The influence of priming exercise on oxygen uptake, cardiac output, and muscle oxygenation kinetics during very heavy-intensity exercise in 9- to 13-yr-old boys

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Barker AR, Jones AM, Armstrong N. The influence of priming exercise on oxygen uptake, cardiac output, and muscle oxygenation kinetics during very heavy-intensity exercise in 9- to 13-yr-old boys. J Appl Physiol 109: 491–500, 2010. First published June 17, 2010; doi:10.1152/japplphysiol.00139.2010.—The present study examined the effect of priming exercise on O2 uptake (VO2) kinetics during subsequent very heavy exercise in 9- to 13-yr-old boys. We hypothesised that priming exercise would 1) elevate muscle O2 delivery prior to the subsequent bout of very heavy exercise, 2) have no effect on the phase II VO2 τ, 3) elevate the phase II VO2 total amplitude, and 4) reduce the magnitude of the VO2 slow component. Each participant completed repeat 6-min bouts of very heavy-intensity cycling exercise separated by 6 min of light pedaling. During the tests VO2, muscle oxygenation (near infrared spectroscopy), and cardiac output (Q) (thoracic impedance) were determined. Priming exercise increased baseline muscle oxygenation and elevated Q at baseline and throughout the second exercise bout. The phase II VO2 τ was not altered by priming exercise (bout 1: 22 ± 7 s vs. bout 2: 20 ± 4 s; P = 0.30). However, the time constant describing the entire VO2 response from start to end of exercise was accelerated (bout 1: 43 ± 8 s vs. bout 2: 36 ± 5 s; P = 0.002) due to an increased total phase II VO2 amplitude (bout 1: 1.73 ± 0.33 l/min vs. bout 2: 1.80 ± 0.59 l/min; P = 0.002) and a reduced VO2 slow component amplitude (bout 1: 0.18 ± 0.08 l/min vs. bout 2: 0.12 ± 0.09 l/min; P = 0.048). These results suggest that phase II VO2 kinetics in young boys is principally limited by intrinsic muscle metabolic factors, whereas the VO2 total phase II and slow component amplitudes may be O2 delivery sensitive.

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The adjustment of pulmonary O2 uptake (VO2) at the onset of exercise has been demonstrated to reflect changes in muscle O2 consumption (29) and the breakdown of muscle phosphocreatine, a metabolite which has been implicated in the control of oxidative phosphorylation (4, 41). Consequently, studying the VO2 kinetic response at exercise onset may provide a valuable noninvasive insight into the factors that regulate and/or limit muscle oxidative metabolism.

Oxidative metabolism; children; warmup; near-infrared spectroscopy; oxygen delivery
METHODS

Participants

Eight 9- to 13-yr-old males volunteered to take part in the study. The participants’ mean age, body mass, and stature were 10.9 ± 1.6 yr, 1.48 ± 0.16 m, and 38.5 ± 10.6 kg, respectively. Estimated mean age from peak height velocity was −2.1 ± 1.6 yr (range: 0.6 to −3.8 years) (35). All participants and their parent(s)/guardian(s) provided informed assent and consent to partake in the project, which was approved by the institutional ethics committee at the University of Exeter. The participants were healthy, recreationally active, and showed no contraindications to exercise to exhaustion.

Experimental Protocol

Participants visited the laboratory on four separate occasions over a 2-wk period, with ≥24 h rest provided between each visit. All participants arrived at the laboratory in a rested state (no intense exercise in the preceding 24 h) and were requested to refrain from food and caffeine ≥2 h prior to testing. The first laboratory session consisted of basic anthropometrical measurements (body mass and stature) and a ramp exercise test to exhaustion to determine their \( \dot{V}O_2 \)max and GET. During each of the subsequent visits, the participants completed repeat square-wave exercise bouts, where two very heavy-intensity exercise bouts were separated by 6 min of light exercise. All tests were performed on an electronically braked cycle ergometer (Lode, Groningen, The Netherlands), with appropriate adjustments made to the ergometer seat, handlebar, and pedal cranks for each participant.

Ramp exercise. Following a 3-min period of cycling at 10 W, a ramp incremental test to exhaustion was undertaken, whereby power output increased at a rate of 10–20 W/min. These ramp rates resulted in a test duration of 742 ± 62 s. Participants cycled at a cadence of ~75–80 rpm throughout the test, and exhaustion was defined as a drop in cadence below 60 rpm for 5 consecutive seconds. Immediately following exhaustion, power output was lowered to 10 W, and the participant cycled at this intensity for 10 min followed by 5 min of rest. To verify the measurement of a valid \( \dot{V}O_2 \)max during the ramp test, each participant performed a supramaximal exercise bout to exhaustion at an intensity corresponding to 105% of the peak power achieved during the ramp test (5). In all cases the highest 15-s averaged \( \dot{V}O_2 \) elicited during supramaximal exercise did not exceed the highest ramp-determined \( \dot{V}O_2 \) by >4%, which is considered to represent the day-to-day reliability of the highest achievable \( \dot{V}O_2 \) during maximal exercise testing in children (44). Therefore, the highest recorded \( \dot{V}O_2 \) during the ramp or supramaximal test was taken as a valid \( \dot{V}O_2 \)max. The \( \dot{V}O_2 \) corresponding to the GET was used to noninvasively estimate the blood lactate threshold by identifying a disproportionate increase in expired carbon dioxide (\( \dot{V}CO_2 \)) relative to \( \dot{V}O_2 \) (7).

Square-wave exercise. During visits 2–4, each participant completed repeat square-wave exercise bouts separated by a period of light exercise. The test consisted of 6 min of cycling at 10 W, followed by a step change in power output that was designed to elicit a \( \dot{V}O_2 \) amplitude corresponding to Δ60%. Participants cycled at Δ60% for 6 min and then undertook a 6-min recovery cycle at 10 W before completing a second 6-min duration step change in power output to Δ60%. The \( \dot{V}O_2 \) corresponding to Δ60% was determined using the “linear” portion of the ramp test by removing the initial 2 and final 3 min of the test data and following adjustment of the \( \dot{V}O_2 \) “lag time” that is observed during ramp exercise (45). Assuming the lag time to be ~30 s, the target \( \dot{V}O_2 \) amplitude was assumed to be attained ~120 s into the square-wave exercise bout (i.e., before the onset of the \( \dot{V}O_2 \) slow component).
[Hb + Mb] and oxy-[Hb + Mb] were calculated using a modified Beer-Lambert law. The deoxy-[Hb + Mb] signal is considered to reflect the dynamic (im)balance between muscle O₂ supply and O₂ utilization, and therefore, it provides information on changes in muscle fractional O₂ extraction (14, 16, 26). The sum of oxy-[Hb + Mb] and deoxy-[Hb + Mb] was used to calculate total Hb + Mb (tot-[Hb + Mb]). All muscle oxygenation variables were interpolated to 1-s intervals and expressed as a change, in arbitrary units, from baseline (following 10 min of rest while seated on the cycle ergometer).

Data Analysis

Breath-by-breath changes in V˙O₂ for each square-wave exercise bout were initially examined for fluctuations that were >4 SD from a local moving mean. Subsequently, the three repeat square-wave exercise bouts for each of the control and primed conditions were linearly interpolated to 1 s, time aligned to exercise onset (t = 0 s) and ensemble averaged, yielding a single response profile for each participant. The resultant averaged V˙O₂ response for each of the control and primed conditions was baseline corrected by subtracting the mean V˙O₂ between −60 and −15 s from the exercise response. Following removal of phase I (cardiodynamic) by omitting the initial 15 s of data, the phase II portion of the V˙O₂ response was characterized using the nonlinear equation:

\[ V_{O2} = \Delta V_{O2} (1 - e^{-\frac{t}{TD}}) \]

where \( V_{O2} \), \( \Delta V_{O2} \), \( TD \), and \( \tau \) represent the value of V˙O₂ at a given time (t), the amplitude change in V˙O₂ from baseline to its asymptote, time delay, and the time constant of the response, respectively.

Following the methods of Rossiter et al. (41), the equation was initially fit up to the first 60 s of exercise and then increased iteratively by 1 s to end exercise using a purpose-designed program (LabView, version 6.1, National Instruments, Newbury, UK). The best fit curve for the phase II portion of the response was established using J) a plot of the V˙O₂ against time to identify the point at which the influence of the V˙O₂ slow component lengthened the estimated \( \tau \) following an initial plateau and 2) deviation from an optimal fitting of the model as judged by a systematic departure of the model’s residuals. The phase II parameter estimates from the equation were then resolved by least-squares nonlinear regression (GraphPad Prism; GraphPad Software, San Diego, CA). The magnitude of the V˙O₂ slow component was calculated from the difference between the mean of the final 30 s at the end of exercise and the phase II asymptote. The V˙O₂ slow-component amplitude was also expressed in relative terms against end-exercise V˙O₂. To provide a description of the overall kinetic response [i.e., mean response time (MRT)], the above equation with TD constrained to 0 s (i.e., no delay term) was fit from exercise onset to the end of exercise. Cardiac output and deoxy-[Hb + Mb] profiles were averaged into 5-s data bins, time aligned to exercise onset, and ensemble averaged to yield a single response for the control and primed exercise conditions. The Q and deoxy-[Hb + Mb] dynamics (primary and slow-component phases) were modeled in a fashion similar to the procedures described for V˙O₂ above, but with some slight modifications. Because no discernible delay was evident in the Q response at exercise onset, the TD parameter in the above equation was constrained to 0 s (monoexponential model with no delay). In contrast, the deoxy-[Hb + Mb] profile increased with exponential-like properties after a short delay. Following the methods of DeLorey et al. (16), the time at which the exponential-like increase in deoxy-[Hb + Mb] commenced was determined as being at 1 SD increase above baseline. The equation was then applied to resolve the deoxy-[Hb + Mb] TD and \( \tau \) following removal of the data preceding this exponential-like increase. The deoxy-[Hb + Mb] MRT was calculated by summing TD and \( \tau \) to provide an overall description of the kinetic response in the primary phase.

To provide a description of the heart rate, stroke volume, and \( C_{O2} + V_{O2} \) changes during exercise, data were averaged into 15-s data bins and analyzed for mean differences between the control and primed bouts at baseline and at 15, 30, 60, 120, 180, and 360 s of exercise. Because the oxy-[Hb + Mb] and tot-[Hb + Mb] signals did not conform to an exponential-like profile, analysis of these variables was restricted to baseline and end of exercise.

Statistics

Paired sample \( t \)-tests were employed to examine the mean differences in V˙O₂, Q, and deoxy-[Hb + Mb] kinetic parameters between the control and primed conditions. In addition, effect size (\( d \)) statistics were calculated using a pooled SD for the control and primed conditions (43). Analysis of the physiological variables (e.g., heart rate, stroke volume) against time was achieved using two-way repeated measures ANOVA. If a significant condition by time interaction effect was noted, the main effect results were not reported. When appropriate, the Greenhouse-Geisser correction factor was applied to the ANOVA model degrees of freedom when Mauchly’s test of sphericity was violated. Significant differences were followed up using planned pairwise comparisons employing the Bonferroni correction factor. All statistical procedures were performed using SPSS version 15.0 (SPSS, Chicago, IL), with the null hypothesis rejected at an \( \alpha \)-level of 0.05. Data are reported as means ± SD.

RESULTS

Ramp and Supramaximal Exercise

The mean V˙O₂max was 2.26 ± 0.68 l/min, which corresponded to a peak power output of 170 ± 68 W. Peak mean PhysioFlow-derived variables during ramp exercise were Q: 16.1 ± 4.9 l/min; heart rate: 205 ± 4 beats/min; and stroke volume: 85 ± 24 ml/beat. The peak mean \( C_{O2} + V_{O2} \) was 13.1 ± 1.1 ml/min⁻¹100 ml⁻¹. The GET occurred at a V˙O₂ of 1.14 ± 0.46 l/min, which equated to 50 ± 8% V˙O₂max. This yielded a target V˙O₂ after ~120 s of exercise during the square-wave bouts at Δ60% of 1.80 ± 0.60 l/min (79 ± 4% V˙O₂max), which corresponded to a power output of 121 ± 50 W.

Square-Wave Exercise

V˙O₂ dynamics. The group mean V˙O₂ response during the control and primed cycling exercise bouts is shown in Fig. 1. Table 1 presents the V˙O₂ kinetic response parameters. Compared with the control bout, priming exercise resulted in an elevated baseline V˙O₂ but the phase II V˙O₂ \( \tau \), TD, amplitude, and “gain” were unaffected by priming exercise. The 95% confidence interval for the phase II V˙O₂ \( \tau \) in both the control and primed condition was 4 ± 2 s. The total V˙O₂ amplitude during phase II was significantly elevated in the primed condition compared with the control. A V˙O₂ slow component was present in all exercise bouts. Priming exercise resulted in a significant reduction in the V˙O₂ slow-component amplitude (in both absolute and relative terms) compared with the control condition. End-exercise V˙O₂ was similar between the two cycling bouts and represented 84 ± 4 and 85 ± 6% of V˙O₂max in the control and primed conditions, respectively. Finally, priming exercise resulted in a reduced V˙O₂ mean response time (i.e., faster overall kinetic response) compared with the control bout.

Q dynamics. The mean Q response during the control and primed cycling exercise bouts is illustrated in Fig. 2, and the mean Q kinetic parameters are presented in Table 2. At
baseline, Q was significantly elevated following priming exercise. No significant difference was noted for the Q primary \( \tau \) between the control and primed exercise bouts. The Q primary \( \tau \) was significantly slower than the phase II VO\(_2\) \( \tau \) for both exercise conditions \((P < 0.01, d = 1.6)\), but the variables were correlated during the control \((r = 0.71, P = 0.049)\) and primed conditions \((r = 0.71, P = 0.049)\). Exercise following priming exercise was associated with a reduced Q primary amplitude, although the total primary amplitude (baseline + primary Q) was significantly elevated compared with control. A Q slow component was manifest in all response profiles and, whether expressed in absolute or relative terms, was unaffected by priming exercise. End-exercise Q was significantly elevated in the primed condition. The Q mean response time \((i.e., \text{overall kinetic response})\) was significantly faster in the primed compared with the control condition. The increase in Q relative to VO\(_2\) \((Q/VO_2)\) was similar between conditions in the primary/phase II portion of the response \((control: 8.0 \pm 1.0 \text{ vs. primed: } 8.1 \pm 1.1; P = 0.28, d = 0.1)\) but was greater in the primed compared with the control condition at the end of exercise \((8.2 \pm 1.2 \text{ vs. } 7.9 \pm 1.0, \text{ respectively; } P = 0.027, d = 0.3)\).

Heart rate, stroke volume, and C\((a-v)O_2\) dynamics. The influence of priming exercise on the dynamics of heart rate, stroke volume, and C\((a-v)O_2\) is illustrated in Fig. 3, with the group means at selected time points presented in Table 3. Repeated-measures ANOVA revealed a significant condition by time interaction effect for heart rate \((P < 0.001)\). Followup comparisons located a significantly higher heart rate in the primed compared with the control condition at baseline \((P = 0.003)\) and at 15 \((P = 0.004)\), 30 \((P = 0.001)\), 60 \((P = 0.003)\), 120 \((P < 0.001)\), 180 \((P = 0.002)\), and 360 s \((P < 0.001)\) (Fig. 3A).

The onset of very heavy exercise was associated with a rapid increase in stroke volume for both conditions and the attainment of a steady-state amplitude after \(\sim 60\) s. Repeated-measures ANOVA revealed a significant condition by time interaction effect for stroke volume changes during exercise \((P = 0.002)\) and at 15 \((P = 0.004)\), 30 \((P = 0.001)\), 60 \((P = 0.003)\), 120 \((P < 0.001)\), 180 \((P = 0.002)\), and 360 s \((P < 0.001)\) (Fig. 3A).

Table 1. Oxygen uptake kinetics during the control and primed heavy cycle bouts

<table>
<thead>
<tr>
<th>Variable</th>
<th>Bout 1 (Control)</th>
<th>Bout 2 (Primed)</th>
<th>( P ) Value</th>
<th>Effect Size ((d))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline VO(_2), l/min</td>
<td>0.63 ± 0.24</td>
<td>0.71 ± 0.29</td>
<td>0.004</td>
<td>0.3</td>
</tr>
<tr>
<td>Phase II ( \tau ), s</td>
<td>22 ± 7</td>
<td>20 ± 4</td>
<td>0.30</td>
<td>0.4</td>
</tr>
<tr>
<td>Phase II ( \tau ), 95% CI, s</td>
<td>4 ± 2</td>
<td>4 ± 2</td>
<td>0.87</td>
<td>0.1</td>
</tr>
<tr>
<td>Phase II TD, s</td>
<td>10 ± 3</td>
<td>10 ± 3</td>
<td>0.56</td>
<td>0.1</td>
</tr>
<tr>
<td>Phase II VO(_2) amplitude, l/min</td>
<td>1.10 ± 0.33</td>
<td>1.09 ± 0.31</td>
<td>0.082</td>
<td>0.0</td>
</tr>
<tr>
<td>Phase II VO(_2) total amplitude, l/min</td>
<td>1.73 ± 0.56</td>
<td>1.80 ± 0.59</td>
<td>0.002</td>
<td>0.1</td>
</tr>
<tr>
<td>Phase II gain, ml-min(^{-1})-W(^{-1})</td>
<td>10.7 ± 1.9</td>
<td>10.7 ± 1.9</td>
<td>0.88</td>
<td>0.0</td>
</tr>
<tr>
<td>VO(_2) slow-component onset, s</td>
<td>138 ± 48</td>
<td>140 ± 64</td>
<td>0.056</td>
<td>0.1</td>
</tr>
<tr>
<td>VO(_2) slow-component amplitude, l/min</td>
<td>0.18 ± 0.08</td>
<td>0.12 ± 0.09</td>
<td>0.048</td>
<td>0.7</td>
</tr>
<tr>
<td>VO(_2) slow-component relative amplitude, %</td>
<td>9 ± 3</td>
<td>6 ± 2</td>
<td>0.037</td>
<td>1.3</td>
</tr>
<tr>
<td>End-exercise VO(_2), l/min</td>
<td>1.90 ± 0.63</td>
<td>1.92 ± 0.66</td>
<td>0.38</td>
<td>0.0</td>
</tr>
<tr>
<td>End-exercise VO(_2) gain, ml-min(^{-1})-W(^{-1})</td>
<td>12.3 ± 1.9</td>
<td>11.8 ± 2.0</td>
<td>0.018</td>
<td>0.3</td>
</tr>
<tr>
<td>VO(_2) mean response time, s</td>
<td>43 ± 8</td>
<td>36 ± 5</td>
<td>0.002</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Data are presented as means ± SD. CI, confidence interval; TD, time delay; \( \tau \), time constant of the response.
0.023). Pairwise comparisons located a significantly higher stroke volume in the primed compared with the control condition at 15 (P = 0.016) and 30 s (P = 0.021) into the exercise protocol (Fig. 3B). No significant differences (P > 0.1) in stroke volume were identified at baseline or at 60, 120, 180, and 360 s.

Exercise was associated with an increase in C(a-v)O2 in both experimental conditions, with a steady-state amplitude attained after ~60 s. Repeated-measures ANOVA revealed a nonsignificant condition by time interaction effect (P = 0.78), highlighting a similar C(a-v)O2 at baseline and throughout the exercise bout in the control and primed conditions (Fig. 3C).

Muscle oxygenation. The mean deoxy- and oxy-[Hb + Mb] response profiles during the control and primed exercise conditions are illustrated in Fig. 4. The parameter estimates from the deoxy-[Hb + Mb] kinetic analysis and baseline and end-exercise values for oxy- and tot-[Hb + Mb] are provided in Table 4. Although nonsignificant, a large effect size (d = 0.9) indicates a meaningful decrease in deoxy-[Hb + Mb] and elevation in oxy-[Hb + Mb] at baseline following priming exercise compared with the control condition. However, tot-[Hb + Mb] remained unchanged at baseline and end of exercise across the two exercise bouts. The kinetic parameters (TD, τ, TD + τ, and amplitude) describing the adaptation of deoxy-[Hb + Mb] during the primary phase were unaffected by priming exercise. A slow-component phase for deoxy-[Hb + Mb] was identified in seven of the eight participants and was unaltered by priming exercise. End of exercise and deoxy-, oxy-, and tot-[Hb + Mb] were not significantly different between the two exercise bouts.

**DISCUSSION**

This is the first study to investigate the influence of a prior bout of very heavy exercise on the kinetics of VO2, Q, and muscle oxygenation during a subsequent bout of very heavy exercise in young people. In line with our experimental hypothesis, prior very heavy exercise resulted in an increase in Q and localized muscle oxygenation prior to initiation of the second bout. However, despite this increase in muscle O2 availability at exercise onset, and in line with our remaining hypotheses, the phase II VO2 τ was not altered, the phase II total amplitude was elevated, and the VO2 slow component was reduced following priming exercise. The dynamics of deoxy-[Hb + Mb], which are considered to reflect muscle fractional O2 extraction, were unchanged following priming exercise, suggesting that the observed changes in the VO2 response were related to alterations in muscle O2 delivery. Changes in central blood flow are consistent with this proposal since Q was increased throughout the exercise bout such that, at the end of exercise, the matching of Q to the metabolic rate (Q/VO2) was elevated compared with the control.

**Priming Exercise and Phase II VO2**

Priming exercise resulted in a significant and marked (d = 1.0) speeding of the overall VO2 kinetic response compared with the control bout, but the phase II τ was unaltered. Unlike treadmill running in adults, where priming exercise has no influence on the VO2 kinetic response (27), this finding is consistent with a body of literature that has investigated the influence of priming exercise on the VO2 kinetic response in healthy young adults cycling in the upright position (2, 10, 12, 26). The more rapid overall VO2 kinetic response was attributable to an increase in the total phase II VO2 amplitude and a reduced VO2 slow-component amplitude, although the latter may be more meaningful (d = 0.7–1.3) than the former (d = 0.1). Therefore, the present results contrast with reports where faster phase II VO2 kinetics have been documented during upright exercise in the elderly (15, 21) and in adults with relatively slow VO2 kinetics (phase II τ > 30–35 s) in the control condition (14, 23).

Gerbini et al. (20) hypothesized that the metabolic acidosis and increased muscle temperature brought about by performing a bout of high-intensity exercise would result in an increase in muscle O2 delivery in a subsequent bout of exercise due to localized vasodilatation and the Bohr effect on the hemoglobin O2 dissociation curve. In the present study, oxy-[Hb + Mb], an index of muscle oxygenation, was markedly elevated (d = 0.9) above control at the beginning of the primed exercise bout, which is consistent with previous priming studies using near-infrared spectroscopy (NIRS) methodology in adults (14, 21, 26). As an indicator of bulk blood flow, Q was elevated prior to (d = 0.6) and throughout the primed exercise bout, albeit with low-magnitude effects (d = 0.1–0.4), compared with the control condition. The elevated Q during the initial 30 s of exercise in the primed bout was caused not only by an elevated heart rate but also by a more rapid increase in stroke volume such that it attained its steady-state amplitude some 30 s earlier than in the control bout (Fig. 3). Faisal et al. (17) observed similar findings for Q dynamics during repeated bouts of heavy-intensity running in endurance-trained adults and attributed an early “overshoot” in stroke volume during the primed bout to an increase in venous return and hence, myocardial preload caused by the muscle pump effect. Faisal et al. (17) noted a significant speeding in the primary kinetics of Q following priming exercise, which coincided with more rapid phase II VO2 kinetics. In the present study, the 4-s speeding of the primary
Q˙ kinetics following priming exercise was nonsignificant, with a low to moderate effect size (d = 0.4), despite the more rapid stroke volume adaptation. In contrast, the overall Q˙ kinetic response was significantly speeded (d = 0.5), which is consistent with the findings of Perrey et al. (37) in healthy adults during cycle ergometry.

In the present study, NIRS was used to quantify the changes of deoxy-[Hb + Mb] at the onset of exercise, since this is believed to represent the (im)balance between local O2 delivery and utilization and thus reflect muscle fractional O2 extraction (14, 16, 26). As has previously been reported in adults during exercise above the GET, a delay of ~5–6 s was noted in the control condition at the onset of exercise, indicating a close matching of O2 delivery to O2 utilization at the myocyte (16, 33). In the present study, the deoxy-[Hb + Mb] delay term was insensitive to priming exercise (d = 0.4), which contrasts with adult studies showing a reduced deoxy-[Hb + Mb] delay term following a bout of prior exercise (15, 23, 33). Following this short delay, deoxy-[Hb + Mb] rose exponentially, at a rate that was unaffected by priming exercise (d = 0.4), meaning that the overall deoxy-[Hb + Mb] kinetics in the primary phase (i.e., MRT) were unaltered by the experimental intervention (d = 0.2). Coupled with the finding that the deoxy-[Hb + Mb] primary amplitude was similar to the control in the primed condition (d = 0.1), these data imply that muscle fractional O2 extraction was insensitive to priming exercise during the non-steady state. Although these results are inconsistent with priming studies in young adults showing an enhanced muscle O2 extraction through more rapid deoxy-[Hb + Mb] kinetics (15, 23, 33), an increased primary deoxy-[Hb + Mb] amplitude (14), or both (22), our findings are not unprecedented. Jones et al. (26) reported unchanged deoxy-[Hb + Mb] primary kinetics following repeat bouts of cycling exercise at ~50% in healthy adults, albeit with a longer recovery interval of 10 min between bouts. Bailey et al. (2) have recently demonstrated that the priming effect on deoxy-[Hb + Mb] kinetics (i.e., consistent with increased muscle O2 extraction) during very heavy exercise recedes as the recovery interval is increased, although the effect is still preserved up to 20 min of recovery. Therefore, our deoxy-[Hb + Mb] data may indicate that the mechanism (i.e., oxidative enzyme activation) underlying the enhanced muscle O2 extraction that is invariably observed following priming exercise in adults is modulated during growth and maturation, with factors such as the intensity of the priming bout and/or the recovery duration employed likely to play an important role.

Although the deoxy-[Hb + Mb] primary kinetics (TD, τ, and MRT) indicate that the dynamic matching of muscle O2 delivery to O2 utilization was unaltered by priming exercise in the non-steady state, the elevated total phase II V˙O2 amplitude following priming exercise suggests, in accordance with the Fick principle, that muscle blood flow was elevated during the “steady state.” Although it could be postulated that the elevated baseline V˙O2 in the present study may have contributed, in part, to the increased total phase II V˙O2 amplitude, studies in healthy adults during cycling exercise at ∆50–70% have shown that this effect is independent of baseline V˙O2 (11, 12). Therefore, based on the reasonable assumption that the phase II V˙O2 amplitude faithfully reflects that of muscle O2 uptake (29), the observation of an unaltered deoxy-[Hb + Mb] primary amplitude coupled with an elevated total phase II V˙O2 amplitude...
in the present study suggests that muscle O₂ delivery was increased following priming exercise in the steady state. This conclusion is consistent with the elevated Q throughout the primary phase and implies that central changes in blood flow brought about a concomitant increase in flow (and hence, O₂ availability) in the muscle microcirculation. However, it must be stressed that, because muscle blood flow was not measured in the present study, we cannot discount the possibility that the systemic consequences of priming exercise, such as an elevated core temperature, contributed in part to the elevated Q.

Despite the evidence of a greater availability of O₂ to the contracting muscles before and possibly during the steady state in the present study, the phase II V˙O₂ τ was unaltered. Therefore, this result lends support to the notion that the principal determinant(s) of the rate at which phase II V˙O₂, and by extension muscle O₂ consumption (29), increases at exercise onset in boys <13 yr of age is of intracellular origin. Indeed, present models of metabolic control for healthy adults favor the intrinsic ability of the myocyte to utilize O₂ as the main limiting factor for oxidative metabolism at exercise onset rather than a muscle O₂ perfusion limitation (32, 34, 38). Therefore, the present results suggest that the notion that O₂ delivery does not appear to limit oxidative metabolism in healthy humans has its origin in childhood and is unlikely to account for the slowing of the V˙O₂ kinetic response that is observed from childhood into adulthood (1, 9, 18, 19). Rather, changes in the O₂ utilization potential of the myocyte (related to, for example, phosphate regulation or rate-limiting oxidative enzyme activation; Refs. 1, 3, 4, and 6) are more likely to be responsible.

In agreement with a number of adult studies (2, 11, 12), the total phase II V˙O₂ amplitude was significantly elevated following priming exercise, although the meaningfulness of this change may be questionable (d = 0.1). As highlighted above, because muscle O₂ extraction remained unchanged following priming exercise, the increased total V˙O₂ phase II amplitude may be related to altered blood flow dynamics since the matching of Q to the metabolic rate (Q/V˙O₂) in the primary phase was comparable between the control and primed bouts. Indeed, adult studies employing supine exercise to reduce perfusion pressure (28) and the inspiration of hyperoxic gas (cf. 31, 46), also suggest that the phase II V˙O₂ amplitude may be O₂ delivery sensitive. However, although not measured in the present study, an alternative or accompanying explanation for the elevated total phase II V˙O₂ amplitude in bout 2 may relate to altered muscle fiber recruitment patterns at exercise onset. Burnley et al. (10) reported, following a priming exercise bout, an elevated integrated electromyogram within the gluteus maximus, vastus lateralis, and vastus medialis muscles in the initial ~2 min of exercise onset, which was proportional to the increase in the phase II V˙O₂ amplitude. In this scenario, an increased recruitment of muscle fibers following the onset
of exercise might have increased the muscle \( \text{O}_2 \) demand and provoked a concomitant rise in \( \dot{Q} \).

**Priming Exercise and the \( \dot{V_\text{O}_2} \) Slow Component**

Priming exercise resulted in a marked reduction in the \( \dot{V_\text{O}_2} \) slow component in the present study \((d = 0.7–1.3)\), which is in agreement with previous adult cycling studies \((2, 12, 26, 31, 37)\). Although the mechanistic bases of the \( \dot{V_\text{O}_2} \) slow component are not fully understood, it is clear that the response reflects intramuscular processes \((29, 39, 41)\), which are likely to be related to the development of muscle fatigue throughout the exercise bout. However, a role for the delivery of \( \text{O}_2 \) to the contracting muscles cannot be discounted. For example, the \( \dot{V_\text{O}_2} \) slow component is markedly reduced during hyperoxic gas breathing in adults during heavy-intensity cycling, without a concomitant change in the phase II amplitude \((46)\). Interestingly, our data also support a possible role for muscle \( \text{O}_2 \) availability in the development of the \( \dot{V_\text{O}_2} \) slow component because, at the end of exercise, \( \dot{Q}/\dot{V_\text{O}_2} \) was elevated in the primed condition, albeit with a low effect size \((d = 0.3)\). Because \( \dot{Q}/\dot{V_\text{O}_2} \) was proportional in the primary/phase II portion of the response, this finding suggests a continued rise in the ratio of central blood flow (and presumably muscle blood flow) to metabolic demand throughout the second bout. However, leg blood flow in young adults during knee extensor exercise has been shown to change proportionately with \( \dot{V_\text{O}_2} \) following priming exercise \((14)\). Interestingly, the amplitude of oxy-[Hb + Mb] was elevated throughout exercise in the primed condition, possibly suggesting that, even in the absence of an increase in blood flow, the distribution of blood flow to the active tissues was improved \((14)\).

In the present study, priming exercise had no influence on the magnitude of the deoxy-[Hb + Mb] slow component, which is in agreement with previous adult studies during cycling exercise that also show a reduced \( \dot{V_\text{O}_2} \) slow-component amplitude \((2, 26)\). Therefore, this finding suggests that the extraction of \( \text{O}_2 \) in the myocytes was unchanged throughout the bout, which corroborates the estimated whole body \( C_(a-v)\text{O}_2 \) data. Because the \( \dot{V_\text{O}_2} \) slow component is predominantly of muscular origin \((29)\), the NIRS-derived data combined with the elevated \( \dot{V_\text{O}_2} \) in the slow-component region of the second bout \((\text{Fig. 1})\) may imply an elevated muscle blood flow and presumably \( \text{O}_2 \) availability. However, despite oxy-[Hb + Mb] being consistently elevated in the primed exercise condition \((\text{Fig. 4})\), this difference was not significant and had a low effect size \((d = 0.1)\) at the end of exercise. Therefore, it is possible that the elevated \( \dot{Q} \) failed to promote an increase in muscle \( \text{O}_2 \) availability following priming exercise in the present study.

An alternative explanation for the reduced \( \dot{V_\text{O}_2} \) slow component following priming exercise may be related to the increased recruitment of muscle fibers at exercise onset. The reduced metabolic strain per fiber in this situation might negate the requirement to recruit additional motor units throughout the exercise bout, as reflected by integrated electromyogram measurements \((2, 10)\). Because altered muscle recruitment patterns, specifically the higher-order type II fibers, at exercise onset and/or progressively throughout the test are known to play a key role in modulating the \( \dot{V_\text{O}_2} \) slow component \((30, 47)\), it is possible that muscle fiber recruitment was altered in the present study. Whether this mechanism acts independently of possible changes in \( \text{O}_2 \) delivery is unknown, but an elevated blood flow and muscle oxygenation following priming exercise would be expected to reduce the metabolic perturbation in the already active muscle fibers \((25)\), thereby slowing the rate of fatigue development and the requirement to recruit additional fibers to maintain the target work rate.

**CONCLUSIONS**

The present investigation provides unique data regarding the adaptation of \( \dot{V_\text{O}_2} \), \( \dot{Q} \), and muscle oxygenation dynamics during a bout of very heavy cycling exercise following an initial priming bout in 9- to 13-yr-old boys. Therefore, the results provide novel insights into the factors that may limit muscle oxidative metabolism in youth. Priming exercise was successful in elevating muscle oxygenation prior to, and \( \dot{Q} \) prior to and throughout, the second exercise bout. However, the phase II \( \dot{V_\text{O}_2} \) kinetics were unaltered, suggesting that the availability of muscle \( \text{O}_2 \) at exercise onset does not limit the initial increase in \( \dot{V_\text{O}_2} \) following the onset of exercise in healthy 9- to 13-yr-old boys. In contrast, priming exercise did result in faster overall \( \dot{V_\text{O}_2} \) kinetics, a finding that can be attributed to an elevated total phase II \( \dot{V_\text{O}_2} \) amplitude and a reduced \( \dot{V_\text{O}_2} \) slow-component amplitude. Because deoxy-[Hb + Mb], and therefore, the muscles’ fractional \( \text{O}_2 \) utilization, was unaltered following priming exercise, the proportional matching of \( \dot{Q} \) to \( \dot{V_\text{O}_2} \) during phase II and an elevated \( \dot{Q}/\dot{V_\text{O}_2} \) at the end of exercise suggests

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**Table 4. Deoxy-, oxy-, and tot-[Hb + Mb] dynamics during the control and primed heavy cycling conditions**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Bout 1 (Control)</th>
<th>Bout 2 (Primed)</th>
<th>( P ) Value</th>
<th>Effect Size ((d))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deoxy-[Hb + Mb] baseline (AU)</td>
<td>(-2 \pm 5)</td>
<td>(-23 \pm 35)</td>
<td>0.14</td>
<td>0.9</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] primary TDF, s</td>
<td>6 \pm 4</td>
<td>5 \pm 2</td>
<td>0.31</td>
<td>0.4</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] primary ( \tau ), s</td>
<td>12 \pm 3</td>
<td>15 \pm 11</td>
<td>0.37</td>
<td>0.4</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] primary MRT, s</td>
<td>18 \pm 6</td>
<td>20 \pm 12</td>
<td>0.45</td>
<td>0.2</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] primary amplitude (AU)</td>
<td>51 \pm 88</td>
<td>59 \pm 100</td>
<td>0.12</td>
<td>0.1</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] slow-component amplitude (AU)</td>
<td>2 \pm 11</td>
<td>5 \pm 9</td>
<td>0.39</td>
<td>0.3</td>
</tr>
<tr>
<td>Deoxy-[Hb + Mb] slow-component amplitude, %</td>
<td>11 \pm 11</td>
<td>6 \pm 7</td>
<td>0.16</td>
<td>0.5</td>
</tr>
<tr>
<td>End-exercise deoxy-[Hb + Mb] (AU)</td>
<td>52 \pm 84</td>
<td>64 \pm 106</td>
<td>0.19</td>
<td>0.1</td>
</tr>
<tr>
<td>Oxy-[Hb + Mb] baseline (AU)</td>
<td>(-6 \pm 15)</td>
<td>(-38 \pm 70)</td>
<td>0.11</td>
<td>0.1</td>
</tr>
<tr>
<td>End-exercise oxy-[Hb + Mb] (AU)</td>
<td>(-48 \pm 84)</td>
<td>(-53 \pm 92)</td>
<td>0.21</td>
<td>0.1</td>
</tr>
<tr>
<td>Oxy-[Hb + Mb] - ( \Delta ) (AU)</td>
<td>(-42 \pm 72)</td>
<td>(-9 \pm 28)</td>
<td>0.86</td>
<td>0.1</td>
</tr>
<tr>
<td>Tot-[Hb + Mb] baseline (AU)</td>
<td>(-7 \pm 13)</td>
<td>2 \pm 21</td>
<td>0.88</td>
<td>0.1</td>
</tr>
<tr>
<td>End-exercise tot-[Hb + Mb] (AU)</td>
<td>(-7 \pm 13)</td>
<td>11 \pm 16</td>
<td>0.83</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Data are presented as means \pm SD. AU, arbitrary units.

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that the altered VO₂ amplitudes may be related, either directly or indirectly, to an increased muscle O₂ availability following priming exercise.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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