6.2 ± 0.3*</td>
<td>Rest 4.4 ± 0.2 Exercise 6.6 ± 0.3*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium, mmol/l</td>
<td>Rest 139 ± 0.5 Exercise 156 ± 1.5*</td>
<td>Rest 138 ± 0.4 Exercise 153 ± 1.6*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium, mmol/l</td>
<td>Rest 1.22 ± 0.02 Exercise 1.28 ± 0.03*</td>
<td>Rest 1.26 ± 0.02 Exercise 1.06 ± 0.04†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \Delta \text{Hb} ), ( \mu \text{mol/l} )</td>
<td>Rest −0.2 ± 0.4 Exercise 21.7 ± 4.1*</td>
<td>Rest 0.1 ± 0.1 Exercise 21.7 ± 5.4*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \Delta \text{HbO}_2 ), ( \mu \text{mol/l} )</td>
<td>Rest 0.6 ± 0.2</td>
<td>Rest −13.1 ± 3.3*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SE for variables obtained in the last minute of maximal exercise (\( n = 5 \)). \( \text{CaO}_2 \), concentration of oxygen in arterial blood; \( \Delta \text{Hb} \) and \( \Delta \text{HbO}_2 \), concentration changes of deoxygenated and oxygenated hemoglobin (per liter tissue) respectively in the vastus lateralis muscle. *Significantly different from rest; †Significantly different from control (\( P < 0.05 \)).
The Vd/Vt was also reduced during exercise with sodium bicarbonate (0.33 ± 0.04 at rest vs. 0.12 ± 0.03 during exercise; P < 0.05), and this reduction tended to be even lower than during exercise with saline (P = 0.09).

**DISCUSSION**

During maximal exercise, bicarbonate infusion attenuated acidosis whereby SaO2 increased, supporting the theory that the Bohr effect contributes to exercise-induced arterial desaturation when PaO2 is low. The increase in SaO2 during bicarbonate-supplemented exercise did not affect pulmonary VO2 or changes in muscle oxygenation. Infusion of sodium bicarbonate did result in a small improvement in performance, a lowered pulmonary ventilation, and a marked increase in blood lactate.

**Blood-gas variables.** The exercise-induced reduction in SaO2 can be attributed to factors that are known to influence the O2 dissociation curve. In particular, PaO2 is critical for SaO2 in the case in which acidosis develops secondary to the pronounced accumulation of lactic acid in blood, and there is limited availability of blood bicarbonate. On the other hand, during exercise with sodium bicarbonate, the concentration of blood bicarbonate was maintained close to the resting level, i.e., there was enough blood bicarbonate to eliminate excess hydrogen ions. In this case, SaO2 was little affected by the decrease in PaO2.

Another factor that influences the Hb affinity to O2 is temperature, which should be considered in the calculation of SaO2. In the present study, SaO2 is independent of small changes in temperature. Thus the observed desaturation in our study is comparable with data from previous studies evaluating hypoxemia during maximal rowing, in which the results were reported at 37°C (22, 40, 41). In addition to SaO2, the increase in blood temperature affects the partial pressure of blood gases. The present study found that PaO2 is higher than that found during other rowing studies (22, 40, 41). This could be because the other studies did not correct for hyperthermia in the analysis of their blood gases.

The use of an average temperature rather than individual temperature changes may have introduced a certain degree of inaccuracy in the assessment of PaO2. However, rowing-induced hyperthermia appears to be a consistent finding among subjects (Fig. 1). Erythrocyte 2,3-diphospho-D-glycerate can also affect SaO2. Although such an effect was not evaluated in the present study, the 2,3-diphospho-D-glycerate concentration appears to be unchanged during maximal exercise (52, 54).

**Table 3. Heart rate and ventilatory variables in response to a 2,000-m maximal ergometer row with infusion of saline or NaHCO3**

<table>
<thead>
<tr>
<th></th>
<th>Saline (1 mM)</th>
<th>NaHCO3 (1 mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>51 ± 2</td>
<td>177 ± 4*</td>
</tr>
<tr>
<td>VT, l/min</td>
<td>9 ± 1</td>
<td>155 ± 14*</td>
</tr>
<tr>
<td>Res, breaths/min</td>
<td>15 ± 2</td>
<td>67 ± 6*</td>
</tr>
<tr>
<td>VT, l/min</td>
<td>0.62 ± 0.05</td>
<td>2.31 ± 0.18a</td>
</tr>
<tr>
<td>PETO2, Torr</td>
<td>104 ± 2</td>
<td>115 ± 2a</td>
</tr>
<tr>
<td>PET-aO2, Torr</td>
<td>1.9 ± 0.6</td>
<td>26.6 ± 1.2a</td>
</tr>
<tr>
<td>VO2, l/min</td>
<td>0.4 ± 0.06</td>
<td>4.8 ± 0.3a</td>
</tr>
<tr>
<td>PETCO2, mmol/l</td>
<td>39.8 ± 1.0</td>
<td>36.7 ± 2a</td>
</tr>
<tr>
<td>VCO2, l/min</td>
<td>0.3 ± 0.04</td>
<td>5.5 ± 0.3a</td>
</tr>
<tr>
<td>RER</td>
<td>0.85 ± 0.03</td>
<td>1.17 ± 0.02a</td>
</tr>
</tbody>
</table>

Values are mean ± SE at rest and during the last minute of exercise (n = 5). PETo2 and PETaO2, end-tidal partial pressures of O2 and CO2; PET-aO2, the difference between PETO2 and arterial O2 pressure; RER, respiratory exchange ratio; Res, respiratory frequency; VT, pulmonary ventilation; VCO2, expired CO2; VO2, pulmonary O2 uptake. Values at rest refer to the samples obtained on the day of the trial. *Significantly different from rest; †Significantly different from control (P < 0.05).
tended to be lower in the bicarbonate trial. This can explain why the widened PETO2-PaO2 difference also tended to be lower during exercise with sodium bicarbonate than with saline. Nevertheless, these trends did not reach statistical significance. In the view of the reduced V̇E, the reason that sodium bicarbonate did not change PaO2 may relate to reduced V̇O2 ventilation.

Lactate. Extracellular acidosis may depress muscle contraction (30) and muscle glycogen utilization (53) and provoke fatigue (10). In fact, preexercise administration of bicarbonate increases the torque in an isometric contraction (56). Furthermore, an increase in extracellular bicarbonate leads to a higher efflux of protons (increased lactate output) from skeletal muscle (25, 38).

The release of lactate from cells is a pH-sensitive lactate-proton translocation in which a low external pH supports its release from the muscle (33) and its uptake by, e.g., erythrocytes, kidney, liver, muscle, and the brain (28). However, muscle lactate release involves not only the lactate-proton transport but also diffusion via bicarbonate-chloride exchange and via sodium-hydrogen exchange (28). Transmembrane transport of bicarbonate also occurs via a sodium-dependent bicarbonate cotransport (58). This could be important for an increase in intracellular bicarbonate when sodium bicarbonate induces an excess extracellular buffer capacity. The result would be an increase in net release of lactate supported by the finding that intracellular pH may increase during exercise after ingestion of bicarbonate (12).

NIRS. NIRS is used to evaluate muscle O2 extraction during muscular activity (3, 4, 7, 31, 42), and maximal rowing affects light absorption related to an increase in muscle HbO2 and a decrease in oxygenated Hb (41). In the present study, NIRS evaluated whether an increase in SaO2 affected the level of muscle oxygenation and in turn muscle O2 delivery. However, although small changes in SaO2 result in increased cerebral oxygenation (18, 41), muscle oxygenation did not appear to be affected by the increased SaO2 during exercise with bicarbonate infusion. In support, with a 10% increase in CaO2 when hyperoxia restores arterial desaturation during rowing (40), the NIRS-determined muscle oxygenation was not different from that during exercise in normoxia (41). These data indicate that there is no significant effect of exercise-induced hypoxemia on muscle O2 delivery.

The use of NIRS is based on the assumption that the small area of muscle evaluated reflects changes in the whole muscle and that motion-induced changes in the scattering of light were similar in the two trials. Furthermore, changes in skin blood flow are not considered to affect the muscle NIRS recordings (32). Another important bias is that changes in HbO2 status may not be distinguished from that of myoglobin (55).

The present data do not suggest that the increase in performance with the infusion of sodium bicarbonate is explained by an increase in O2 extraction or in V̇O2. The effect of sodium bicarbonate on ionic calcium reflects the binding to plasma proteins (59) and may not affect

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The mechanisms proposed to reduce PaO2 during exercise (15) were not evaluated separately in the present paper. We did observe marked variations in PaCO2 among the subjects during the all-out row (Fig. 4). In one subject, PaCO2 was close to 30 Torr, whereas hypoventilation was manifest in others with PaCO2 at, or above, 40 Torr during exercise with infusion of saline. Furthermore, as in previous studies (40, 41), the PETO2-PaO2 difference increased during maximal rowing, indicating ventilation-perfusion inequality and diffusion limitation.

Maximal rowing elicits a cardiac output in excess of 30 l/min (41), and the subject with the lowest PaO2 also had the lowest PaCO2, suggesting a pulmonary limitation to O2 transport rather than insufficient breathing in this individual. Thus hypoventilation, ventilation-perfusion mismatch, and a fast blood transit time may all have contributed to reduce PaO2 during maximal rowing.

Ventilation. Breathing appears entrained to the rowing rhythm (31). Both peripheral and central factors influence the control of ventilation during exercise. In the present study, potassium increased to the same extent in the two trials, whereas sodium bicarbonate increased SaO2 and pH. Oren et al. (43) found that bicarbonate treatment also tended to reduce the increase in V̇E during cycling. In that study, hyperoxia slowed the ventilatory kinetics to a greater degree during acidosis than during control or alkalosis, indicating the influence of carotid bodies on respiration. Previously, our laboratory demonstrated that hyperoxia does not affect the ventilatory response to maximal rowing (40, 41). Thus, in the case of maximal rowing, a likely explanation for the reduction in V̇E with bicarbonate infusion is that a central influence of excess H+ concentration on respiration is attenuated.

With the lowered ventilatory response during exercise with sodium bicarbonate, we expected a decrease in PaO2. From Table 3 it appears that the average PETO2
muscle contraction. The reduced \( V_{\text{O}_2} \) with sodium bicarbonate could affect the level of respiratory muscle work of consequence for work capacity (24). It is also considered that the small changes in performance and perceived exertion may reflect that fatigue is related to intracellular pH (28).

We conclude that infusion of sodium bicarbonate attenuates the rise in pulmonary ventilation during maximal exercise, supporting a role of pH for ventilatory control. An enhanced Hb affinity to O\(_2\) binding did not appear to impede muscle O\(_2\) extraction. Rather, performance increased with an even higher concentration of blood lactate. During maximal exercise with a marked reduction in the arterial O\(_2\) pressure, a reduction in pH affects the arterial O\(_2\) saturation of Hb.

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