Oxygen uptake kinetics during two bouts of heavy cycling separated by fatiguing sprint exercise in humans

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Oxygen uptake kinetics during two bouts of heavy cycling separated by fatiguing sprint exercise in humans. J Appl Physiol 94: 533–541, 2003. First published October 11, 2002; 10.1152/japplphysiol.00532.2002.—We tested the hypothesis that O2 uptake (Vo2) kinetics at the onset of heavy exercise would be altered in a state of muscle fatigue and prior metabolic acidosis. Eight well-trained cyclists completed two identical bouts of 6-min cycling exercise at >85% of peak Vo2 separated by three successive bouts of 30 s of sprint cycling. Not only was baseline Vo2 elevated after prior sprint exercises but also the time constant of phase II Vo2 kinetics was faster (28.9 ± 2.4 vs. 22.2 ± 1.7 s; P < 0.05). CO2 output (VCO2) was significantly reduced throughout the second exercise bout. Subsequently Vo2 was greater at 3 min and increased less after this after prior sprint exercise. Cardiac output, estimated by impedance cardiography, was significantly higher in the first 2 min of the second heavy exercise bout. Normalized integrated surface electromyography of four leg muscles and normalized mean power frequency were not different between exercise bouts. Vo2 and VCO2 kinetic responses to heavy exercise were markedly altered by prior multiple sprint exercises.

cardiac output; high-intensity cycling; muscle fatigue; electromyography

THE PULMONARY OXYGEN UPTAKE (Vo2) response to constant-intensity exercise reflects the time course of the adjustment of muscle Vo2 toward a steady state (4, 40). Gerbino et al. (18) first demonstrated that Vo2 kinetics during heavy exercise were accelerated by a prior bout of heavy exercise and proposed that this speeding was the consequence of an increased vasodilatation and a greater O2 availability during the second bout of heavy exercise. MacDonald et al. (31) demonstrated even faster adaptation of Vo2 in the first as well as the second bout of exercise when arterial O2 content was increased. Also of interest during the adaptation to heavy-intensity exercise is the “slow component” or the progressive increase in Vo2 above that predicted for the work rate (33). The slow component has its origin in the working muscles (37), but the mechanism is unre-solved (5, 41). The magnitude of this slow component is reduced by prior heavy intensity exercise (13, 18, 31), and this corresponds with less depletion of muscle phosphocreatine (41).

In contrast to the early reports by Gerbino et al. (18) and MacDonald et al. (31), some investigators have stated the higher Vo2 at the onset of a second bout of high-intensity exercise or after a single 30-s sprint was simply a function of increased recruitment of muscle fibers early in exercise (13), perhaps reducing the metabolic disturbance in individual fibers (11, 12, 26). Of particular interest is that these investigators have interpreted their data to indicate that although Vo2 is elevated early in exercise, the actual rate of increase as assessed by the phase II time constant was not altered (13, 26). Results such as these have lead some investigators to propose that O2 delivery was not limiting the rate of increase in oxidative metabolism at the onset of heavy- to very heavy-intensity exercise (3, 11–13). Recent direct investigations of O2 transport during repeated bouts of high-intensity exercise demonstrated residual effects of the first bout of high-intensity exercise on forearm (30) and leg (28) acid-base status and other potential vasodilators in venous blood from the formerly working muscle. These factors might have contributed to elevated blood flow and greater O2 extraction early in a subsequent exercise bout. In light of these experiments (28, 30), we hypothesized that a greater increase in the level of metabolic acidosis during heavy-intensity exercise might cause a further, and detectable, acceleration in Vo2 kinetics in the second bout of heavy, constant-load exercise. To achieve this marked acidosis, we had subjects complete three all-out 30-s sprint cycle tests between the two bouts of heavy constant-load exercise. It has been suggested that the fiber recruitment, and by extension a delayed and additional slow component of Vo2, could be related to exercise intensity and the muscle pH (42). Therefore, we hypothesized that the slow component would occur earlier and be of larger magnitude in the second exercise bout after fatiguing sprint exercises.

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METHODS

Subjects. Eight well-trained male cyclists (age 29.8 ± 1.9 yr, height 177.2 ± 2.3 cm, and weight 71.3 ± 2.4 kg) participated in this study. After receiving complete verbal and written details of the protocol, each subject gave informed, written consent on a form approved by the office of research ethics at the University of Waterloo.

Experimental design. Each subject performed preliminary testing consisting of an incremental exercise to volitional fatigue in which the work rate increased as a ramp function by 30 W/min at a cycling frequency of 80 rpm. We defined fatigue as the point when the subject was unable to maintain a pedaling rate of 75 rpm, despite strong verbal encouragement. The gas-exchange data obtained from the ramp test were used to estimate the ventilatory threshold (VT) and the peak \( \dot{V}O_2 \) (\( \dot{V}O_2 \text{ peak} \)). VT was determined from the point of increased minute ventilation (VE)-to-\( \dot{V}O_2 \) ratio. \( \dot{V}O_2 \text{ peak} \) was taken as the average of the highest five consecutive breaths attained in the last minute of exercise.

On the second test day, subjects performed two identical step transitions lasting 6 min after 4-min baseline cycling at 25 W. This work rate was estimated to require \( \dot{V}O_2 \) equal to \( \sim 85\% \) of \( \dot{V}O_2 \text{ peak} \), although progressive increase in \( \dot{V}O_2 \) above this value was expected. After the first submaximal exercise bout, subjects rested for 10 min before completing three 30-s bouts of all-out sprint cycling [Wingate test (16)]. The three sprint exercises were separated by 4 min of passive recovery. After the third sprint, subjects rested for 10 min divided in 6 min of passive recovery and 4 min of baseline cycling at 25 W, and then they performed the second step exercise.

All submaximal tests were conducted on the same electromagnetically braked cycle ergometer (Excalibur, Lode, Groningen, Netherlands). Seat and handlebar positions were kept constant for individual subjects during the course of the study and subjects used their own shoes and pedals. Pedal frequency was maintained at 80 rpm for both identical step transitions.

For the sprint exercises, the subjects performed a standard Wingate test (16). This consisted of 30 s of maximal effort on a mechanically braked cycle ergometer (model 841E, Monark, Varberg, Sweden) against a preset friction load of 625 W. The gas-exchange data obtained from the ramp test were used to estimate the ventilatory threshold (VT) and the peak \( \dot{V}O_2 \) (\( \dot{V}O_2 \text{ peak} \)). VT was determined from the point of increased minute ventilation (VE)-to-\( \dot{V}O_2 \) ratio. \( \dot{V}O_2 \text{ peak} \) was taken as the average of the highest five consecutive breaths attained in the last minute of exercise.

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Data analysis. Breath-by-breath data for \( \dot{V}O_2 \) for each subject were linearly interpolated between breaths to give values at 1-s intervals and fit to a curve using a three-component exponential model starting at the onset of exercise. The computer model utilized to describe the kinetic response provides an estimate of the amplitude of baseline \( \dot{V}O_2 \) kinetics (\( A_0 \)), amplitudes of phases I, II, and III of \( \dot{V}O_2 \) kinetics (\( A_1, A_2, \) and \( A_3 \); time delays of phases I, II, and III of \( \dot{V}O_2 \) kinetics (\( \tau_1, \tau_2, \) and \( \tau_3 \)). The phase fitting parameters were set to achieve a completed response before the start of phase II, thus allowing for complete comparison with other investigations that have omitted the comparison with other investigations that have omitted the part of the muscle belly. Before electrode placement, the skin was shaved and cleaned with alcohol. The electrodes and cables were then taped firmly in place to reduce artifact and to maintain position throughout the entire experiment. The myoelectrical signal was band-pass filtered (20–500 Hz), differentially amplified (gain 2,000 times), input impedance 2 M\( \Omega \), and sampled at 1,024 Hz during the last 15 s of each minute of exercise and continuously during the Wingate tests. The raw EMG signals were then converted from analog to digital by using a 12-bit analog-to-digital conversion box (MacByte DAS-16). The digitized signal was displayed on computer using collection software and saved for later analysis.

\( \dot{V}O_2 = A_0 + A_1[1 - e^{-[t - TD_1]/\tau_1}] + A_2[1 - e^{-[t - TD_2]/\tau_2}] + A_3[1 - e^{-[t - TD_3]/\tau_3}] \)

where

\( \mu_1 = 0 \) for \( t < TD_1 \) and \( \mu_1 = 1 \) for \( t \geq TD_1 \)

\( \mu_2 = 0 \) for \( t < TD_2 \) and \( \mu_2 = 1 \) for \( t \geq TD_2 \)

\( \mu_3 = 0 \) for \( t < TD_3 \) and \( \mu_3 = 1 \) for \( t \geq TD_3 \)

Model parameters were determined by least squares nonlinear regression (except phase I as noted above) in which the best fit was defined by minimization of the residual sum of squares. The overall time course of the response was determined from the mean response time (MRT). The MRT is equivalent to the time required to achieve \( \sim 63\% \) of the difference between \( A_0 \) and the final \( \dot{V}O_2 \) value as previously used (31).

Beat-by-beat data for cardiac output were averaged every five heartbeats and were fit to a curve by using a two-component exponential model starting at the onset of exercise. This fitting procedure allowed for the best-fit estimate and minimized random error. The values obtained at the end of each minute from the curve fitting were used to evaluate the cardiac output response profile for each subject.
The raw EMG signals were processed off-line (34) by full-wave rectification and digital low-pass filtering with a second-order Butterworth filter (cutoff frequency = 4 Hz) to produce a linear envelope. Five sequential bursts were then selected for each signal and integrated (iEMG). Mean power frequency (MPF) was determined using a fast Fourier transform algorithm over the 15-s window (cutoff frequency 512 Hz). Similar methods evaluated the EMG during the Wingate cycling. In this case, data were binned in 5-s intervals starting between 5 and 10 s of exercise and then expressed as a ratio relative to the power output in that same interval. Normalization of data in the constant-load tests was to the values at the first minute of the first exercise bout for each subject. In the Wingate tests, data were normalized to the second 5-s interval of the first all-out sprint. Muscle activation was evaluated for each muscle separately.

Mean values of VO₂, VC0₂, HR, and VE were calculated during the final 2 min before the step increase in work rate, from 5 s before to 5 s after 3 min and during the last 10 s of exercise. In addition to the curve-fitting procedure, the VO₂ slow component was also computed as the difference in VO₂ between minutes 3 and 6 (ΔVO₂ e.g., 2) (18).

Statistical analysis. For simple comparisons of values that were present in only one condition for pre- vs. post-Wingate, paired t-tests were computed. This included analysis of respiratory variables and HR changes on baseline and at minutes 3 and 6. Cardiac output means were compared by using the nonparametric Wilcoxon test. Significance was set at P < 0.05. All data are presented as means ± SE.

The constant-load cycling EMG response was evaluated by a two-way repeated-measures ANOVA on the effects of condition (i.e., pre- and post-Wingate) and time. Both power output and the ratio of EMG to power output from the three Wingate tests were also evaluated by two-way repeated-measures ANOVA. If the normality or the equality of variance failed, the nonparametric Friedman test was used for post hoc comparisons.

RESULTS

Incremental test. The VO₂ peak of 65.5 ± 0.8 ml·min⁻¹·kg⁻¹ was associated with a peak work rate of 441.2 ± 9.7 W during the 30 W/min ramp incremental test. VT occurred at 76.5 ± 1.6% of VO₂ peak.

Wingate tests. The power peak decreased during the three successive Wingate tests (705.4 ± 27.9, 638.5 ± 29.3, and 598.8 ± 26.1 W; P < 0.05). Power output declined during each successive Wingate test (Fig. 1; P < 0.05), and the ratio of EMG to power output was increased with time and from bout to bout in certain muscles (see Fig. 2).

Surface EMG during heavy exercise. Normalized MPF and iEMG (Fig. 3) did not change significantly with time within each exercise bout. Nor were there significant changes in MPF or iEMG between pre- and post-Wingate bouts of heavy exercise. The maximum duration for MPF was <5% from the value observed at the first minute of the first exercise bout (data not shown).

Heavy cycling exercises. VO₂ measured at the end of the pre- and post-Wingate heavy exercises correspond to 97.8 ± 2.5 and 97.0 ± 1.9% of the VO₂ peak, respectively (P > 0.05). Because the VO₂ tended toward VO₂ peak, this exercise might be categorized as severe (36). The time courses of increase in VO₂ for one subject (Fig. 4) and the group mean responses (with 95% confidence intervals, Fig. 5) reveal the faster VO₂ response in the post-Wingate tests. The model parameters for curve fitting of the VO₂ response to heavy exercise in pre- and post-Wingate are summarized in Table 1. After the Wingate tests, a significant (P < 0.05) increase in A₀ was found compared with pre-Wingate (Table 1). A₁ was not significantly affected by prior sprint exercise. The t₂ was significantly faster, by 23% (P < 0.05), after the 30-s sprint repetitions. Neither A₂ nor the sum of A₁ + A₂ was affected, but VO₂ was significantly greater at 3 min in post-compared with pre-Wingate tests (+124 ml/min; Table 2). The 3-min VO₂ was influenced by the elevated A₀ and the faster t₂ as well as the earlier onset of phase III (TD₃) in the post-Wingate bout of heavy exercise (Table 1). There were no differences in A₃ or t₃ between the two bouts of heavy exercise, but the mean response time obtained as a weighted average of the time constants was significantly faster post-Wingate (Table 1). End-exercise VO₂ was not different between exercise bouts. The ΔVO₂ e.g., was ~30% less in post-compared with pre-Wingate tests (300.6 ± 55.6 vs. 210.2 ± 92.4 ml/min, respectively; P < 0.05).

Pre- to post-Wingate comparisons at three different times (baseline, 3rd min, end exercise) showed significant changes in VE, HR, and VC0₂ (Table 2). During the 2-min baseline period preceding heavy exercise, HR was significantly higher in post-Wingate, whereas VE and VC0₂ were not significantly different. At the third minute in post-Wingate test, VO₂, VE, and HR were all higher, whereas VC0₂ was lower compared with pre-Wingate test. During the last minute of the post-Wingate test, despite the significant increase in VE the VO₂ was still significantly lower. HR in the last minute of exercise was not different between pre- and post-Wingate test. The different time course for VC0₂ is plotted for pre- and post-Wingate tests in Fig. 5.
Cardiac output was significantly greater during the baseline and the first 2 min of heavy exercise but not thereafter (Table 3).

DISCUSSION

This study was designed to investigate the effects of preexisting metabolic acidosis on the adaptation of \( \dot{V}O_2 \) at the onset of a second bout of heavy constant-load exercise. The study was also designed to investigate the effects of prior fatiguing exercise on the \( \dot{V}O_2 \) and muscle activity in this second bout of exercise. In support of our first hypothesis, we observed that the time constant (\( \tau_2 \)) for \( \dot{V}O_2 \) in the second bout of exercise was significantly faster than in the first bout. Taken together with the recent observation by Rossiter et al. (41) of faster \( \dot{V}O_2 \) kinetics in the second of two bouts of knee extension exercise, our data are in contrast with the conclusion of several recent studies using slightly different exercise models in that the elevated \( \dot{V}O_2 \) was not associated with faster \( \tau_2 \) (11–13). Our second hypothesis was partially supported by the results because the phase III response started earlier in the second

Fig. 2. Ratio of integrated muscle electromyogram (iEMG) to power output within 5-s windows normalized to the value between 5 and 10 s in the first repetition of 30-s all-out sprint cycling (Wingate test). Values are means \pm SE for Wingate test 1 (■), test 2 (○), and test 3 (▲). A: vastus lateralis (VL). B: rectus femoris (RF). C: vastus medialis (VM). D: gastrocnemius medialis (GM). Increase in iEMG/power output between tests was different at specified times as indicated (*\( P < 0.05 \) relative to Wingate test 1, #\( P < 0.05 \) relative to Wingate test 2).

Fig. 3. iEMG normalized to values at first minute of the first bout of heavy exercise for each of VM, VL, RF, and GM. Values are means \pm SE. There were no differences within a bout as a function of time or between bouts of repeated heavy exercise. Values are offset slightly at each time point for clarity.

Fig. 4. Superimposed \( O_2 \) uptake (\( \dot{V}O_2 \)) data for a single subject from heavy exercise bout 1 (○) and bout 2 after three repetitions of 30-s all-out sprint cycling (■) to show more rapid adaptation in bout 2.
bout of exercise, but the prior fatiguing exercise had no significant effect on the indicator of muscle activation from the EMG signal. This latter observation was similar to that of a recent study that investigated a single muscle during repeated heavy exercise (42).

It has been consistently observed for several different exercise models that VO2 is elevated during the early phase of a heavy exercise bout when it was performed after previous heavy exercise (11–13, 18, 27, 28, 30, 31, 42). Initial reports from studies of leg cycling exercise similar to the present study did not confirm that this was a consequence of faster phase II kinetics after prior heavy exercise (10, 13, 27, 42). We incorporated three bouts of all-out sprint cycling between the two bouts of heavy exercise (18, 31). Both of these indicators were also significantly reduced in the second exercise bout in the present study. Several recent studies did not find significant acceleration of phase II kinetics after prior heavy exercise (10–13, 27, 42). We incorporated three bouts of all-out sprint cycling between the two bouts of heavy exercise to induce a greater metabolic acidosis (32, 44) that we reasoned might enhance vasodilata-

tion and O2 extraction at the onset of the second bout of exercise. Three bouts of sprint cycling rather than one bout as in a recent study (12) would have caused a greater and more sustained increase in plasma lactate. Whereas Burnley et al. (12) observed plasma lactate to be 5.6 mmol/l after their single bout of exercise, McCartney et al. (32) found plasma lactate to remain high (~20 mmol/l) for at least 20 min after repeated bouts of 30-s all-out sprint cycling. With the same exercise model, Spriet et al. (44) observed muscle H+ to increase from baseline of 195 ± 12 mmol/l to 274 ± 19 after the first bout of sprinting to 315 ± 24 (i.e., corresponding pH = 6.50) after the third bout of sprint cycling.

Previously, Gerbino et al. (18) and MacDonald et al. (31) postulated that enhanced O2 delivery and possibly a right shift of the O2-hemoglobin dissociation curve in the second bout of heavy exercise would supply more O2 and allow faster adaptation of oxidative metabolism. Although we do not have data for leg blood flow in this study, two lines of evidence would support the proposal that O2 delivery has increased. First, we did find significantly elevated HR and estimated cardiac output throughout the early phase of the second bout of exercise that were consistent with elevated bulk O2 transport. Second, two recent studies that examined arm (30) and leg exercise (28) observed that higher muscle VO2 in the second bout of exercise was a consequence of both higher blood flow and greater O2 extraction early in the second bout of exercise. A potential increase in delivery of O2 along with probable changes in muscle enzymes and/or metabolic intermediates (22) certainly contributed to our observation of faster $\tau_2$. Because we did not measure blood flow or its distribution we cannot exclude the possibility that temperature effects caused greater blood flow to skin for thermoregulation.

It was important to optimize experimental conditions to determine whether $\tau_2$ would be altered (41). Recently, Hughson et al. (25) demonstrated from a computer simulation model that it is extremely difficult to detect either dynamic nonlinearities or differences in time constant even when they are present. It is possible that the recent investigations that did not find an effect on $\tau_2$ (11–13, 27, 42) either did not have enough residual effect on local vasodilator influences to Table 1. VO2 kinetics parameters during heavy exercise pre- and post-Wingate tests

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre-Wingate</th>
<th>Post-Wingate</th>
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<tr>
<td>A0, ml/min</td>
<td>970.5 ± 37.9</td>
<td>1,119 ± 50.3*</td>
</tr>
<tr>
<td>A1, ml/min</td>
<td>666.8 ± 50</td>
<td>660.0 ± 45.4</td>
</tr>
<tr>
<td>TD1, s</td>
<td>1.1 ± 0.4</td>
<td>1.2 ± 0.5</td>
</tr>
<tr>
<td>$\tau_1$, s</td>
<td>1.0 ± 0.1</td>
<td>0.9 ± 0.03</td>
</tr>
<tr>
<td>A2, ml/min</td>
<td>2,352.6 ± 113.1</td>
<td>2,288.7 ± 87.7</td>
</tr>
<tr>
<td>TD2, s</td>
<td>10.9 ± 0.8</td>
<td>9.3 ± 0.4</td>
</tr>
<tr>
<td>$\tau_2$, s</td>
<td>28.9 ± 2.4</td>
<td>22.2 ± 1.7*</td>
</tr>
<tr>
<td>A1 + A2, ml/min</td>
<td>3,020.4 ± 97.9</td>
<td>2,928.7 ± 65.1</td>
</tr>
<tr>
<td>A3, ml/min</td>
<td>624.0 ± 89.4</td>
<td>534.4 ± 44.8</td>
</tr>
<tr>
<td>TD3, s</td>
<td>120.9 ± 6.4</td>
<td>96.0 ± 3.1*</td>
</tr>
<tr>
<td>$\tau_3$, s</td>
<td>154.2 ± 17.1</td>
<td>127.1 ± 12.2</td>
</tr>
<tr>
<td>MRT, s</td>
<td>73.6 ± 7.3</td>
<td>56.1 ± 3.8*</td>
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</table>

Values are means ± SE for 8 subjects. A0, amplitude of baseline O2 uptake (V02) kinetics; A1, A2, and A3, amplitude of phase I, II, and III of VO2 kinetics, respectively; $\tau_1$, $\tau_2$, and $\tau_3$, time constants of phase I, II, and III of VO2 kinetics, respectively; TD1, TD2, and TD3, time delay of phase I, II, and III of VO2 kinetics, respectively; MRT, mean response time. *Significantly different from pre-Wingate, P < 0.05.
achieve an effect, that an effect was present but was undetectable by the curve-fitting methods employed, or that the studies had insufficient statistical power to observe an effect. The observation that even heavy-intensity prior arm exercise has an effect on subsequent heavy leg exercise VO₂ kinetics (8) suggests that changes in arterial blood acid-base can have an impact on cardiac output and/or local vasodilatation at the onset of exercise and that the effect, at least with the prior arm exercise, can be independent of altered muscle metabolic factors. Another argument often used to claim that T₂ is independent of O₂ delivery is the comparison between heavy (above ventilatory threshold) vs. moderate (below threshold) intensities of exercise (11, 13, 42). Some studies have shown very markedly slower mean values for T₂ with heavy compared with moderate exercise [e.g., 24% (14) to 38% (42)] or progressive lengthening with heavier exercise (6), but they failed to find statistical significance. These observations again point to limited sensitivity of kinetic analysis (25) especially when the response becomes nonlinear with heavy exercise (41).

The present study and the recent investigation by Rossiter et al. (41) were designed to examine in detail the time constant for VO₂ during the phase II response. Both investigations found significant acceleration of phase II time constant by prior heavy exercise. Other research that observed elevated VO₂ but without faster phase II kinetics was taken to indicate an unchanged response on top of an existing baseline of higher VO₂ (13, 42). To account for this elevated baseline, Burnley et al. (13) subtracted the baseline for both first and second exercise bouts. The problem with this analysis was that end-exercise VO₂ was reduced in the second bout, suggesting improved efficiency and VO₂ recovered after the second bout of exercise to values below the baseline. More recently, Burnley et al. (11) allowed baseline VO₂ to recover completely by interposing 12-min of recovery rather than 6 min. Again they observed elevated VO₂ at the onset of the second bout of exercise but it was attributed to greater amplitude of phase II rather than faster kinetics. The mechanism proposed for this was that a greater number of muscle fibers were recruited early in exercise (10) and that oxidative metabolism in these fibers continued to adapt at the same rate, assumed to be independent of O₂ but limited by metabolic control factors.

One objective of the present research was to investigate muscle recruitment patterns through surface EMG. If we first consider the EMG pattern during the completion of the Wingate tests, the characteristic pattern of muscle fatigue is evident. Within each 30-s all-out sprint, the ratio of iEMG to power output increased progressively, suggesting progressive fatigue of muscle fibers. In contrast with the findings from the successive all-out sprints, observations during the constant-load cycling tasks did not provide evidence of altered muscle function even after the fatiguing all-out sprints. Throughout the first constant-load heavy cycling bout, normalized iEMG did not deviate from the value observed at the end of the first minute of exercise. In the second bout of heavy exercise after the three 30-s all-out sprints, iEMG tended to be greater than that in the first bout, which would have been consistent with residual fatigue, but individual variability precluded statistically significant findings. As in the first exercise bout, there were no significant changes in iEMG over time within the exercise bout. The MPF of the EMG also showed a small trend to lower values, again a potential indicator of fatigue, but the difference was not statistically significant. The EMG is an indirect method of assessing muscle fiber recruitment. The limitations of the technique might be responsible for the variable observations where some researchers (9, 10, 43) have found changes in EMG corresponding to the onset of the phase III slow component. Other researchers did not find any effects on the EMG to correspond with the onset of the slow component during prolonged heavy exercise in trained cyclists (29) or during repeated bouts of heavy exercise (42). The fact that our subjects were well-trained cyclists and we were able to monitor only four muscles meant that any subtle difference in muscle recruitment

Table 2. Respiratory variables and heart rate changes during heavy exercise pre- and post-Wingate tests

<table>
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<th></th>
<th>Pre-Wingate</th>
<th>Post-Wingate</th>
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<tr>
<td></td>
<td>BL</td>
<td>3 min</td>
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</table>
| VO₂, ml/min      | 1,024 ± 40.5| 4,160 ± 127.3| 4,461 ± 110.6| 1,157 ± 51.1*| 4,284 ± 120.5*| 4,494 ± 108.5*
| VE, l/min        | 27.7 ± 1.4  | 124.8 ± 5.8  | 142.5 ± 8.5  | 35.5 ± 4.1  | 143.4 ± 9.5* | 184.5 ± 8.1* |
| VCO₂, ml/min     | 930.7 ± 29.7| 4,607.9 ± 152.4| 4,718.3 ± 146.8| 868.7 ± 55.5| 4,146.5 ± 83.0*| 4,519.7 ± 139.3*|
| HR, beats/min    | 79.1 ± 4.4  | 160.6 ± 4.3  | 174.0 ± 4.4  | 111.5 ± 3.1*| 170.4 ± 2.8* | 177.1 ± 3.4  |

*Values are means ± SE for 8 subjects. BL, baseline level; VE, minute ventilation; HR, heart rate; VCO₂, carbon dioxide output.

Table 3. Evolution of cardiac output in pre- and post-Wingate tests normalized to baseline values before the first bout of heavy exercise

<table>
<thead>
<tr>
<th></th>
<th>Pre-Wingate</th>
<th>Post-Wingate</th>
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<tbody>
<tr>
<td></td>
<td>BL, minute 3, and minute 4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Minute 1</td>
<td>2.22 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>Minute 2</td>
<td>2.35 ± 0.12</td>
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<tr>
<td></td>
<td>Minute 3</td>
<td>2.43 ± 0.13</td>
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<td></td>
<td>Minute 4</td>
<td>2.48 ± 0.13</td>
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<td></td>
<td>Minute 5</td>
<td>2.53 ± 0.14</td>
</tr>
<tr>
<td></td>
<td>Minute 6</td>
<td>2.56 ± 0.15</td>
</tr>
</tbody>
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*Values are means ± SE; n, no. of subjects. *Significantly different from pre-Wingate, P < 0.05.
strategies probably introduced sufficient variability in individual muscle or combined muscle activity patterns to prevent us from observing the anticipated signatures of muscle fatigue.

Oxidative metabolism in the transition from rest or light to heavier exercise is regulated by the interaction of O$_2$ and biochemical factors (45). To date the details of this interaction are not completely understood. In the human calf muscle, Richardson et al. (39) observed by magnetic resonance spectroscopy that intramuscular P$_{O_2}$ dropped within 20 s to values $<$5 Torr and that this value was relatively constant across a wide range of work rates. If it can be assumed that similar low, or lower, values occurred during heavy cycling exercise, then it is necessary for the biochemical factors that determine flux rate for ATP formation to adapt by increasing the phosphorylation potential (1, 46). The recent observations of Rossiter et al. (41) of less reduction of muscle phosphocreatine concentration in the second bout of heavy exercise coincided with faster V$_{O_2}$ kinetics and were consistent with less disturbance of the phosphorylation potential as a consequence of prior heavy exercise. Recent animal experiments have also explored models of two bouts of electrical stimulation. In the isolated Xenopus muscle fiber there was a faster decline in intracellular P$_{O_2}$ in the second compared with the first bout of stimulation, but this was due to a shorter delay to the onset of depletion of O$_2$ rather than faster time constant (22). These results suggested that a metabolic factor was modified by the previous stimulation. Observations of microvascular P$_{O_2}$ in rat spinotrapezius muscle also found shorter time to onset of depletion of O$_2$ in a second exercise bout of electrical stimulation (7). Unfortunately, in this latter study, muscle blood flow could not be measured in the transition from rest to stimulation so no comments could be made about O$_2$ delivery except in the steady state except that the ratio of O$_2$ delivery to O$_2$ utilization did not change. Also this rat muscle model differed in that there was no preexisting metabolic acidemia or elevated blood flow (7). These animal experiments differ from observations in human muscle where the onset of oxidative metabolism at the onset of heavy or severe exercise was demonstrated to be very rapid (3). The more rapid adaptation of blood flow and O$_2$ transport in the second heavy exercise bout has parallels in other exercise models. When blood flow was higher in the early phase of forearm exercise by placing the arm below rather than above the heart (24) or by elevating mean arterial pressure before exercise (35) muscle V$_{O_2}$ adapted more rapidly. An additional biochemical adaptation that might have contributed to the increased V$_{O_2}$ in the second bout of exercise was the higher level of activity of the pyruvate dehydrogenase complex due to residual effects of prior exercise (19). Whether this would have allowed faster V$_{O_2}$ kinetics without a coincident increase in O$_2$ delivery is not known. Recent experiments achieving greater activation of pyruvate dehydrogenase complex by dichloroacetate before the start of heavy exercise in dogs (20) and humans (2) found no acceleration of muscle V$_{O_2}$.

Direct measurements of muscle V$_{O_2}$ across working forearm (30) or knee-extensor muscles (28) have confirmed that the elevated V$_{O_2}$ in a second bout of heavy to intense exercise occurred at the working muscle. In both of these experiments, muscle blood flow was higher and O$_2$ extraction was greater early in the second bout of exercise. These findings confirmed the original hypothesis of Gerbino et al. (18). Interestingly the maximum O$_2$ extraction in the two experiments was relatively low, reaching peak values of $<$155 ml/l (28, 30). Krstrup et al. (28) observed that once this peak value of extraction in these experiments was reached, any further increase in muscle V$_{O_2}$ followed an increase in muscle blood flow.

Heavy prior exercise has a major impact on metabolism in the subsequent exercise bout (18, 28). The greater oxidative metabolism early in the second bout allowed markedly reduced anaerobic energy production (28). The consequence of this, perhaps in conjunction with a greater percentage of fat metabolism and altered metabolic respiratory quotient (21), was that V$_{O_2}$ was markedly lower throughout the entire second exercise bout. Changes in CO$_2$ storage between the first and second bouts of exercise also contributed to the different time courses for adaptation of V$_{O_2}$ in heavy exercise. CO$_2$ stores would have decreased with the metabolic acidosis of the first exercise bout (23). CO$_2$ stores would have been further decreased after the repeated 30-s all-out sprint cycling also as a function of the increased V$_{E}$ that contributed to the respiratory compensation for the metabolic acidosis. The mean value for V$_{CO_2}$ in the second heavy exercise bout barely reached the lower 95% confidence interval of the first exercise bout. This observation might indicate that the elevated lactate from the repeated sprint cycling acted as a substrate for oxidative metabolism in the second bout, reducing the metabolic acidosis. This speculation remains to be confirmed under these experimental conditions.

In summary, the data from the present experiments demonstrated that for heavy cycling exercise, $T_2$ was significantly faster when the exercise was preceded by three bouts of all-out sprint cycling. This prior intense exercise would have established a marked metabolic acidosis within the exercising muscles that remained through to the start of the subsequent heavy exercise bout (44). The acidosis probably contributed to enhanced vasodilatation early in exercise so that increased muscle blood flow and O$_2$ delivery were supported by the higher HR and cardiac output in the first minutes of the heavy exercise. The prior exercise and intracellular acidosis probably also affected metabolic pathways and substrates for oxidative phosphorylation so that these too contributed to the more rapid adaptation of V$_{O_2}$. Thus consistent with the initial hypothesis of Gerbino et al. (18) and contrary to the conclusions of some recent investigations (13, 27), the more rapid adaptation of oxidative metabolism in heavy exercise that followed prior heavy or intense exercise was a function of improved O$_2$ delivery. The functional significance of our results is that we have confirmed...
the nonlinear characteristics of VO2 during the phase II response in the heavy exercise domain (18). From a physiological perspective our observations of whole body VO2 suggest that adaptation of oxidative phosphorylation at the onset of heavy exercise is dependent on at least two factors that we have proposed previously (45) to be the interaction between O2 supply and O2 utilization (i.e., biochemical) mechanisms (1, 45). Surface recording of EMG from four different leg muscles was unable to detect significant differences in fiber recruitment patterns in these highly trained cyclists that might have coincided with the onset of the VO2 slow component.

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REFERENCES


