Optimizing the “priming” effect: influence of prior exercise intensity and recovery duration on O₂ uptake kinetics and severe-intensity exercise tolerance

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Bayley SJ, Vanhatalo A, Wilkerson DP, DiMenna FJ, Jones AM. Optimizing the “priming” effect: influence of prior exercise intensity and recovery duration on O₂ uptake kinetics and severe-intensity exercise tolerance. J Appl Physiol 107: 1743–1756, 2009. First published October 1, 2009; doi:10.1152/japplphysiol.00810.2009.—It has been suggested that a prior bout of high-intensity exercise has the potential to enhance performance during subsequent high-intensity exercise by accelerating the O₂ uptake (V˙O₂) on-response. However, the optimal combination of prior exercise intensity and subsequent recovery duration required to elicit this effect is presently unclear. Eight male participants, aged 18–24 yr, completed step cycle ergometer exercise tests to 80% of the difference between the preestablished gas exchange threshold and maximal V˙O₂ (i.e., 80%Δ) after no prior exercise (control) and after six different combinations of prior exercise intensity and recovery duration: 40%Δ with 3 min (40-3-80), 9 min (40-9-80), and 20 min (40-20-80) of recovery and 70%Δ with 3 min (70-3-80), 9 min (70-9-80), and 20 min (70-20-80) of recovery. Overall V˙O₂ kinetics were accelerated relative to control in all conditions except for 40-9-80 and 40-20-80 conditions as a consequence of a reduction in the V˙O₂ slow component amplitude; the phase II time constant was not significantly altered with any prior exercise/recovery combination. Exercise tolerance at 80%Δ was improved by 15% and 30% above control in the 70-9-80 and 70-20-80 conditions, respectively, but was impaired by 16% in the 70-3-80 condition. Prior exercise at 40%Δ did not significantly influence exercise tolerance regardless of the recovery duration. These data demonstrate that prior high-intensity exercise (~70%Δ) can enhance the tolerance to subsequent high-intensity exercise provided that it is coupled with adequate recovery duration (>9 min). This combination presumably optimizes the balance between preserving the effects of prior exercise on V˙O₂ kinetics and providing sufficient time for muscle homeostasis (e.g., muscle phosphocreatine and H+ concentrations) to be restored.

priming exercise; oxygen consumption kinetics; exercise performance; near-infrared spectroscopy; surface electromyography

After the onset of constant-work rate exercise, pulmonary O₂ uptake (V˙O₂) rises with near-exponential kinetics to attain a steady state within 2–3 min in young healthy adults performing moderate-intensity [below the gas exchange threshold (GET)] exercise (60, 61). For exercise above the GET, the fundamental V˙O₂ response is supplemented by a delayed-onset V˙O₂ “slow component,” which delays the attainment of a steady state during heavy-intensity exercise [performed below the critical power (CP)] or sets the V˙O₂ on a trajectory toward its maximum during severe-intensity (>CP) exercise (50, 61, 64). The magnitude of the “O₂ deficit” incurred after the onset of exercise, which is compensated by increased substrate-level phosphorylation, is a function of both the rate at which V˙O₂ rises after the onset of exercise (V˙O₂ “kinetics”) and the required “steady-state” amplitude. During high-intensity exercise, sparing the utilization of the finite anaerobic energy reserve and the accumulation of metabolites associated with the fatigue process by speeding overall V˙O₂ kinetics and/or delaying the attainment of maximal V˙O₂ (V˙O₂max) by reducing the V˙O₂ slow component would therefore be expected to result in enhanced exercise performance (3, 13, 59).

One acute intervention known to elicit an overall speeding of V˙O₂ dynamics during supra-GET exercise is the performance of a prior bout of high-intensity “warm-up” or “priming” exercise. Gerbino et al. (26) demonstrated that prior heavy-intensity exercise (but not moderate-intensity exercise) resulted in faster overall V˙O₂ kinetics during a second heavy-intensity exercise bout. Subsequent investigations established that this overall speeding of V˙O₂ kinetics was chiefly consequent to a reduced V˙O₂ slow component amplitude (8, 14, 40). It has been shown that although these effects on the V˙O₂ response recede with time, they are preserved for at least 30–45 min after the completion of the prior exercise bout (9). The performance of higher-intensity exercise, such as constant-work rate severe-intensity exercise or single or repeated bouts of sprint exercise, can also speed the overall V˙O₂ response during subsequent exercise (10, 11, 17, 22, 63). The mechanistic basis for the enhanced V˙O₂ kinetics after prior high-intensity exercise remains a topic of intense debate (35, 49, 56). Such exercise induces a myriad of changes in skeletal muscle physiology, including increases in blood flow (4, 21, 41), oxygenation (19, 34, 43, 63), oxidative enzyme activity (15, 29, 53), O₂ extraction (19, 20, 25, 41, 43), and electromyographic (EMG) activity (7, 43), but which of these changes is essential for facilitating the speeding of V˙O₂ kinetics during subsequent exercise is presently obscure.

While the aforementioned alterations in V˙O₂ kinetics after prior exercise might be expected to enhance exercise tolerance, direct evidence for this is limited or equivocal (10, 17, 22, 36, 39, 63). This is due, in large part, to between-study differences in the intensities of the prior exercise and criterion exercise bouts and in the intervening recovery durations. The available evidence suggests that the tolerance to severe-intensity exercise is impaired after repeated all-out sprint exercise with 15 min of recovery (63) and prior severe-intensity exercise with 2 min of recovery (22). An unchanged severe-intensity exercise performance has been reported 10 min after a single 30-s sprint (10), 8 min after both low-intensity and high-intensity exercise (39), and 6 min after severe-intensity exercise (17). Conversely, improved tolerance to severe-intensity exercise has been reported 10 min after both moderate-intensity and heavy-intensity exercise (10) and 6 min (17) and 10 min (36) after heavy-intensity exercise. It should be noted here that the “heavy” exercise completed in these previous studies (10, 17, 36) was conducted at an intensity of “50%Δ” (i.e., GET plus

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50% of the difference between the GET and V̇O₂max). This is problematic in that this intensity lies close to the CP (50, 64), and it is therefore possible that at least some subjects in these previous studies actually performed severe-intensity prior exercise.

The available data therefore indicate that prior exercise can be detrimental to subsequent exercise performance when it is excessively intense (63) or when insufficient recovery is provided (22, 57). However, given that effects on V̇O₂ kinetics are greater after severe-intensity prior exercise than heavy-intensity prior exercise (35) and that these effects can be maintained for at least 30–45 min (9), it is possible that severe-intensity prior exercise combined with an extended recovery period [≥15 min (57)] might maximize the potential for exercise tolerance to be enhanced. Studies that provide information on the prior exercise/recovery combination that optimizes exercise tolerance are important because applied physiologists and coaches are becoming increasingly interested in manipulating precompetition warm-up regimes to enhance athletic performance (10, 30, 47).

The purpose of this investigation was to elucidate how prior exercise intensity and subsequent recovery duration interact to affect V̇O₂ kinetics and the tolerance to severe-intensity exercise. We studied the physiological responses to severe-intensity exercise after no prior exercise (control) and after six different combinations of prior exercise (performed at either 40%Δ or 70%Δ, reflecting heavy and severe-intensity exercise, respectively) and subsequent recovery (of 3, 9, and 20 min in duration). We hypothesized that prior severe-intensity exercise followed by a 20-min recovery period would result in the greatest improvement in exercise tolerance, whereas prior severe-intensity exercise followed by a 3-min recovery period would impair tolerance to subsequent severe-intensity exercise. To provide insight into the physiological bases for the effects observed, we measured the dynamics of pulmonary gas exchange, heart rate (HR), muscle oxygenation [using near-infrared spectroscopy (NIRS)], blood [lactate], and EMG during all conditions.

**METHODS**

**Subjects.** Eight healthy male subjects (means ± SD; age: 21 ± 2 yr, height: 177 ± 4 cm, and body mass: 76 ± 6 kg) volunteered to participate in this study. Subjects participated in exercise at a recreational level, but were not highly trained, and were familiar with laboratory exercise testing procedures, having previously participated in studies using similar procedures in our laboratory. The study was approved by the University of Exeter Research Ethics Committee, and all subjects were required to give their written informed consent before the commencement of the study once the experimental procedures, associated risks, and potential benefits of participation had been explained. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial, and to avoid strenuous exercise in the 24 h before each testing session. Each subject was also asked to refrain from caffeine and alcohol 6 and 24 h before each test, respectively. All tests were performed at the same time of day (±2 h).

**Experimental design.** Subjects were required to report to the laboratory on 15 occasions over a 6-wk period, and all tests were interspersed with at least 24 h of recovery. After an initial ramp incremental test, all subjects completed a number of “double-step” exercise tests during which pulmonary V̇O₂ and HR kinetics, blood [lactate], parameters of muscle oxygenation (by NIRS), muscle activation [integrated EMG (iEMG)], and exercise tolerance were assessed. To determine the interactive influence of prior exercise intensity and recovery duration on subsequent exercise performance, we used a paradigm comprising two different exercise intensities and three different recovery durations.

**Incremental test.** On the first laboratory visit, subjects completed a ramp incremental exercise test for the determination of the V̇O₂ peak and GET. All cycle tests were performed on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Initially, subjects performed 3 min of baseline cycling at 0 W, after which the work rate was increased at a rate of 30 W/min until the limit of tolerance. Subjects cycled at a self-selected pedal rate (between 70 and 90 rpm), and this pedal rate, along with the saddle and handle bar height and configuration, was recorded and reproduced in subsequent tests. Breath-by-breath pulmonary gas exchange data were collected continuously during the incremental tests and averaged over consecutive 10-s periods. The V̇O₂ peak was taken as the highest 30-s average value attained before the subject’s volitional exhaustion in the test. The GET was determined from a cluster of measurements including 1) the first disproportionate increase in CO₂ production (VCO₂) from visual inspection of individual plots of VCO₂ versus V̇O₂, 2) an increase in expired ventilation (VE/V̇O₂ with no increase in VE/VCO₂, and 3) the increase in end-tidal PCO₂ with no fall in end-tidal PCO₂. The work rates that would require 40%Δ [GET plus 40% of the difference between the work rate at the GET and V̇O₂ peak (heavy exercise)], 70%Δ [GET plus 70% of the difference between the work rate at the GET and V̇O₂ peak (severe exercise)], and 80%Δ [GET plus 80% of the difference between the work rate at the GET and V̇O₂ peak (severe exercise)] were subsequently calculated.

**Square-wave tests.** A total of seven experimental conditions were investigated in this study, and these were administered in a randomized order. In the control condition, subjects completed 4 min of “unloaded” 20-W baseline cycling before an abrupt step increment to the target 80%Δ work rate was imposed. The remaining six conditions comprised double-step tests wherein the transitions to 80%Δ were preceded by bouts of prior exercise. The prior exercise conditions involved 4 min of baseline unloaded pedaling followed by step increments of 6-min duration to either 40%Δ (heavy exercise) or 70%Δ (severe exercise). A 3-min period of unloaded cycling took place before the 80%Δ criterion work rate, which was performed either immediately after the priming exercise or after 6 or 17 min of seated passive recovery. This scheme resulted in 3, 9, or 20 min of recovery, with the unloaded cycling considered part of the recovery duration. The six prior exercise permutations were, therefore, 40%Δ with 3 min (40-3-80), 9 min (40-9-80), and 20 min (40-20-80) of recovery and 70%Δ with 3 min (70-3-80), 9 min (70-9-80), and 20 min (70-20-80) of recovery. Each participant completed the seven protocols (control plus six prior exercise/recovery conditions) on two separate occasions. On one of these occasions, exercise at 80%Δ was continued until task failure as a measure of exercise tolerance, whereas on the other occasion the exercise was performed for 6 min only. The time to task failure was recorded when the pedal rate fell by >10 rpm below the required pedal rate. The V̇O₂ responses from these like transitions were averaged before any analysis to enhance the signal-to-noise ratio and improve confidence in the parameters derived from the model fits (58).

**Measurements.** During all tests, pulmonary gas exchange and ventilation were measured continuously using a portable metabolic cart (MetaMax 3B, Cortex Biophysik, Leipzig, Germany), as previously described (3, 20). A DVT turbine digital transducer measured inspired and expired airflow, whereas an electrochemical cell O₂ analyzer and ND infrared CO₂ analyzer simultaneously measured expired gases. Subjects wore a nose clip and breathed through a low-dead space, low-resistance mouthpiece that was securely attached to the volume transducer. The inspired and expired gas volume and gas concentration signals were continuously sampled via a capillary line connected to the mouthpiece. The gas analyzers were calibrated

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before each test with gases of known concentration, and the turbine
volume transducer was calibrated using a 3-liter syringe (Hans Rud-
dolph, Kansas City, MO). Pulmonary gas exchange and ventilation
were calculated and displayed by breath. HR was measured
during all tests using short-range radiotelemetry (Polar S610, Polar
Electro Oy, Kempele, Finland).

During the 80%Δ criterion transitions, a blood sample was col-
lected from a fingertip into a capillary tube over the 20 s before the
step transition in work rate, at 6 min into the transition, and also at
the limit of tolerance. These whole blood samples were subsequently
analyzed to determine blood [lactate] (YSI 1500, Yellow Springs
Instruments, Yellow Springs, OH) within 30 s of collection. Blood
lactate accumulation (change in blood [lactate]) was calculated as the
difference between the blood [lactate] at 6 min and blood [lactate] at
baseline.

The oxygenation status of the vastus lateralis muscle of the right
leg was monitored using a commercially available NIRS system
(model NIRO 300, Hamamatsu Photonics, Hiugashi-ku, Japan). The
system consisted of an emission probe that irradiates laser beams and
a detection probe. Four different wavelength laser diodes provided the
light source (776, 826, 845, and 905 nm), and the light returning from
the tissue was detected by a photomultiplier tube in the spectrometer.
The intensity of incident and transmitted light was recorded continu-
quously at 2 Hz and used to estimate concentration changes from the
fractional hemoglobin (HbO2) and oxyhemoglobin (HHb), respectively. The
NIRS data represent a relative change based on the optical density measured in
the first datum collected. The [HHb] signal can be regarded as being
effectively blood volume insensitive during exercise (18, 28) and was,
therefore, assumed to provide an estimate of changes in the fractional
O2 extraction in the field of interrogation (19, 23, 28, 34). It should be
noted here that the contribution of oxyhemoglobin myoglobin to the
NIRS signal is presently unclear, and, as such, the terms [HbO2],
[HbO2]), and [HHb] used in this article should be considered to refer to
the combined concentrations of total, oxygenated, and deoxy-
genated hemoglobin and myoglobin, respectively. The leg was initially
cleaned and shaved around the belly of the muscle, and the optodes
were placed in the holder, which was secured to the skin with adhesive
at 20 cm above the lateral epicondyle. To secure the holder and wires
in place, an elastic bandage was wrapped around the subject’s leg. The
wrap helped to minimize the possibility that extraneous light could
influence the signal and also ensured that the optodes did not move
during exercise. Pen marks were made around the holder to enable
precise reproduction of the placement in subsequent tests. The probe
gain was set with the subject at rest in a seated position with the leg
extended at down stroke on the cycle ergometer before the first
exercise bout, and NIRS data were collected continuously throughout
the exercise protocols. The data were subsequently downloaded onto
a personal computer, and the resulting text files were stored on disk
for later analysis.

Neuromuscular activity of the vastus lateralis muscle of the left leg
was measured using bipolar surface EMG. The leg was initially
shaved and cleaned with alcohol around the belly of the muscle, and
graphite snap electrodes (Unilect 40713, Unomedical, Stonehouse,
UK) were adhered to the prepared area in a bipolar arrangement
(interelectrode distance: 40 mm). A ground electrode was positioned on
the rectus femoris equidistant from the active electrodes. The sites
of electrode placement were chosen according to the recommenda-
tions provided in the EMG software (Mega Electronics). To secure
electrodes and wires in place and to minimize movement during
cycling, an elastic bandage was wrapped around the subject’s leg. Pen
marks were made around the electrodes to enable reproduction of the
placement in subsequent tests. The EMG signal was recorded using a
ME3000PB Muscle Tester (Mega Electronics). EMG measurements
at a sampling frequency of 1,000 Hz were recorded throughout all
test bouts. The bipolar signal was amplified (amplifier input
impedance >1 MΩ), and data were collected online in raw form and
stored on a personal computer using MegaWin software (Mega
Electronics). The raw EMG data were subsequently exported as an
ASCII file and digitally filtered using a custom-designed filter devel-
oped through Labview 8.2 (National Instruments, Newbury, UK).
Initially, the signals were filtered with a 20-Hz high-pass, second-
order Butterworth filter to remove contamination from movement
artifacts. The signal was then rectified and low pass filtered at a
frequency of 500 Hz to produce a linear envelope. The average iEMG
was calculated for 10-s intervals throughout the 80%Δ criterion
exercise bout and the preceding baseline, with these values normal-
ized to the average measured during 10–180 s of unloaded cycling
before the initial prior exercise transition. Therefore, all iEMG data
are presented as a percentage of the prior exercise unloaded cycling
phase. Data from repeat trials were averaged, and ΔiEMG(2 – 6 min)
was defined as the difference between the average iEMG over the last
10 s of exercise and the average from 110–120 s.

Table 1. VO2 kinetics during severe-intensity exercise in the control and variously primed conditions

<table>
<thead>
<tr>
<th>Primed Conditions</th>
<th>40-3-80</th>
<th>40-9-80</th>
<th>40-20-80</th>
<th>70-3-80</th>
<th>70-9-80</th>
<th>70-20-80</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline VO2, l/min</td>
<td>0.98±0.08</td>
<td>1.12±0.13</td>
<td>1.03±0.16</td>
<td>1.03±0.12</td>
<td>1.31±0.14</td>
<td>1.15±0.14</td>
</tr>
<tr>
<td>End-exercise VO2 at 6 min, l/min</td>
<td>3.80±0.38</td>
<td>3.84±0.39</td>
<td>3.81±0.36</td>
<td>3.88±0.45</td>
<td>3.79±0.35</td>
<td>3.87±0.38</td>
</tr>
<tr>
<td>VO2 exhaustion, l/min</td>
<td>3.92±0.41</td>
<td>3.87±0.37</td>
<td>3.85±0.36</td>
<td>3.85±0.45</td>
<td>3.80±0.37</td>
<td>3.90±0.40</td>
</tr>
<tr>
<td>Phase II r, s</td>
<td>31±9</td>
<td>30±8</td>
<td>27±10</td>
<td>29±10</td>
<td>28±7</td>
<td>29±6</td>
</tr>
<tr>
<td>95% Confidence interval, s</td>
<td>5±3</td>
<td>5±2</td>
<td>4±3</td>
<td>4±3</td>
<td>4±3</td>
<td>6±3</td>
</tr>
<tr>
<td>Fundamental amplitude, l/min</td>
<td>2.27±0.33</td>
<td>2.30±0.32</td>
<td>2.26±0.20</td>
<td>2.35±0.39</td>
<td>2.21±0.24</td>
<td>2.36±0.30</td>
</tr>
<tr>
<td>Absolute fundamental amplitude, l/min</td>
<td>3.27±0.34</td>
<td>3.42±0.34</td>
<td>3.29±0.19</td>
<td>3.38±0.40</td>
<td>3.51±0.38</td>
<td>3.53±0.35</td>
</tr>
<tr>
<td>Fundamental gain, ml·min−1·l−1</td>
<td>6.8±0.7</td>
<td>8.7±0.8</td>
<td>8.6±0.7</td>
<td>8.9±0.9</td>
<td>8.4±0.4</td>
<td>9.0±0.7</td>
</tr>
<tr>
<td>Slow component amplitude, l/min</td>
<td>0.65±0.12</td>
<td>0.44±0.13</td>
<td>0.53±0.26</td>
<td>0.54±0.14</td>
<td>0.30±0.14</td>
<td>0.39±0.20</td>
</tr>
<tr>
<td>Slow component amplitude, %</td>
<td>22±6.4</td>
<td>16.7±4.3</td>
<td>19±5.5</td>
<td>19±5.4</td>
<td>12±5.4</td>
<td>14±6.6</td>
</tr>
<tr>
<td>Overall r, s</td>
<td>8.8±2.2</td>
<td>7.2±1.2</td>
<td>7.1±1.7</td>
<td>7.5±1.7</td>
<td>5.2±1.3</td>
<td>6.0±1.8</td>
</tr>
<tr>
<td>End-exercise gain, ml·min−1·l−1</td>
<td>10.3±0.5</td>
<td>10.3±0.5</td>
<td>9.9±0.8</td>
<td>10.1±0.7</td>
<td>8.8±0.5</td>
<td>9.7±0.8</td>
</tr>
</tbody>
</table>

Values are means ± SD. The six prior exercise permutations were as follows: 40%Δ with 3 min (40-3-80), 9 min (40-9-80), and 20 min (40-20-80) of recovery and 70%Δ with 3 min (70-3-80), 9 min (70-9-80), and 20 min (70-20-80) of recovery. VO2, O2 uptake; r, time constant. *Significantly different from control (P < 0.05); #significantly different from control (P < 0.01); $significantly different from the 40-3-80 condition (P < 0.05); %significantly different from the 40-9-80 condition (P < 0.05); #significantly different from the 40-20-80 condition (P < 0.05); $significantly different from the 70-3-80 condition (P < 0.05); %significantly different from the 70-9-80 condition (P < 0.05); *significantly different from the 70-20-80 condition (P < 0.05).
vide second-by-second values, and, for each individual, identical repetitions were time aligned to the start of exercise and ensemble averaged. The first 20 s of data after the onset of exercise (i.e., the phase I response) were deleted (58), and a nonlinear least-square algorithm was used to fit the data thereafter. A single-exponential model was used to characterize the kinetics of the overall \( \dot{V}O_2 \) response to the criterion exercise bouts, and a biexponential model was used to characterize the \( \dot{V}O_2 \) response kinetics in its constituent fundamental and slow components, as described by the following equations:

\[
\dot{V}O_2(t) = \dot{V}O_{2\text{baseline}} + A_p[1 - e^{-\frac{t - TD_p}{\tau_p}}]
\]

\[
\dot{V}O_2(t) = \dot{V}O_{2\text{baseline}} + A_s[1 - e^{-\frac{t - TD_s}{\tau_s}}] + A_f[1 - e^{-\frac{t - TD_f}{\tau_f}}]
\]

where \( \dot{V}O_2(t) \) represents the absolute \( \dot{V}O_2 \) at a given time \( t \); \( \dot{V}O_{2\text{baseline}} \) represents the mean \( \dot{V}O_2 \) in the baseline period; \( A_p, TD_p, \) and \( \tau_p \) represent the amplitude, time delay, and time constant, respectively, describing the fundamental phase II increase in \( \dot{V}O_2 \) above baseline; and \( A_s, TD_s, \) and \( \tau_s \) represent the amplitude, time delay before the onset, and time constant describing the development of the \( \dot{V}O_2 \) slow component, respectively.

An iterative process was used to minimize the sum of the squared errors between the fitted function and the observed values. \( \dot{V}O_{2\text{baseline}} \) was defined as the mean \( \dot{V}O_2 \) measured over the final 90 s of baseline pedaling. The end-exercise \( \dot{V}O_2 \) was defined as the mean \( \dot{V}O_2 \) measured over the final 30 s of the 6-min exercise bouts, whereas the \( \dot{V}O_2 \) at exhaustion was defined as the mean \( \dot{V}O_2 \) measured over the 30 s before the subject’s termination of exercise. The absolute fundamental component amplitude (absolute \( A_p \)) was defined as the sum of \( \dot{V}O_{2\text{baseline}} \) and \( A_p \). Because the asymptotic value (\( A_s \)) of the exponential term describing the \( \dot{V}O_2 \) slow component was

Fig. 1. Comparisons of the group mean pulmonary \( O_2 \) uptake (\( \dot{V}O_2 \)) dynamics during a step increment from an unloaded baseline to a severe-intensity work rate during control (Con) and in the following priming conditions: 40-3-80 (top left), 40-9-80 (middle left), 40-20-80 (bottom left), 70-3-80 (top right), 70-9-80 (middle right), and 70-20-80 (bottom right). ○, responses in control; •, responses to the various primed conditions. Error bars are omitted for clarity. Notice that the priming effect is greatest when the criterion bout is preceded by severe-intensity exercise and that it wanes with increasing recovery time after both heavy-intensity and severe-intensity prior exercise. The insets in the respective \( \dot{V}O_2 \) graphs show the individual (dashed lines) and group mean ± SE (solid lines) changes in the tolerance to severe-intensity exercise relative to control. *Significantly different from control (\( P < 0.05 \)); #significantly different from the 70-9-80 condition (\( P < 0.05 \)).
component may represent a higher value than is actually reached at the end of the exercise, the actual amplitude of the VO2 slow component at the end of exercise was defined as A'. The A' parameter was compared at the same isotime (360 s) pre- and postintervention. The amplitude of the slow component was also described relative to the entire VO2 response. In addition, the functional “gain” of the fundamental VO2 response was computed by dividing A' by the change in work rate. The functional gain of the entire response (i.e., end-exercise gain) was calculated in a similar manner.

To provide information on muscle oxygenation, we also modeled the [HHb] response to exercise. [HHb] kinetics during the criterion exercise bouts in the fundamental phase were determined by fitting a biexponential model from the first data point, which was 1 SD above the baseline mean through the entire response. [HHb] TD and τ values were summed to provide information on overall [HHb] response dynamics in the fundamental phase of the response. The [HbO2] and [Hbtot] responses do not approximate an exponential (19) and were, therefore, not modeled. Rather, we assessed priming-induced changes in these parameters by determining the [HbO2] and [Hbtot] at baseline (90-s before the step transition) and at 60, 120, and 360 s (average response over the final 30 s of exercise).

We also modeled the HR response to exercise in each condition. For this analysis, HR data were linearly interpolated to provide second-by-second values, and, for each individual, identical repetitions from like transitions were time aligned to the start of exercise and ensemble averaged. A nonlinear least squares monoexponential model without TD was used to fit the data to the criterion 80% Δ exercise, with the fitting window commencing at t = 0 s. The derived HR mean response time (MRT; equivalent to the overall τ for the response) provides an insight into the overall rate of adjustment of HR dynamics. Priming-induced changes in HR were also assessed through comparing the HR at baseline (90-s before the step transition) and at 60, 120, and 360 s (average response over the final 30 s) of exercise for each experimental condition.

Statistical analysis. One-way repeated-measures ANOVA was used to determine the effects on the relevant physiological variables elicited by the differing prior exercise permutations. Where the analysis revealed a significant difference, individual paired t-tests were used with a Fisher’s least-significant difference correction to determine the origin of such effects. Pearson’s product-moment correlation was used to examine the interrelationships between the parameters of VO2 kinetics, iEMG, muscle oxygenation, and exercise tolerance. All data are presented as means ± SD. Statistical significance was accepted when P < 0.05.

**RESULTS**

During the ramp incremental test, subjects attained a peak work rate of 354 ± 41 W and a VO2 peak of 3.80 ± 0.45 l/min, whereas the work rate and VO2 values at the GET were 107 ± 13 W and 1.54 ± 0.16 l/min, respectively. The 40%Δ and 70%Δ prior exercise work rates were calculated to be 186 ± 15 and 260 ± 27 W, respectively, and the work rate corresponding to 80%Δ (used in the criterion bout) was 284 ± 32 W.

**VO2 kinetics.** The parameters of VO2 dynamics during the control and primed criterion severe-intensity exercise bouts are reported in Table 1 and shown as group mean responses in Fig. 1. The baseline VO2 was significantly elevated above control in the 40-3-80, 70-3-80, and 70-9-80 prior exercise conditions, with the greatest elevation occurring in the 70-3-80 condition (Table 1 and Fig. 1). The end-exercise VO2 during the trial to exhaustion was similar among all experimental conditions, attaining a value of ~100–103% of the VO2 peak. Phase II τ was not significantly affected by any of the prior exercise conditions (Table 1). In the 70-20-80 condition only, the VO2 fundamental component amplitude was significantly elevated above control (control: 2.27 ± 0.33 l/min and 70-20-80: 2.44 ± 0.30 l/min, P < 0.01). The absolute fundamental component amplitude (baseline + fundamental component amplitude), however, was significantly elevated above control in the 40-3-80, 70-3-80, 70-9-80, and 70-20-80 conditions (Table 1 and Fig. 1). Compared with control, the amplitude of the VO2 slow component was significantly reduced in the 40-3-80, 70-3-80, 70-9-80, and 70-20-80 conditions (Table 1 and Fig. 1). The overall VO2 kinetics (as assessed with a

**Table 2. Near-infrared spectroscopy-derived [HHb], [HbO2], and [Hbtot] during severe-intensity exercise in the control and variously primed conditions**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>40-3-80</th>
<th>40-9-80</th>
<th>40-20-80</th>
<th>70-3-80</th>
<th>70-9-80</th>
<th>70-20-80</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>[HHb]</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline, AU</td>
<td>−95 ± 58</td>
<td>−127 ± 71</td>
<td>−97 ± 59</td>
<td>−105 ± 41</td>
<td>−116 ± 87</td>
<td>−99 ± 72</td>
<td>−92 ± 60</td>
</tr>
<tr>
<td>End exercise, AU</td>
<td>252 ± 125</td>
<td>297 ± 151</td>
<td>258 ± 116</td>
<td>263 ± 132</td>
<td>266 ± 160</td>
<td>249 ± 105</td>
<td>268 ± 133</td>
</tr>
<tr>
<td>Primary TD, s</td>
<td>5 ± 1</td>
<td>2 ± 2.0d,e</td>
<td>4 ± 2</td>
<td>4 ± 2</td>
<td>2 ± 1b,d,e,f</td>
<td>3 ± 1b,d,e,f</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>Primary τ, s</td>
<td>9 ± 4</td>
<td>9 ± 4</td>
<td>8 ± 2</td>
<td>8 ± 2</td>
<td>9 ± 2</td>
<td>8 ± 2</td>
<td>12 ± 2</td>
</tr>
<tr>
<td>TD + τ, s</td>
<td>14 ± 4</td>
<td>11 ± 3b</td>
<td>12 ± 2</td>
<td>12 ± 2</td>
<td>9 ± 2b</td>
<td>11 ± 3b</td>
<td>12 ± 2</td>
</tr>
<tr>
<td>Primary phase amplitude, AU</td>
<td>294 ± 81</td>
<td>364 ± 116a</td>
<td>307 ± 92</td>
<td>316 ± 110</td>
<td>360 ± 82a</td>
<td>313 ± 69</td>
<td>318 ± 116a</td>
</tr>
<tr>
<td>Slow phase amplitude, AU</td>
<td>54 ± 26</td>
<td>62 ± 39</td>
<td>52 ± 27</td>
<td>58 ± 21</td>
<td>23 ± 17b</td>
<td>39 ± 27</td>
<td>47 ± 19</td>
</tr>
<tr>
<td><strong>[HbO2]</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline, AU</td>
<td>25 ± 62</td>
<td>213 ± 99b,d,e,f</td>
<td>127 ± 71b</td>
<td>90 ± 99</td>
<td>205 ± 131a</td>
<td>96 ± 63a</td>
<td>65 ± 121</td>
</tr>
<tr>
<td>At 60 s, AU</td>
<td>−235 ± 79</td>
<td>−175 ± 69a</td>
<td>−152 ± 72a</td>
<td>−189 ± 55</td>
<td>−226 ± 187</td>
<td>−197 ± 68</td>
<td>−216 ± 70</td>
</tr>
<tr>
<td>At 120 s, AU</td>
<td>−197 ± 90</td>
<td>−157 ± 66</td>
<td>−121 ± 64a,d,e,f</td>
<td>−167 ± 53</td>
<td>−208 ± 196</td>
<td>−168 ± 72</td>
<td>−189 ± 71</td>
</tr>
<tr>
<td>End exercise, AU</td>
<td>−187 ± 87</td>
<td>−159 ± 77</td>
<td>−114 ± 55c</td>
<td>−149 ± 71</td>
<td>−215 ± 190</td>
<td>−167 ± 74</td>
<td>−178 ± 87</td>
</tr>
<tr>
<td><strong>[Hbtot]</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline, AU</td>
<td>−70 ± 80</td>
<td>86 ± 90b,e</td>
<td>30 ± 112b</td>
<td>−15 ± 105</td>
<td>89 ± 90b,e</td>
<td>−4 ± 86</td>
<td>−27 ± 144</td>
</tr>
<tr>
<td>At 60 s, AU</td>
<td>−320 ± 87</td>
<td>68 ± 102b,e</td>
<td>61 ± 95b</td>
<td>22 ± 102</td>
<td>18 ± 102</td>
<td>17 ± 84</td>
<td>9 ± 124</td>
</tr>
<tr>
<td>At 120 s, AU</td>
<td>20 ± 70</td>
<td>108 ± 111a</td>
<td>102 ± 77a</td>
<td>55 ± 119</td>
<td>42 ± 107</td>
<td>54 ± 81</td>
<td>48 ± 115</td>
</tr>
<tr>
<td>End exercise, AU</td>
<td>65 ± 77</td>
<td>138 ± 113a</td>
<td>144 ± 89b</td>
<td>114 ± 109</td>
<td>51 ± 101</td>
<td>82 ± 82</td>
<td>91 ± 119</td>
</tr>
</tbody>
</table>

Values are means ± SD. HHb, HbO2, and Hbtot are deoxygenated, oxygenated, and total hemoglobin/myoglobin. AU, arbitrary units; TD, time delay. aSignificantly different from control (P < 0.05); bsignificantly different from control (P < 0.01); csignificantly different from the 40-3-80 condition (P < 0.05); dsignificantly different from the 40-9-80 condition (P < 0.05); esignificantly different from the 40-20-80 condition (P < 0.05); fsignificantly different from the 70-9-80 condition (P < 0.05); gsignificantly different from the 70-20-80 condition (P < 0.05).
monoexponential model) were significantly faster than control (88 ± 22 s) in the 40-3-80 (72 ± 12 s), 70-3-80 (52 ± 13 s), 70-9-80 (60 ± 10 s), and 70-20-80 (68 ± 19 s) conditions (Table 1 and Fig. 1).

**NIRS and HR kinetics.** The NIRS parameters during severe-intensity exercise in the control and primed conditions are shown in Table 2. [HHb] dynamics during exercise after each prior exercise condition are shown relative to control in Fig. 2. [HHb] during baseline and at the end of exercise were similar across the prior exercise conditions and did not differ from control (Table 2 and Fig. 2). While [HHb] τ was similar across the experimental conditions investigated in this study (P > 0.05), [HHb] TD was significantly reduced below control in the 40-3-80, 70-3-80, and 70-9-80 prior exercise conditions. As such, [HHb] MRT (TD + τ) was significantly faster in the 40-3-80 (11 ± 3 s), 70-3-80 (11 ± 2 s), and 70-9-80 (11 ± 3 s) prior exercise conditions relative to control (14 ± 4 s; Table 2). The [HHb] primary amplitude was significantly greater than control in the 40-3-80, 70-3-80, and 70-20-80 prior exercise conditions. However, the [HHb] slow component amplitude was only reduced significantly below the control value in the 70-3-80 condition (Table 2 and Fig. 2).

The priming-induced changes in [HbO2] compared with control are shown in Fig. 3. [HbO2] was significantly elevated during the baseline of the criterion bout in the 40-3-80, 40-9-80, 70-3-80, and 70-9-80 prior exercise conditions relative to control (Table 2 and Fig. 3). [HbTot] was significantly elevated during the baseline of the criterion bout in the 40-3-80, 40-9-
80, and 70-3-80 prior exercise conditions relative to control (Table 2).

HR dynamics in the CON and primed conditions are reported in Table 3 and shown as group mean responses in Fig. 4. Compared with control, the HR at baseline and at 60 and 120 s of exercise was significantly elevated in all of the prior exercise conditions investigated. Overall kinetics of the HR response, on the other hand, were only significantly faster than control in the 40-9-80, 70-3-80, and 70-20-80 prior exercise conditions (Table 3 and Fig. 4).

**Blood [lactate].** The baseline blood [lactate] was significantly elevated above control (1.1 ± 0.2 mM) during the 40-3-80, 40-9-80, 70-3-80, 70-9-80, and 70-20-80 prior exercise conditions (Table 3). The accumulation of blood lactate over the first 6 min of the criterion exercise bout was significantly reduced compared with control in the 70-3-80 and 70-9-80 conditions (Table 3).

**iEMG response.** iEMG responses during the criterion exercise bout in the control and experimental conditions are shown in Table 4 and Fig. 5. The average iEMG response over the first 60, 120, and 360 s of exercise as well as the absolute values at 120 and 360 s did not differ across the experimental conditions (Table 4). The change in iEMG from 2 to 6 min [ΔiEMG(2 – 6 min)] was reduced relative to control (104 ± 115% increase) only in the
70-20-80 prior exercise condition (16 ± 45% increase, P < 0.05; Table 4 and Fig. 5). However, intracondition analyses revealed that the iEMG signal at 120 s was significantly lower than that observed at 360 s in the control and 40-9-80 conditions, whereas these values were similar at 120 and 360 s in the remaining prior exercise conditions (Table 4 and Fig. 5). In the 70-20-80 prior exercise condition, the reduced ΔiEMG(6–2 min) was significantly correlated with the reduction in the V̇O₂ slow component amplitude (r = 0.75, P < 0.05).

**Exercise tolerance.** The changes in the tolerance of severe-intensity exercise consequent to the various prior exercise interventions were compared with control in the insets of the respective V̇O₂ graphs in Fig. 1, and the group mean change is shown in Fig. 6. Prior exercise at 40% Δ did not significantly alter severe-intensity exercise tolerance, regardless of the recovery duration with which this intensity of prior exercise was coupled (Fig. 1). Significant improvements in exercise tolerance above control (437 ± 79 s) were observed in the 70-9-80 condition (504 ± 84 s, +15% improvement, P < 0.05) and in the 70-20-80 condition (567 ± 125 s, +30% improvement, P < 0.05; Figs. 1 and 6). The time to exhaustion was significantly longer in the 70-20-80 condition compared with the 70-9-80 condition (P < 0.05). Exercise tolerance was significantly impaired in the 70-3-80 condition, being reduced 16% below control (P < 0.05; Fig. 6).

**DISCUSSION**

The principal original finding of this investigation was that severe-intensity prior exercise significantly improved subsequent severe-intensity exercise tolerance when it was coupled with a recovery period of 9 min (15% improved performance) or 20 min (30% improved performance; Fig. 6). In contrast, prior severe-intensity exercise followed by 3 min of recovery resulted in a significant 16% impairment in the tolerance of subsequent severe-intensity exercise. The performance of prior heavy-intensity exercise did not enhance performance during subsequent severe-intensity exercise irrespective of the intervening recovery duration. Prior severe-intensity exercise was more effective than prior heavy-intensity exercise in altering V̇O₂ kinetics, but the magnitude of effect receded with time for both intensities of prior exercise. Overall V̇O₂ kinetics were accelerated in the 40-3-80, 70-3-80, 70-9-80, and 70-20-80 conditions. That exercise tolerance was only enhanced in the latter two conditions indicates that a speeding of V̇O₂ kinetics, per se, is not necessarily ergogenic. Rather, exercise tolerance was enhanced when the prior exercise bout was sufficiently intense to provoke an accelerated V̇O₂ response and the subsequent recovery duration was sufficiently long for homeostasis (as reflected by the baseline V̇O₂ and blood [lactate]) to return toward control values. Interestingly, in the 70-20-80 condition (where exercise tolerance was increased the most), V̇O₂ kinetics were accelerated in association with a significantly blunted ΔiEMG(6–2 min) relative to control.

**Physiological effects of prior exercise.** Overall V̇O₂ kinetics were significantly faster than control in all conditions other than the 40-9-80 and 40-20-80 conditions. Phase II V̇O₂ kinetics were not altered by any combination of prior exercise intensity and recovery duration in the young healthy subjects who participated in our study, results that are consistent with a large number of previous studies (e.g., Refs. 5, 7–11, 14, 21, 33, 39, 40, 48, 53, 54, and 63). Rather, the faster overall V̇O₂ adjustment was consequent to an increase in the absolute V̇O₂ fundamental component amplitude and, in particular, to a reduction in the V̇O₂ slow component amplitude. The latter was reduced below control in the 40-3-80 (by 32%), 70-3-80 (by 54%), 70-9-80 (by 40%), and 70-20-80 (by 26%) conditions, resulting, respectively, in an 11%, 41%, 32%, and 23% speeding of overall V̇O₂ kinetics relative to control.

A novel finding in this study was that heavy-intensity prior exercise did not elicit the typical prior exercise effect on the V̇O₂ response during subsequent exercise when the recovery duration separating the prior and criterion exercise bouts equaled or exceeded 9 min. This contrasts with previous reports (7, 8, 36). However, in the majority of the previous studies, “heavy-intensity” prior exercise was performed at 50% Δ (7, 8, 11, 14, 36), based on the initial study of Gerbino et al. (26). However, this is problematic as 50% Δ approximates the boundary between the heavy-intensity exercise domain, within which V̇O₂ will eventually stabilize, and the severe-intensity exercise domain, within which V̇O₂ will continue to rise with time until V̇O₂max is attained (50, 64). It is therefore possible that at least some of the subjects in these previous

---

**Table 4. HR dynamics and blood [lactate] during severe-intensity exercise in the control and variously primed conditions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Control</th>
<th>40-3-80</th>
<th>40-9-80</th>
<th>40-20-80</th>
<th>70-3-80</th>
<th>70-9-80</th>
<th>70-20-80</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline, beats/min</td>
<td>91 ± 12</td>
<td>113 ± 15d,e</td>
<td>104 ± 13b</td>
<td>101 ± 13a</td>
<td>128 ± 13b,c,d,e,g,h</td>
<td>116 ± 16b,d,e,g</td>
<td>108 ± 11b,e</td>
</tr>
<tr>
<td>At 60 s, beats/min</td>
<td>140 ± 5</td>
<td>156 ± 11b,e</td>
<td>153 ± 10b</td>
<td>149 ± 11a</td>
<td>165 ± 10b,c,d,e,g,h</td>
<td>160 ± 13b,e</td>
<td>154 ± 9b,e</td>
</tr>
<tr>
<td>End exercise, beats/min</td>
<td>157 ± 7</td>
<td>167 ± 9b</td>
<td>165 ± 9b</td>
<td>163 ± 10b</td>
<td>176 ± 9b,c,d,e,g,h</td>
<td>170 ± 11b,c,e</td>
<td>166 ± 8b,e</td>
</tr>
<tr>
<td>Mean response time, s</td>
<td>180 ± 7</td>
<td>182 ± 11</td>
<td>182 ± 10</td>
<td>182 ± 9</td>
<td>187 ± 9b,c,d,e,h</td>
<td>186 ± 9</td>
<td>180 ± 7</td>
</tr>
</tbody>
</table>

**Blood [lactate]**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Control</th>
<th>40-3-80</th>
<th>40-9-80</th>
<th>40-20-80</th>
<th>70-3-80</th>
<th>70-9-80</th>
<th>70-20-80</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline, mM</td>
<td>1.1 ± 0.2</td>
<td>3.1 ± 0.9b,d,e</td>
<td>2.0 ± 0.4b</td>
<td>1.3 ± 0.3</td>
<td>6.7 ± 0.9b</td>
<td>5.3 ± 1.0b</td>
<td>3.0 ± 0.8b</td>
</tr>
<tr>
<td>At 360 s, mM</td>
<td>7.3 ± 1.5</td>
<td>9.2 ± 1.2r</td>
<td>8.2 ± 1.4</td>
<td>7.8 ± 1.1</td>
<td>9.9 ± 2.1b,c,d,e,g,h</td>
<td>9.3 ± 1.8b,c,d,e,h</td>
<td>8.0 ± 1.6b,c,e,h</td>
</tr>
<tr>
<td>Change in baseline – 360 s, mM</td>
<td>6.2 ± 1.7</td>
<td>6.1 ± 1.2</td>
<td>6.2 ± 1.4</td>
<td>6.5 ± 0.9</td>
<td>3.2 ± 1.4b,c,d,e,h</td>
<td>4.0 ± 1.3b,c,d,e,h</td>
<td>5.0 ± 1.3b,c,e,h</td>
</tr>
<tr>
<td>Time to exhaustion, mM</td>
<td>10.1 ± 1.6</td>
<td>9.4 ± 1.3</td>
<td>10.1 ± 1.8</td>
<td>9.9 ± 1.8</td>
<td>9.8 ± 1.5</td>
<td>9.9 ± 1.9</td>
<td>10.1 ± 1.6</td>
</tr>
<tr>
<td>Change in baseline – time to exhaustion, mM</td>
<td>8.9 ± 1.7</td>
<td>6.3 ± 1.0b,d,e</td>
<td>8.1 ± 1.8</td>
<td>8.6 ± 1.7</td>
<td>3.1 ± 1.5b,c,d,e,h</td>
<td>4.5 ± 1.5b,c,d,e,h</td>
<td>7.1 ± 1.3b,c,d,e,h</td>
</tr>
</tbody>
</table>

Values are means ± SD. HR, heart rate. *Significantly different from control (P < 0.05); †significantly different from the 70-3-80 condition (P < 0.05); ‡significantly different from the 40-3-80 condition (P < 0.05); §significantly different from the 40-9-80 condition (P < 0.05); ¶significantly different from the 40-20-80 condition (P < 0.05); ‖significantly different from the 70-9-80 condition (P < 0.05); ||significantly different from the 70-20-80 condition (P < 0.05).
studies performed prior exercise at an intensity that exceeded CP. We selected 40% \( H_9004 \) to represent heavy-intensity exercise in the present study, and, although we did not formally assess CP, the \( \dot{V}O_2 \) response profiles elicited by our prior exercise bouts at 40% \( H_9004 \) and 70% \( H_9004 \) were entirely characteristic of heavy-intensity and severe-intensity exercise (50, 64). Our data show that prior heavy-intensity exercise reduced the \( \dot{V}O_2 \) slow component during subsequent severe-intensity exercise after 3 min of recovery, but that the effect was lost when recovery was extended to 9 or 20 min. The results of the present study therefore indicate that prior severe-intensity exercise provides a more potent and longer-lasting stimulus than heavy-intensity exercise for reducing the \( \dot{V}O_2 \) slow component during subsequent exercise.

HR was significantly elevated above control in all the prior exercise conditions investigated in the study, both during the baseline period and over the first 120 s of exercise, as noted previously (e.g., Refs. 7, 25, 55, and 63). At the muscular level, however, \([Hb_{tot}]\) (an index of hyperemia) was significantly greater than control only in the baseline period of the 40-3-80, 40-9-80, and 70-3-80 conditions relative to control. Despite this evidence for greater muscle \( O_2 \) availabil-
ity, VO₂ τp was not significantly different from control for any of the prior exercise conditions studied. In young healthy subjects performing high-intensity upright cycle exercise, VO₂ τp appears to be intransigent to interventions that would be expected to enhance the muscle O₂ supply (Refs. 14, 33, 49, 62, and 65; cf. Ref. 29), indicating that the finite rate at which VO₂ rises after the onset of exercise is determined principally by an intracellular limitation to the rate of O₂ utilization (27, 49, 52). It is of interest that overall VO₂ kinetics were accelerated compared with control in the 70-9-80 and 70-20-80 conditions despite the baseline [HbO₂] and [HHb] having returned to control values. These data suggest that an enhanced muscle vasodilation at baseline after a prior exercise intervention is not necessary to invoke a speeding of overall VO₂ kinetics. Similarly, prior contractile activity has been reported to result in the faster activation of muscle oxidative metabolism in the absence of enhanced muscle O₂ availability in animal models (6, 32). Also, although the MRT for HR kinetics was faster in the 70-9-80 and 70-20-80 conditions compared with control, this was also true for the 40-9-80 condition in which VO₂ kinetics were not significantly altered, indicating a dissociation between changes in HR kinetics and VO₂ kinetics.

The NIRS-derived [HHb] response reflects the balance between local O₂ delivery and utilization and has been used previously as an index of muscle fractional O₂ extraction (3, 19, 20, 23, 28, 29, 34). Relative to control, [HHb] TD and also [HHb] TD + τ was significantly reduced in the 40-3-80, 70-3-80, and 70-9-80 conditions. Moreover, the [HHb] primary amplitude was significantly increased above control in the 40-3-80, 70-3-80, and 70-20-80 conditions. The shorter TD + τ and/or greater [HHb] amplitude observed in these conditions indicates that, in the region of NIRS interrogation, muscle VO₂ increased relatively more than blood flow in the transition to the higher metabolic rate in the primed condition. In the 70-20-80 condition, where [HbO₂] and [HHb] were not different from control, the faster overall VO₂ kinetics was apparently facilitated, in part, by an increased muscle O₂ extraction. These data are consistent with previous reports showing that prior intense exercise increases muscle O₂ extraction during subsequent exercise (19, 20, 25, 41). The increased ability for muscle to extract O₂ after priming is presumably a consequence of an increased oxidative enzyme activity, which might be expected to reduce the intrinsic metabolic inertia to muscle O₂ utilization (27, 29, 49, 53). Certainly, the data indicate that the rate of change of VO₂ exceeded the rate of change of blood flow to a greater extent in the presence with the absence, of priming exercise. It should be noted that, like many of the other physiological responses measured in this study, the NIRS indexes of muscle O₂ extraction during exercise became less pronounced as the recovery interval increased (Fig. 2). This is consistent with the time course of recovery of muscle enzyme activity after exercise. For example, it has been reported that pyruvate dehydrogenase (PDH) activity, which has been suggested to represent a possible limitation to VO₂ kinetics by restricting carbon substrate availability (15, 29, 56), recovers with a half-time of ~4 min after intense exercise (51). On this basis, PDH activity would have essentially recovered to baseline after 20 min of recovery, yet overall VO₂ kinetics remained 23% faster than control in the 70-20-80 condition. This suggests a possible dissociation between PDH (and possibly other oxidative enzyme) activity and the observed changes in VO₂ kinetics after prior exercise.

iEMG quantifies the gross electrical activity of the muscle and therefore provides information pertaining to motor unit recruitment and firing frequency during a given exercise task. It has been reported that, after a prior high-intensity exercise bout, iEMG is higher over the first 2 min of subsequent high-intensity exercise and then increases at a lower rate as exercise proceeds (7, 43). This has been interpreted to indicate that motor unit recruitment is increased after the onset of exercise in the primed condition such that the metabolic demand per fiber is reduced and the requirement for additional fiber recruitment as high-intensity exercise continues is reduced (7, 43). The development of the VO₂ slow component has been ascribed to the progressive recruitment of motor units with time (7, 35, 42, 48, 58), and thus changes in motor unit recruitment profiles could explain the reciprocal changes noted in the VO₂ fundamental and slow component amplitudes after prior exercise interventions (7, 12). While there is reasonable consensus that muscle fiber type and motor unit recruitment patterns play an important role in the development of the VO₂ slow component, it should be noted that the contribution of progressive motor unit recruitment, per se, to the development of the VO₂ slow component remains a matter of debate (see Refs. 20, 54, and 66 for a discussion).

In the present study, the iEMG values increased significantly between 2 and 6 min in the control and 40-9-80 conditions, with there being a strong trend for a similar pattern in the 40-20-80 condition (Fig. 5). However, the iEMG at 6 min was not significantly higher than at 2 min in the other prior exercise and recovery conditions, implying that the recruitment of additional motor units with time was attenuated. Interestingly, the VO₂ slow component was reduced below control in these

### Table 4. Surface iEMG during severe-intensity exercise in the control and variously primed conditions

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>40-3-80</th>
<th>40-9-80</th>
<th>40-20-80</th>
<th>70-3-80</th>
<th>70-9-80</th>
<th>70-20-80</th>
</tr>
</thead>
<tbody>
<tr>
<td>iEMG average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>From 0 to 60 s, %baseline</td>
<td>531 ± 149</td>
<td>526 ± 146</td>
<td>514 ± 148</td>
<td>549 ± 160</td>
<td>511 ± 189</td>
<td>544 ± 242</td>
<td>623 ± 301</td>
</tr>
<tr>
<td>From 0 to 120 s, %baseline</td>
<td>536 ± 146</td>
<td>541 ± 163</td>
<td>522 ± 155</td>
<td>558 ± 175</td>
<td>523 ± 191</td>
<td>558 ± 251</td>
<td>629 ± 306</td>
</tr>
<tr>
<td>From 0 to 360 s, %baseline</td>
<td>604 ± 193</td>
<td>574 ± 195</td>
<td>558 ± 174</td>
<td>602 ± 219</td>
<td>539 ± 235</td>
<td>589 ± 266</td>
<td>650 ± 317</td>
</tr>
<tr>
<td>iEMG at 120 s, %baseline</td>
<td>559 ± 157</td>
<td>560 ± 184</td>
<td>540 ± 177</td>
<td>572 ± 193</td>
<td>549 ± 209</td>
<td>574 ± 265</td>
<td>640 ± 322</td>
</tr>
<tr>
<td>iEMG at 360 s, %baseline</td>
<td>663 ± 2649</td>
<td>607 ± 225</td>
<td>600 ± 2101</td>
<td>622 ± 269</td>
<td>492 ± 316</td>
<td>585 ± 312</td>
<td>656 ± 325</td>
</tr>
<tr>
<td>Change in iEMG from 6 to 2 min, %baseline</td>
<td>104 ± 115</td>
<td>47 ± 61</td>
<td>59 ± 71</td>
<td>50 ± 89</td>
<td>22 ± 157</td>
<td>12 ± 113</td>
<td>16 ± 45*</td>
</tr>
</tbody>
</table>

Values are means ± SD. iEMG, integrated electromyography. *Significantly different from control (P < 0.05); †significantly different from the 120-s value in the respective experimental condition (P < 0.05).
same conditions, supporting the notion that the progressive recruitment of muscle fibers might contribute to the development of the \( \dot{V}O_2 \) slow component (42, 48, 58) and suggesting that alterations in muscle fiber recruitment might be, at least in part, responsible for the speeding of overall \( \dot{V}O_2 \) kinetics after prior exercise (7, 12, 35, 43). This was particularly the case for the 70-20-80 condition, in which there was a clear trend for iEMG to be higher over the first 2 min of exercise and in which \( \Delta iEMG(6 - 2 \text{ min}) \) was significantly reduced. Therefore, in the 70-20-80 condition, the overall \( \dot{V}O_2 \) speeding appears to be related to an initial increase in muscle activity and fractional \( O_2 \) extraction and reduced recruitment of additional motor units with time. These data support the previous findings of Burnley et al. (7), who reported that muscle iEMG was 19% higher at the onset of the second of two bouts of severe-intensity exercise and noted that the iEMG responses (higher initial iEMG and a blunted increase in iEMG as exercise proceeded) were qualitatively similar to the changes in \( \dot{V}O_2 \) kinetics (increased \( \dot{V}O_2 \) fundamental component amplitude and reduced \( \dot{V}O_2 \) slow component) in the second bout. The mechanism responsible for these changes in iEMG is not clear. However, it has been shown that prior muscle activity reduces the “recruitment threshold” of motor units during subsequent contractions and that the restoration of the original thresholds may take considerably longer than the restoration of voluntary force-generating capacity (1, 16).
Exercising tolerance. A significantly improved tolerance of severe-intensity exercise was observed after prior severe-intensity exercise coupled with 9 and 20 min of recovery but not after severe-intensity exercise and 3 min of recovery or after heavy-intensity prior exercise irrespective of the recovery duration. This potential for prior high-intensity exercise to enhance performance during subsequent exercise confirms some previous reports (10, 36). Importantly, the improved exercise tolerance in the 70-9-80 and 70-20-80 conditions manifested when the VO2 slow component amplitude was reduced and overall VO2 kinetics were speeded. While these data imply an interdependence between a speeding of overall VO2 kinetics after prior exercise interact to determine the tolerance to severe-intensity exercise we observed.

The performance of prior severe-intensity exercise does not appear to alter the CP or VO2max, but does have the potential to alter the finite quantity of work that can be performed above CP (W'). During severe-intensity exercise (22, 36, 57). As such, it seems plausible that changes in W' and VO2 kinetics after prior exercise interact to determine the tolerance to subsequent high-intensity exercise. The magnitude of W' is determined by the phosphocreatine (PCr) and glycolytic (anaerobic) energy reserves along with a small contribution from stored O2 (44, 45) and/or the accumulation of fatiguerelated metabolites [e.g., H+, P, H2PO4−, and extracellular K+] (2, 22, 34, 50, 57)]. During severe-intensity exercise, W' is gradually expended with time, and exercise cannot be continued at the same rate when W' has been entirely depleted (44, 50). It has been shown that severe-intensity exercise performance is impaired after a prior severe-intensity exercise bout and short recovery period as a consequence of a reduced W' at baseline (22, 57). Forbes et al. (24) reported that intramuscular [PCr] and pH were reduced below the control value 3 min after prior heavy-intensity plantar flexion exercise. When the recovery duration was extended to 6 and 15 min, however, [PCr] and pH were not different from control, with the pH recovering further between 6 and 15 min (24). In the present study, baseline VO2 was elevated above control in the 40-3-80, 70-3-80, and 70-9-80 conditions. An elevated baseline VO2 would be expected to reflect an incomplete [PCr] recovery after prior exercise given the close agreement between pulmonary VO2 and intramuscular [PCr] (52). It should be noted that a low intramuscular [PCr] might facilitate an acceleration of VO2 kinetics by reducing metabolic capacitance and hence enabling the stimuli to oxidative phosphorylation to rise more rapidly (37, 38); however, as noted above, a low [PCr] at the start of exercise might be expected to impair performance. It has been reported that the estimated W' is restored to baseline within ~15 min after prior sprint exercise (57) and that the effect of prior high-intensity exercise on VO2 kinetics is preserved for at least 30 min but declines in a time-dependent manner (9). Therefore, extending the recovery duration to restore W' (and hence the muscle energetic reserve and tissue homeostasis) after prior severe exercise, while preserving faster overall VO2 dynamics, appears to have facilitated the increased tolerance to severe-intensity exercise we observed.

The performance of prior high-intensity exercise resulted in an elevated baseline blood [lactate] and a reduced accumulation of blood lactate during the criterion bout (7, 14, 20, 36). We have previously reported that a baseline blood [lactate] of ~3 mM appeared to be associated with improvements in performance after prior exercise (10, 36, 47). Residual acidosis provides a stimulus for an increased O2 availability through facilitating vasodilatation and a Bohr shift in the O2 dissociation curve, potentially supporting faster overall VO2 kinetics as initially hypothesized by Gerbino et al. (26). Additionally, a small reduction in muscle pH might provide a stimulus for increased muscle excitability during the subsequent bout by offsetting the detrimental effects of muscle depolarization that accompanies repeated intense muscle contractions (2, 31, 46). Moderate-intensity prior exercise does not appreciably elevate blood [lactate] above resting values and does not enhance VO2 kinetics or performance during subsequent exercise (8, 14, 26, 39). In the present study, prior heavy-intensity exercise did not significantly enhance exercise tolerance irrespective of the recovery duration. For the 40-9-80 and 40-20-80 conditions, baseline blood [lactate] was low (~2.0 and 1.3 mM, respectively) and VO2 kinetics were not enhanced. Similarly, Burnley et al. (9) have shown that the effect of prior exercise on VO2 kinetics is lost when baseline blood [lactate] recovers to ~2 mM. In the 40-3-80 condition, baseline blood [lactate] was elevated (~3.1 mM) and overall VO2 kinetics were speeded, but the 3-min recovery duration was unlikely to be sufficient to enable a full restoration of muscle [PCr] (24). Previous studies have shown that prior multiple sprint exercise or severe-intensity exercise with a short recovery, leading to a baseline blood [lactate] of ~6–7 mM, enhances VO2 kinetics but is detrimental to subsequent exercise performance (22, 63). In these circumstances, it is likely that the high blood [lactate] reflects a situation in which W' has not been fully restored and/or the muscle fatigue-related metabolite concentration remains high. In the present study, the baseline blood [lactate] was ~7 mM in the 70-3-80 condition, where, relative to
control, performance was significantly impaired, ~5 mM in the 70-9-80 condition, where performance was enhanced by 15%, and ~3 mM in the 70-20-80 condition, where performance was enhanced by 30%. These results are therefore consistent with previous studies in suggesting that a baseline blood [lactate] in the range of ~3–5 mM is associated with enhanced performance during subsequent exercise (10, 36, 47), with the proviso that the recovery interval is sufficient to enable muscle homeostasis (including the concentration of fatigue-related metabolites) to be restored toward control values.

Conclusions. The appropriate combination of prior exercise intensity and recovery duration enables an acceleration of VO₂ kinetics during subsequent severe-intensity exercise. This acceleration is principally the result of a reduction in the amplitude of the VO₂ slow component. The present study shows that VO₂ kinetics are not accelerated if the prior bout of exercise is not sufficiently intense or the recovery interval is too long (i.e., the 40-9-80 and 40-20-80 conditions), whereas the effect is greatest if a high-intensity prior exercise bout is coupled with a short recovery interval (the 70-3-80 condition). The mechanistic bases for the speeded overall VO₂ kinetics after prior high-intensity exercise are complex and can potentially involve the interaction of a number of separate effects including increased muscle O₂ availability, greater muscle oxidative enzyme activity and carbon substrate supply, and altered motor unit recruitment profiles (19, 29, 35). All of these effects would be expected to precede as the recovery interval separating the priming and criterion bouts is extended. An important finding in the present study was that, after severe-intensity prior exercise, overall VO₂ kinetics remained significantly speeded after 20 min of recovery, when the baseline [Hb,art] and [HbO₂] had returned to control values. In this condition, the reduced VO₂ slow component was associated with a reduced change in iEMG between 2 and 6 min of exercise, suggesting that changes in motor unit recruitment after prior exercise has a long latency and that such changes, along perhaps with increased activity of rate-limiting enzymes in the respiratory chain, might underpin the effects of prior exercise on VO₂ kinetics and performance during subsequent exercise, at least in young physically active subjects.

The fact that exercise tolerance was significantly reduced in the 70-3-80 condition despite it producing the fastest overall VO₂ response suggests that the presence of faster VO₂ kinetics, per se, will not necessarily enhance performance during subsequent high-intensity exercise. Exercise tolerance was substantially improved in the 70-9-80 and 70-20-80 conditions and was greatest in the latter, in which the overall VO₂ response was speeded relative to control and the baseline VO₂ was restored. We suggest that the extent to which a prior exercise/recovery regimen will be ergogenic is determined by an interaction between the magnitude of the acceleration of overall VO₂ kinetics and the magnitude of the reconstitution of W’. There is presently significant interest among applied physiologists in optimizing the precompetition warm-up to enhance athletic performance (30, 47). The present data suggest that a protocol involving a 6-min bout of severe-intensity exercise followed by a 20-min recovery period, enabling baseline VO₂ to be restored and blood [lactate] to decline to ~3 mM, can significantly speed overall VO₂ kinetics and improve the tolerance to subsequent severe-intensity exercise by as much as 30%, an effect that appears to be linked to changes in motor unit recruitment. This protocol appears to optimize the balance between maintaining faster overall VO₂ kinetics and allowing complete or near-complete restoration of muscle energetic reserves and homeostasis, thereby facilitating performance gains.

DISCLOSURES
No conflicts of interest are declared by the author(s).

REFERENCES

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