Effects of prior heavy exercise on \( \dot{V}O_2 \) kinetics during heavy exercise are related to changes in muscle activity

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Burnley, Mark, Jonathan H. Doust, Derek Ball, and Andrew M. Jones. Effects of prior heavy exercise on \( \dot{V}O_2 \) kinetics during heavy exercise are related to changes in muscle activity. J Appl Physiol. 93: 167–174, 2002; 10.1152/japplphysiol.01217.2001.—We hypothesized that the elevated primary \( \dot{V}O_2 \) response without a change in the time constant of the primary response (i.e., in the first 2 min of exercise). We speculated that the elevated primary \( \dot{V}O_2 \) amplitude observed after heavy exercise is related to a greater recruitment of motor units at the onset of exercise. \( \dot{V}O_2 \) uptake primary component; \( \dot{V}O_2 \) uptake slow component; electromyogram; warm-up

GERBINO ET AL. (12) originally observed that prior heavy exercise (above the lactate threshold) could significantly speed the overall \( \dot{V}O_2 \) uptake (\( \dot{V}O_2 \)) kinetics during heavy exercise. These authors suggested that the residual lactic acidosis at the onset of the second heavy exercise bout may have led to vasodilatation and improved \( O_2 \) delivery: alleviation of an \( O_2 \) delivery limitation then led to a speeding of the \( \dot{V}O_2 \) kinetics. These data and interpretations were supported by MacDonald et al. (22), who showed that both prior heavy exercise and inspiratory hyperoxia could reduce the “mean response time” during heavy exercise. However, a number of recent studies, which partitioned the \( \dot{V}O_2 \) response into its three identifiable kinetic components, found no evidence of a speeding of the primary \( \dot{V}O_2 \) kinetics (7, 10, 11, 20, 34). Instead, the speeding of the overall \( \dot{V}O_2 \) kinetics noted by Gerbino et al. and MacDonald et al. could be attributed to a reduction in the amplitude of the \( \dot{V}O_2 \) slow component (11). These recent reports suggest that \( O_2 \) delivery is not limiting at the onset of heavy exercise and that some other mechanism is responsible for the characteristic effect of prior heavy exercise on the \( \dot{V}O_2 \) response profile.

Our laboratory’s most recent work on this issue (10) demonstrated that the reduction in the amplitude of the \( \dot{V}O_2 \) slow component during a second bout of heavy exercise was preceded by an increase in the amplitude of the primary \( \dot{V}O_2 \) response without a change in the primary \( \dot{V}O_2 \) kinetics. We speculated that the elevated primary \( \dot{V}O_2 \) amplitude observed after heavy exercise may have been the result of increased motor unit recruitment at the onset of heavy exercise. However, to date, this hypothesis remains untested. Recent work by Schuermann et al. (34) suggests that patterns of motor unit recruitment do not play a significant role in the development of the \( \dot{V}O_2 \) slow component or in the effects of prior heavy exercise. One limitation of this study, acknowledged by the authors, was the measurement of the integrated electromyogram (iEMG) of a single muscle group to infer the muscle activity responsible for power production during cycling.

Therefore, the aim of this study was to test the hypothesis that the elevated primary \( \dot{V}O_2 \) amplitude during the second of two bouts of heavy exercise is accompanied by an increase in iEMG during the primary phase of the response (i.e., in the first 2 min of exercise).

METHODS

Eight healthy male subjects (mean ± SD, age 24 ± 4 yr; height 1.79 ± 0.04 m; mass 74.0 ± 5.1 kg; peak \( \dot{V}O_2 \) 51 ± 8 ml·kg\(^{-1}\)·min\(^{-1}\)) gave written informed consent to partici-
pate in this study, which was approved by the ethics commit-
tee of the Manchester Metropolitan University.

**Experimental design.** The subjects initially performed a
ramp exercise test to estimate the ventilatory threshold (VT) and
determine peak Vo2 (Vo2 peak) (see below). Subsequently,
on two separate occasions, the subjects performed two 6-min
bouts of heavy leg cycle exercise separated by 12-min recov-
ery. On one of the test days, “noninvasive” data [near-infra-
red spectroscopy (NIRS) and electromyography (EMG)] were
acquired. On the other test day, “invasive” data (muscle pH and
arterialized venous blood) were acquired. Pulmonary gas
exchange and heart rate (HR) were measured throughout all
exercise tests, as described below.

**Measurement of VT and Vo2 peak.** The VT and Vo2 peak were
determined by using a ramp exercise test (30 W/min) per-
fomed to the limit of tolerance on an electrically braked cycle
ergometer (Ergoline, Jaeger). Subjects self-selected a cadence
of 70–90 rpm and maintained this throughout all subsequent
exercise tests. The breath-by-breath data were collected and
displayed at 10-s intervals. The VT was determined visually
as an increase in CO2 production relative to Vo2. Vo2 peak was
taken as the highest 10-s value recorded before volitional
exhaustion. The intensity of the heavy leg exercise bouts was
set at a power output that would elicit a V˙O2 that was 70% of
the difference between VT and Vo2. This power output was
chosen to maximize the potential effect of prior exercise on
the response parameters measured.

**Experimental protocol.** The protocol was similar in design
to that of Burnley et al. (10). The subjects performed two
“square-wave” bouts of heavy exercise of 6-min duration,
separated by 12 min of recovery. The heavy exercise bouts
were each preceded by 3 min of baseline pedaling (at 20 W,
the lowest power output available on the cycle ergometer),
and pulmonary gas exchange was measured breath by breath
throughout exercise. This protocol was performed twice.

During one test session, NIRS and EMG data were
acquired. The oxygenation status of the right vastus lateralis
muscle at approximately midthigh was monitored by use of a
commercially available NIRS system (Omron Heo-200). The
system consisted of a probe attached to a data logger that
received NIRS signals at 2 Hz. The probe consisted of two
light-emitting diodes and two photodetectors, which detected
photons at wavelengths of 840 [oxyhemoglobin (HbO2)] and
760 nm (Hb). The penetration depth when using these probes
for each muscle was placed over inert tissue (tendon, liga-
 ment, or bone). The electrodes were attached via preamplifi-
ers to a telemetry belt that relayed the data to an analog-to-
digital converter. All wiring connecting the electrodes to the
belt were taped down to prevent movement artifact. EMG
recordings, of 30-s duration, began 45 s before exercise onset,
at exercise onset, and every 45 s thereafter during both heavy
exercise bouts. The signals detected by the electrodes were
preamplified with a gain of 4,000 and a frequency band of
20–500 Hz. The EMG signal was sampled at a frequency of
1,000 Hz and underwent 12-bit analog-to-digital conversion.
The digital signal was first displayed raw and subsequently
full-wave rectified and integrated over 10-ms intervals. The
iEMG was calculated over each 30-s sampling period, and all
iEMG data were normalized to the value recorded in the
unloaded pedaling phase 30 s before the onset of the first
bout of leg exercise. All iEMG data were, therefore, expressed
as a percentage of the initial unloaded pedaling value. The
normalized iEMG of the individual muscles was summed and
averaged to provide an estimate of “total” muscle activity
during exercise. The first three EMG data sets acquired
during the heavy exercise bouts were used as an index of
muscle activity in the primary phase of the response to
exercise (i.e., muscle activity in the first 2 min of exercise).
The mean power frequency (MFP) was calculated as the
mean frequency generated by the fast Fourier transform of
the sampled EMG signal.

During the other test session, muscle pH was measured
before each of the heavy exercise bouts by using a needle-
tipped pH electrode, which was inserted 3 cm into the right
vastus lateralis muscle through an incision made under local
anesthetic (3–4 ml of 1% lignocaine). The pH meter (Corning
245 pH meter) was calibrated before each test and checked
by using distilled water. Muscle pH was determined −1 min
before the period of unloaded cycling began for both exercise
bouts. Arterialized venous blood (~2.5 ml) was sampled from
the dorsum of a heated hand by using a 21-gauge butterfly
cannula at rest, at time 0 (the onset of exercise) and at 2, 4,
and 6 min of exercise. The cannula tubing was flushed be-
 tween each sample with saline. These samples were imme-
diately capped, stored in an ice slurry, and analyzed in
duplicate for pH by use of an automated blood-gas analyzer
(CIBA-Corning 278 blood gas system, Medfield, MA) within
1 h of collection. The remainder of the sample was analyzed
in duplicate for blood lactate concentration (lactate); YSI
1500, Yellow Springs Instruments, OH).

**Measurement of pulmonary gas exchange and HR.** Pulmo-
 nary gas exchange was measured breath by breath during all
exercise tests. Subjects breathed through a low-resistance
turbine volume transducer (Jaeger Triple V), which had a
dead space of 90 ml. Gas was continuously drawn down a
capillary line into rapid-response gas analyzers (Jaeger Oxy-
con Alpha, Hoechberg, Germany). Vo2, carbon dioxide pro-
duction, and minute ventilation were calculated and dis-
played breath by breath once the delay between the volume
and concentration signals had been accounted for. The vol-
ume transducer was calibrated before each test with a 3-liter
calibration syringe (Hans Rudolph), and the analyzers were
calibrated with gases of known concentration. HR was re-
corded every 5 s by using short-range radio telemetry (Polar
Sports Tester, Kempele, Finland). The O2 pulse was calcu-
lated in the unloaded pedaling baseline and after 2 min and
at the end of exercise as absolute Vo2/HR.

**Data analysis.** The breath-by-breath data were linearly
interpolated to provide second-by-second values. For each
subject, the two performances of each protocol were time
aligned and averaged to provide one set of second-by-second
data for each variation of the protocol. The time course of the
Vo2 response after the onset of exercise was described in
terms of a three-component exponential function, using ite-
rat ive nonlinear regression techniques in which minimizing
the sum of squared error was the criterion for convergence, using purpose-built fitting software. Each exponential curve was used to describe one phase of the response. The first phase began at the onset of exercise, whereas the other terms began after independent time delays (5)

\[ \dot{V}_{O_2}(t) = \dot{V}_{O_2}(b) + A_c \cdot (1 - e^{-t/\tau_c}) + A_p \cdot (1 - e^{-(t-\tau_p)/\tau_p}) + A_s \cdot (1 - e^{-(t-\tau_d)/\tau_d}) \]

where \( \dot{V}_{O_2}(b) \) is the baseline \( \dot{V}_{O_2} \) measured in the 3 min preceding the onset of exercise; \( A_c, A_p, \) and \( A_s \) are the amplitudes of the exponential curves fitting the cardiodynamic, primary, and slow components, respectively; \( \tau_c, \tau_p, \) and \( \tau_s \) are the time constants; \( t \) is time; and \( \tau_d \) and \( \tau_s \) are the time delays. The cardiodynamic component was terminated at \( \tau_d \) and given the value for that time (defined \( A_c \)). The amplitude of the primary response \( (A_p) \) was defined as the increase in \( \dot{V}_{O_2} \) from baseline to the asymptote of the primary component. The absolute amplitude of the primary \( \dot{V}_{O_2} \) response was calculated as the sum of baseline \( \dot{V}_{O_2} \) and \( A_p \). The amplitude of the \( \dot{V}_{O_2} \) slow component was determined as the increase in \( \dot{V}_{O_2} \) from \( \tau_d \) to the end of exercise (defined \( A_s \)), rather than from the asymptotic value \( (A_s) \), which may project beyond the value at 6 min (end exercise).

Statistical analysis. The responses to the square-wave bouts of heavy exercise were compared by paired t-tests. The iEMG data series was analyzed by using a two-way (exercise primary, and slow components, respectively; ph and ph, time constants of primary and slow components, respectively; \( A_p \) and \( A_s \), amplitude of primary response and slow component, respectively. *Significantly different from first heavy exercise bout \((P < 0.05)\) and blood pH was reduced \((P < 0.01)\) after 12 min of recovery. Baseline muscle pH was similar before both exercise bouts \((P = 0.84)\). The increase in blood [lactate] as a result of the performance of heavy exercise is also shown in Table 1. The increase in blood lactate during the second exercise bout was significantly reduced compared with that during the initial bout of heavy exercise \((P < 0.001)\).

The primary \( \dot{V}_{O_2} \) time constant was unaltered by prior heavy leg exercise \((P = 0.36)\), whereas the primary \( \dot{V}_{O_2} \) response amplitude \( A_p \) was significantly increased by prior heavy exercise \((P < 0.001)\). The amplitude of the \( \dot{V}_{O_2} \) slow component was reduced by \( P = 0.001)\). The effects of prior heavy leg exercise on the amplitudes of the \( \dot{V}_{O_2} \) response led to the attainment of a similar end-exercise \( \dot{V}_{O_2} \) for the initial exercise bout and the second heavy leg exercise bout \((P = 0.94)\). The \( \dot{V}_{O_2} \) responses for each subject are shown in Fig. 1.

The temporal profiles of the normalized iEMG responses to the two bouts of heavy exercise are shown in Fig. 2. In the first bout of exercise, the averaged iEMG increased systematically throughout

Table 1. \( \dot{V}_{O_2} \) uptake, blood [lactate], and pH, and muscle pH responses to heavy exercise

<table>
<thead>
<tr>
<th>First Heavy Exercise Bout</th>
<th>Second Heavy Exercise Bout</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline ( \dot{V}_{O_2} ), l/min</td>
<td>0.74 ± 0.05</td>
</tr>
<tr>
<td>Baseline blood [lactate], mM</td>
<td>1.2 ± 0.1</td>
</tr>
<tr>
<td>Baseline muscle pH</td>
<td>7.43 ± 0.01</td>
</tr>
<tr>
<td>( \tau_p ), s</td>
<td>20.7 ± 1.4</td>
</tr>
<tr>
<td>( \dot{V}_{O_2}(b) + A_p ), l/min</td>
<td>2.11 ± 0.12</td>
</tr>
<tr>
<td>( \dot{V}_{O_2}(b) + A_p ) l/min</td>
<td>2.11 ± 0.15</td>
</tr>
<tr>
<td>End exercise ( \dot{V}_{O_2} ), l/min</td>
<td>0.79 ± 0.08</td>
</tr>
<tr>
<td>( \Delta ) Blood [lactate], mM</td>
<td>3.69 ± 0.18</td>
</tr>
</tbody>
</table>

Values are means ± SE. Baseline blood lactate concentration ([lactate]) and pH were determined immediately before the onset of heavy exercise; baseline muscle pH was determined immediately before the onset of unloaded cycling. \( \dot{V}_{O_2} \) uptake; \( \tau_d \) and \( \tau_s \), time delays; \( \tau_c \) and \( \tau_s \), time constants of primary and slow components, respectively; \( A_p \) and \( A_s \), amplitude of primary response and slow component, respectively. *Significantly different from first heavy exercise bout \((P < 0.05)\).
exercise, reaching its peak value at the end of exercise (Fig. 2A). In contrast, the iEMG response to the second bout of heavy exercise tended to be higher in the first 2 min of exercise but then remained essentially unchanged until the end of exercise. A similar pattern was evident in all three muscles (Fig. 2, B–D). The MPF was not significantly different between the first and second bouts of heavy exercise (80.1 ± 0.9 vs. 80.6 ± 1.0 Hz, P = 0.38; Fig. 2). To gain insight into the relationship between the primary $\dot{V}O_2$ amplitude and the iEMG at the onset of exercise, the iEMG in the first 2 min of each bout was averaged and compared. The results of this analysis are shown in Fig. 3A. Prior heavy exercise significantly increased the iEMG in the first 2 min of the second bout of heavy exercise (from 491 to 604% of the unloaded pedaling value, on average; P < 0.05). Furthermore, when the primary $\dot{V}O_2$ amplitude ($\dot{A}_{V}^{\dot{V}O_2}$) was divided by the normalized iEMG (Fig. 3B), there was no difference between the two exercise bouts (P = 0.45), indicating that the increased primary $\dot{V}O_2$ amplitude in the second exercise bout was correlated to an increased EMG.

The NIRS responses are shown in Fig. 4. Figure 4A shows the superimposed relative $O_2$ saturation profiles for the two bouts of heavy exercise. The level of HbO$_2$ was higher before the onset of the second bout of heavy exercise and remained higher throughout the second bout of heavy exercise compared with the initial heavy bout. In both heavy bouts, an initial period of hemoglobin desaturation at the onset of exercise reverted to a...
small and gradual resaturation from 1 min onward. (It should be noted here that NIRS is unable to distinguish between changes in HbO2 and oxymyoglobin because the absorption spectra of Hb and myoglobin are identical, and the relative contribution to the NIRS signal from these two sources is uncertain.) The total Hb (the summation of the Hb and HbO2 signals) is shown in Fig. 4B. Total Hb was higher before the onset of the second heavy exercise bout and remained higher during the first 5 min of exercise. In both bouts, there was evidence of a transient fall in total Hb at exercise onset, reaching a nadir at \( t = 30 \) s. Subsequently, total Hb increased systematically in both bouts until the cessation of exercise.

The HR responses to heavy exercise are shown in Table 2. HR was higher in the baseline period before the second bout of heavy exercise commenced \( (P < 0.001) \), whereas baseline \( \dot{V}O_2 \) pulse \( (\dot{V}O_2/HR) \) was significantly lower \( (P < 0.001) \). After 2 min of exercise, HR was significantly higher after prior leg exercise, although \( \dot{O}_2 \) pulse was similar \( (P = 0.47) \). HR and \( \dot{O}_2 \) pulse were similar at the end of exercise.

**DISCUSSION**

The present study demonstrates that the increased primary \( \dot{V}O_2 \) amplitude observed after heavy cycle exercise is accompanied by an increased iEMG in the first 2 min of exercise, consistent with our hypothesis. A common feature of previous studies, in which the recovery between consecutive heavy exercise bouts has been extended to allow the restoration of baseline \( \dot{V}O_2 \), is an elevation of the primary \( \dot{V}O_2 \) amplitude in the second heavy exercise bout \( (7, 10) \). This effect occurs without a change in the primary \( \dot{V}O_2 \) time constant and is followed by a significant reduction of the \( \dot{V}O_2 \) slow component amplitude as exercise continues \( (10, 11, 20, 34) \). It has been suggested that the elevated primary \( \dot{V}O_2 \) amplitude may be caused by an increase in motor unit recruitment at the onset of exercise \( (10) \). Previous studies have suggested that increased motor unit recruitment may \( (35) \) or may not \( (34) \) contribute to the development of the \( \dot{V}O_2 \) slow component. However, these studies measured only the EMG signal from one muscle (the vastus lateralis). Because cycle exercise involves the activity of most of the lower limb muscle mass for power production \( (16) \), and because the pulmonary \( \dot{V}O_2 \) response to exercise primarily represents muscle oxygen consumption \( (30) \), it is important to measure the EMG from as many muscles as practically possible to relate muscle activity to pulmonary \( \dot{V}O_2 \) \( (31) \).

In the present study, methodological factors such as differences in electrode placement were eliminated by only comparing consecutive bouts of heavy leg exercise after a single preparation process. Three muscles were studied in the present work, and the normalized iEMG values from each were summed and averaged to provide an indication, only, of overall muscle activity during consecutive bouts of cycling exercise. It is acknowledged that this treatment is somewhat arbitrary because the contribution of each of the three muscles to
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Table 2. HR and $O_2$ pulse responses to heavy exercise

<table>
<thead>
<tr>
<th>First Heavy Exercise Bout</th>
<th>Second Heavy Exercise Bout</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline HR, beats/min</td>
<td>79 ± 3</td>
</tr>
<tr>
<td>HR at 2 min, beats/min</td>
<td>156 ± 3</td>
</tr>
<tr>
<td>EE HR, beats/min</td>
<td>176 ± 3</td>
</tr>
<tr>
<td>Baseline $O_2$ pulse, ml/beat</td>
<td>9.7 ± 0.5</td>
</tr>
<tr>
<td>$O_2$ pulse at 2 min, ml/beat</td>
<td>18.6 ± 0.6</td>
</tr>
<tr>
<td>EE $O_2$ pulse, ml/beat</td>
<td>20.5 ± 0.6</td>
</tr>
</tbody>
</table>

Values are means ± SE; HR, heart rate; EE, end-exercise value; *Significantly different from initial bout ($P < 0.05$).

force generation and $O_2$ consumption is not known. The iEMG during the early part of the second bout of heavy exercise was significantly higher compared with the first bout in all three muscles studied. These data do not concur with those of Scheuermann et al. (34), who did not detect any change in the iEMG during exercise at 50% of the change between lactate threshold and peak $V_0_2$. The reason for these divergent findings may reside in the differences in exercise intensity (being higher in the present study) or to the normalization methods employed (Scheuermann et al. normalized their data to the end, rather than the beginning, of exercise). In the present study, the increase in the primary $V_0_2$ amplitude in the second heavy bout was accompanied by a 19% increase in the iEMG during the first 2 min of exercise and no change in the MPF, clearly indicating an increase in motor unit recruitment after prior heavy exercise. It was also of interest that the increase in iEMG with time was less steep in the final 3 min of the second bout of heavy exercise (Fig. 2) commensurate with the reduction in the amplitude of the $V_0_2$ slow component. Indeed, the iEMG profiles during the two heavy exercise bouts were remarkably similar to the $V_0_2$ profiles. However, there was no significant relationship between the reduction in the $V_0_2$ slow component after prior heavy exercise and changes in the EMG responses. This was due, in large part, to substantial intersubject variability in iEMG toward the end of the exercise bouts. The present data, therefore, provide only qualitative support to the hypothesis that the $V_0_2$ slow component can be attributed, in part, to the serial recruitment of additional motor units (29, 31, 33).

Previous investigators have suggested that $A_t$ represents a “target amplitude” dictated by external power output and work efficiency that is subsequently modified with time during heavy exercise (5, 6, 26). This target amplitude is altered after prior heavy exercise such that the primary response initially projects to a $V_0_2$ that is closer to the $V_0_2$ required later in exercise (as estimated from end-exercise $V_0_2$). In the present study, it is clear from the unchanged MPF that the increase in iEMG early in the second heavy exercise bout represents additional motor unit recruitment (presumably comprising both principal fiber types) and not increased firing frequency or the preferential recruitment of higher threshold motor units. Fatigued fibers may be activated but may fail to produce any tension, resulting in the need to recruit more muscle fibers to maintain the power output and an increase in the $O_2$ cost of exercise (18). An increase in motor unit recruitment for the same power output could reduce the apparent work efficiency because an estimated 30–50% of the energetic cost of fiber recruitment is committed to processes independent of tension development (i.e., those maintaining intracellular homeostasis; Ref. 36). This is consistent with the proportionality between the iEMG and the primary $V_0_2$ amplitude presented in Fig. 3B. Recruitment of a larger motor unit pool at the onset of exercise, the fibers of which are able to generate tension, would result in a reduction in the tension generated by each individual fiber and therefore reduce the metabolic disturbance experienced by the muscle as a whole. The smaller increase in [lactate] and smaller $V_0_2$ slow component responses in the second heavy leg bout seem to support this interpretation. In addition, Rossiter et al. (32) have recently demonstrated that phosphocreatine degradation during heavy leg extension exercise in the prone position is significantly blunted (although not speeded) by a prior priming bout. Interestingly, these authors showed that this attenuation of the phosphocreatine concentration decrement was associated with speeded primary $V_0_2$ kinetics and not an elevated primary amplitude. The reason for the difference between these findings and those of other studies, including the present study, is not clear, although the possibility that differences in exercise mode account for these findings has been considered previously (32). The elevation of HR in the baseline period, coupled with the greater $HbO_2$ saturation and total $Hb$ in the vastus lateralis observed before the onset of the second bout of heavy leg exercise, provides indirect evidence that $O_2$ delivery to the exercising muscle was increased by prior heavy exercise. These findings are consistent with the original proposal that a prior bout of exercise that induces a residual lactic acidosis increases muscle perfusion (12), although it should be noted that differences in the NIRS signal might also have been due to greater cutaneous blood flow in the second bout consequent to heat storage. However, it is important to emphasize that any increase in muscle perfusion did not alter the primary $V_0_2$ time constant because this was unchanged in the second bout of exercise. This provides additional evidence that the primary $V_0_2$ kinetics are not limited by $O_2$ availability during heavy exercise (4, 14, 15, 17). Krstrup et al. (21) recently demonstrated that $O_2$ delivery exceeded muscle $O_2$ consumption, even during the first of a series of high-intensity exercise bouts in humans. The increased EMG signal at the onset of the second bout of heavy exercise indicates that there is an increased demand for $O_2$ and that the increase in $O_2$ delivery supports rather than causes the increased primary $V_0_2$ amplitude.

The kinetics of muscle oxygenation at the onset and offset of exercise have recently been estimated (8, 37). Mathematical modeling of the $HbO_2$ signal with a model similar to that used to characterize the $V_0_2$ response has also been attempted in a preliminary
report (28), and the time constants derived correlated with the primary \( V_{O2} \) time constants, although the actual values of the muscle oxygenation and \( V_{O2} \) time constants differed considerably. Mathematical modeling of the present data has not been attempted, because the temporal profile of the \( HbO_2 \) signal did not conform to a simple monoeXponential function with or without a delay. Indeed, as shown in Fig. 4A, there appeared to be an apparent reoxygenation beyond the first minute of the exercise bout. As emphasized by MacDonald et al. (23), this response is not evident in measurements of venous \( O2 \) saturation, indicating that it is likely to be an artifact of the manner in which NIRS data is acquired. McCully and Hamaoka (24) suggest that, because the NIRS probe measures the weighted average of oxygenation status in the blood contained in the arterioles, capillaries, and venules, the weighting during exercise might shift from the venules toward the capillaries as blood flow increases and a greater red cell volume is located in the more oxygenated capillaries.

It was of interest that, despite significant residual lactacidemia at the onset of the second heavy exercise bout, muscle pH (measured 8 min after the first heavy exercise bout) was not significantly different from that measured under rested conditions before the first bout. We measured muscle pH by use of a needle-tipped electrode inserted into the vastus lateralis muscle. By using this technique, a measure of pH in the muscle compartment is obtained, which will represent the average pH of the intracellular and extracellular fluid (1). Allsop et al. (1) suggested that the muscle pH values recorded by using needle-tipped electrodes are \( \pm 0.1-0.2 \) units higher than those measured by using the homogenate technique employed in biopsy studies, reflecting a larger contribution of the extracellular fluid to the electrode measure. However, this technique should still identify any changes in muscle pH before the onset of subsequent exercise.

The similarity of the muscle pH values at rest and 8 min after heavy exercise is at first sight counterintuitive. Both blood [lactate] and blood pH were significantly altered compared with the control condition, consistent with previous reports (9, 10, 12). However, it is known that the maintenance of muscle pH homeostasis involves several mechanisms, including \( Na^+/H^+ \) exchange, lactate-proton cotransport, and bicarbonate-dependent transport (19). It has been demonstrated that net muscle \( H^+ \) release occurs at a significantly faster rate than lactate release (19). This net \( H^+ \) release continues even when the muscle that had previously been releasing lactate has switched to net lactate uptake (3). It is, therefore, likely that the present data reflect the relatively rapid restoration of muscle pH homeostasis; as a consequence, the present data provide evidence against a role for reduced muscle pH, per se, in the effect of prior heavy exercise.

The data of the present study might provide some insight into the spatial and temporal character of the factor or substance that causes an increase in motor unit recruitment. Because the effect of prior heavy exercise occurred after 12 min of recovery, the present study provides additional evidence that the factor responsible demonstrates a slow recovery time course (10, 27). The role of lactate as a mediator of the effect of prior heavy exercise has yet to be refuted. The present data confirm that an elevation in the primary \( V_{O2} \) amplitude is associated with a residual elevation in blood [lactate]. Moritani et al. (25) showed that surface EMG amplitude increased after occlusion during repeated isometric contractions at 20% of the maximal voluntary contraction. The fact that EMG amplitude remained elevated for 2 min after cuff release and was accompanied by elevated blood (and presumably muscle) [lactate] demonstrated the important role of the metabolic state of the exercising muscle in motor unit recruitment strategy. Thus it is possible that lactate itself may be responsible for altered motor unit recruitment at the onset of a second bout of heavy exercise. Simultaneous arterial and venous blood sampling has shown that inactive muscle may undergo net lactate uptake (2, 13), and this may explain the similar (but quantitatively smaller) effect on the \( V_{O2} \) kinetics when the prior heavy exercise is performed by the arms (9).

In summary, the present study has shown that the elevation in the primary \( V_{O2} \) amplitude after a bout of prior heavy exercise is associated with an elevation in iEMG and no change in MPF during the first 2 min of heavy exercise. Prior heavy leg exercise produced a residual increase in blood [lactate] and a reduction in blood pH but did not affect baseline muscle pH. There was evidence of an increased \( O2 \) delivery to the exercising leg muscle in the second exercise bout, as indicated by increased baseline HR, total Hb, and \( HbO_2 \) saturation, and a reduced \( O2 \) pulse. However, the primary \( V_{O2} \) time constant was unaffected by prior heavy exercise. These results suggest that the elevated primary \( V_{O2} \) amplitude during a second bout of heavy exercise can be attributed to an increase in motor unit recruitment at the onset of exercise.

REFERENCES


