Primed Exercise Speeds Pulmonary \( O_2 \) Uptake Kinetics During Supine “work-to-work” High-Intensity Cycle Exercise

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DiMenna FJ, Wilkerson DP, Burnley M, Bailey SJ, Jones AM. Priming exercise speeds pulmonary \( O_2 \) uptake kinetics during supine “work-to-work” high-intensity cycle exercise. J Appl Physiol 108: 283–292, 2010. First published December 3, 2009; doi:10.1152/japplphysiol.01047.2009.—We manipulated the baseline metabolic rate and body position to explore the effect of the interaction between recruitment of discrete sections of the muscle fiber pool and muscle \( O_2 \) delivery on pulmonary \( O_2 \) uptake (\( V\dot{O}_2 \)) kinetics during cycle exercise. We hypothesized that phase II \( V\dot{O}_2 \) kinetics (\( \tau_p \)) in the transition from moderate- to severe-intensity exercise would be significantly slower in the supine than upright position because of a compromise to muscle perfusion and that a priming bout of severe-intensity exercise would return \( \tau_p \) during supine exercise to \( \tau_p \) during upright exercise. Eight male subjects [35 ± 13 (SD) yr] completed a series of “step” transitions to severe-intensity cycle exercise from an “unloaded” (20-W) baseline and a baseline of moderate-intensity exercise in the supine and upright body positions. \( \tau_p \) was not significantly different between supine and upright exercise during transitions from a 20-W baseline to moderate- or severe-intensity exercise but was significantly greater during moderate- to severe-intensity exercise in the supine position (54 ± 19 vs. 38 ± 10 s, \( P < 0.05 \)). Priming significantly reduced \( \tau_p \) during moderate- to severe-intensity supine exercise (34 ± 9 s), returning it to a value that was not significantly different from \( \tau_p \) in the upright position. This effect occurred in the absence of changes in estimated muscle fractional \( O_2 \) extraction (from the near-infrared spectroscopy-derived deoxygenated \( Hb \) concentration signal), such that the priming-induced facilitation of muscle blood flow matched increased \( O_2 \) utilization in the recruited fibers, resulting in a speeding of \( V\dot{O}_2 \) kinetics. These findings suggest that, during supine cycling, priming speeds \( V\dot{O}_2 \) kinetics by providing an increased driving pressure for \( O_2 \) diffusion in the higher-order (i.e., type II) fibers, which would be recruited in the transition from moderate- to severe-intensity exercise and are known to be especially sensitive to limitations in \( O_2 \) supply.

Oxygen uptake kinetics; phase II time constant; supine exercise; work-to-work transition; priming exercise

When a transition to high-intensity upright cycling is initiated from a moderate-intensity exercise baseline, the resultant “fundamental” (i.e., phase II) increase in pulmonary \( O_2 \) consumption (\( V\dot{O}_2 \)) is slower than when the same transition is elicited from unloaded cycling (16, 18, 55, 56). It has been proposed that this slowing of the phase II \( V\dot{O}_2 \) kinetics during “work-to-work” exercise can be explained, at least in part, by the population of muscle fibers contributing to power production under these circumstances (16, 18, 28, 55, 56). That is, the initiation of heavy- and severe-intensity exercise (i.e., above the gas exchange threshold (GET) and critical power, respectively) from a moderate-intensity (<GET) baseline would be expected to require a greater proportional contribution to power production from fibers that are higher in the recruitment hierarchy (e.g., type II fibers) (24, 36). There is evidence to suggest that these “higher-order” fibers have slower \( V\dot{O}_2 \) kinetics in isolated mouse and human muscle (2, 12, 28, 45).

High-intensity cycling results in slower \( V\dot{O}_2 \) kinetics in the supine position because of a lengthened phase II time constant (\( \tau_p \)) or a reduced amplitude of the \( V\dot{O}_2 \) fundamental component, with an increased amplitude of the \( V\dot{O}_2 \) slow component, than in the upright position (15, 25, 31). During supine exercise and any other contractile activity where the active musculature is at or above heart level, the gravitational assist to muscle blood flow is absent; therefore, perfusion pressure is reduced and the adaptation of \( O_2 \) delivery is slowed (39). Consequently, slower \( V\dot{O}_2 \) kinetics under these circumstances have been attributed to insufficient muscle \( O_2 \) availability (11, 15, 25, 31, 33, 38).

When high-intensity cycling is preceded by a high-intensity “priming” exercise bout, a faster overall \( V\dot{O}_2 \) response is observed (21, 27). Depending on the circumstances, this overall speeding of the \( V\dot{O}_2 \) kinetics is due to an increased amplitude of the \( V\dot{O}_2 \) fundamental component and a reduced amplitude of the \( V\dot{O}_2 \) slow component (1, 6–9, 17, 20, 32, 47–49, 57) or a shortened \( \tau_p \) (14, 17, 23, 25, 33, 51). The latter situation (reduced \( \tau_p \)) is typically reported only when \( \tau_p \) is relatively long (e.g., when \( O_2 \) delivery is compromised) in the control condition (14, 23, 25, 33). For example, Jones et al. (25) reported that priming exercise significantly reduces \( \tau_p \) during high-intensity supine cycling, thereby returning it to a value that is not significantly different from that measured during high-intensity upright cycling.

We recently reported that priming exercise does not alter the \( \tau_p \) during high-intensity work-to-work cycling in the upright posture (16). This suggests that the lengthened \( \tau_p \) during work-to-work exercise transitions in the upright position does not result from an \( O_2 \) delivery limitation but is, instead, related to an intrinsically slow oxidative metabolic response in the recruited higher-order muscle fibers. It is likely that these higher-order fibers would be more susceptible to interventions that decrease \( O_2 \) delivery (3, 40), such as postural alterations that reduce perfusion pressure (e.g., during cycling in the supine position). Higher-order fibers evince a greater reliance on fractional \( O_2 \) extraction to attain a given rate of oxidative metabolism (3, 40); therefore, it is possible that they would be unable to fully offset reduced perfusion pressure to prevent a further slowing of phase II \( V\dot{O}_2 \) kinetics during work-to-work supine cycling.

The purpose of the present study was to investigate fiber type-specific responses to reduced \( O_2 \) availability at the onset
of muscular work by using the work-to-work exercise model in conjunction with cycling performed in the supine position. Specifically, by dividing severe-intensity supine cycling transitions into two discrete steps (i.e., unloaded-to-moderate and moderate-to-severe), we examined the extent to which compromised muscle perfusion might influence the \( V\dot{O}_2 \) response to contraction of different segments of the fiber pool. In the first part of the study, we hypothesized that \( \tau_p \) would be similar in the supine and upright positions during transitions from unloaded to moderate-intensity exercise but that \( \tau_p \) would be significantly longer in the supine than in the upright position during transitions from moderate- to severe intensity work-to-work exercise. In the second part of the study, subjects performed the same supine work-to-work transitions after prior severe-intensity supine cycling and hypothesized that \( \tau_p \) would be reduced to a value similar to \( \tau_p \) in the upright control condition after priming. To provide insight into the mechanistic bases for differences in \( V\dot{O}_2 \) kinetics between the conditions, we used the deoxyhemoglobin concentration ([HHb]) signal derived from near-infrared spectroscopy (NIRS) to infer the degree to which body position and priming exercise influenced muscle fractional \( O_2 \) extraction and electromyography (EMG) to assess the degree to which motor unit activation was altered by priming.

**METHODS**

*Subjects*

Eight male subjects [35 ± 13 (SD) yr old, 1.83 ± 0.08 m stature, 80.3 ± 6.7 kg body mass] volunteered and gave written informed consent to participate in this study, which had been approved by the local Research Ethics Committee. All the subjects were recreationally active and were familiar with the experimental procedures used in the present study. On test days, subjects were instructed to report to the laboratory in a rested state, having completed no strenuous exercise within the previous 24 h and having abstained from food, alcohol, and caffeine for the preceding 3 h.

*Experimental Overview*

All testing was completed in an air-conditioned (20–22°C) laboratory. The subjects visited the laboratory on 14 occasions over a 5-wk period to perform exercise tests on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Testing was conducted at the same time of day (±2 h) for each subject. On each of the first two visits, the subjects completed a ramp incremental exercise test for determination of peak \( V\dot{O}_2 \) (\( V\dot{O}_2 \)peak) and GET. One test was performed in the upright position and the other with the subject lying supine, and test order was alternated between subjects. To create the supine condition, the front of the ergometer was braced against the wall, with the rear of the ergometer supported on a horizontal structure specifically constructed for this purpose. Owing to this configuration, the angle formed between the front of the ergometer and the floor was 32°, and the crank shaft was positioned 45 cm above the level of the subject’s back. Subjects lay supine on a mat inside a fixed structure equipped with handles that could be gripped to maintain body position, with their feet strapped securely to the pedals. On each of 12 subsequent visits, subjects completed bouts of severe-intensity exercise (at a work rate calculated to require 70% of the difference between posture-specific GET and \( V\dot{O}_2 \)peak, i.e., 70%Δ) initiated from “unloaded” (i.e., 20-W) cycling or a baseline of moderate-intensity cycling (95% of posture-specific GET). Transitions to moderate-intensity and work-to-work severe-intensity supine exercise were performed in the absence and presence of a preceding bout of severe-intensity supine cycling (70%Δ) and a rest period of 5 min. Work-to-work transitions and transitions from an unloaded baseline to severe-intensity cycling were also completed in the upright position. The experimental protocol is schematized in Fig. 1. Each of these protocols was presented to subjects three times in random order, and laboratory visits were separated by ≥48 h.

*Experimental Procedures*

The ramp incremental exercise tests consisted of 3 min of pedaling at 0 W followed by a continuous ramped increase in work rate of 30 W/min until the subject was unable to continue. The subjects cycled at 80 rpm, and saddle and handlebar heights for upright cycling and body distance relative to the crank shaft for supine cycling were recorded. The same pedal rate and settings were reproduced on subsequent tests. The \( V\dot{O}_2 \)peak was defined as the highest 30-s mean...
value recorded before the subject’s volitional termination of the test. The GET was determined from a cluster of measurements, including 
1) the first disproportionate increase in CO₂ output (VCO₂) from visual 
inspection of individual plots of VCO₂ vs. VO₂, 2) an increase in 
Ve/VO₂ (where Ve is expiratory ventilation), with no increase in 
Ve/VCO₂, and 3) an increase in end-tidal PCO₂, with no fall in end-tidal 
PcO₂. The work rates that would require 95% of the posture-specific 
GET (moderate exercise) and 70% of the difference between the 
posture-specific GET and VO₂ peak (severe exercise) were estimated, 
with account taken of the mean response time (MRT) of the VO₂ 
response to ramp exercise [assumed to be approximately two-thirds of 
the ramp rate, i.e., 20 W (52)]. These work rates were subsequently 
applied during the severe-intensity exercise and work-to-work transitions 
for the upright and supine conditions.

As outlined above, the subjects returned to the laboratory on 12 
occasions to perform 1 of the following protocols: 1) 3 min of 
unloaded cycling at 20 W and 6 min of severe-intensity cycling in the 
prioritization phase; 2) 3 min of unloaded cycling at 20 W, 4 min of 
moderate-intensity cycling, and 6 min of severe-intensity cycling in the 
upright position; and 3) 3 min of unloaded cycling at 20 W, 6 min of 
severe-intensity cycling, 5 min of passive rest, 3 min of unloaded 
cycling at 20 W, 4 min of moderate-intensity cycling, and 6 min of 
severe-intensity cycling in the supine position; and 4) 3 min of 
unloaded cycling at 20 W, 4 min of moderate-intensity cycling, and 6 
min of severe-intensity cycling in the supine position (Fig. 1). The 
VO₂ responses from like-transitions were averaged before any analysis 
was performed to enhance the signal-to-noise ratio and improve 
confidence in the parameters derived from the model fits (37, 53).

During all tests, pulmonary gas exchange and ventilation were mea-
sured breath-by-breath, with subjects wearing a nose clip and breathing 
through a low-dead space, low-resistance mouthpiece and bidirectional 
digital volume sensor (Jaeger TripleV). The inspired and expired gas 
volume and gas concentration signals were continuously sampled at 100 
Hz via a capillary line connected to the mouthpiece, the latter using 
paramagnetic (O₂) and infrared (CO₂) analyzers (Jaeger Oxycon Pro, 
Hoechberg, Germany). The gas analyzers were calibrated before each test 
with gases of known concentration, and the volume sensor was calibrated 
using a 3-liter syringe (Hans Rudolph, Kansas City, MO). Heart rate 
(HR) was measured every 5 s during all tests by short-range radiotelemetry 
(model S610, Polar Electro, Kempele, Finland). Baseline and 
end-exercise HR were defined as the mean HR measured over the final 
90 s of cycling before each transition and the final 30 s of each exercise bout, 
respectively. During one of the three trials for each condition, a blood 
sample from a fingertip was collected into a capillary tube over the 20 s 
preceding any step transition in work rate and within the last 20 s of 
exercise and subsequently analyzed to determine blood lactate concen-
tration (lactate); model 1500, Yellow Springs Instruments, Yellow 
Springs, OH). Blood lactate accumulation (∆blood concentration) was 
calculated as the difference between blood [lactate] at end exercise and 
blood [lactate] before the transition.

The oxygenation status of the vastus lateralis of the right leg was 
monitored using a commercially available NIRS system (model NIRO 
300, Hamamatsu Photonics, Higashi-ku, Japan). The system con-
sisted of an emission probe that irradiates laser beams and a detection 
probe that is positioned several centimeters from the emission probe 
in the spectrometer. The intensity of incident and transmitted light was 
recorded continuously at 2 Hz and used to estimate concentration 
changes from the resting baseline for oxygenated Hb (HbO₂) and 
HHb/myoglobin (Mb). Therefore, the NIRS data represent a relative 
change based on the optical density measured in the first datum 
collected. The Hb concentration signal can be regarded as being 
especially blood volume-insensitive during exercise (13, 22) and was, 
therefore, assumed to provide an estimate of changes in oxygenation 
status and fractional O₂ extraction in the field of interpretation (14, 22, 
26). It is not possible to determine the relative contribution of Mb to 
the total NIRS signal, but it is generally believed to be relatively small 
(e.g., <10%) (50). Nevertheless, in this study, the terms [Hbtotal] and 
[HHb] should be considered to reflect the combined concentrations of 
total Hb and HHb + Mb, respectively.

The leg was initially cleaned and shaved around the belly of the muscle, and the probes were placed in the holder, which was secured 
to the skin with adhesive at 20 cm above the fibular head. The holder 
and wires were secured in place by an elastic bandage that was 
wrapped around the subject’s leg. The wrap helped 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 reproduction of the placement 
in subsequent tests. The probe gain was set with the subject at rest in 
a seated position for upright exercise and in a supine position for 
supine exercise, with the leg extended at downstroke before the first 
exercise bout, and NIRS data were collected continuously throughout 
all bouts. 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 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 
neural analyzers (Unilect 40713, Unomedical, Stonehouse, UK) were 
attached to the prepared area in a bipolar arrangement (40-mm 
interelectrode distance). A ground electrode was positioned on the 
rectus femoris equidistant from the active electrodes. The sites of 
electrode placement were chosen according to the recommendations 
provided in the EMG software (Mega Electronics). To secure elec-
trodes and wires in place and 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 muscle tester 
(model ME3000PB, Mega Electronics).

EMG measurements at a sampling frequency of 1,000 Hz were 
recorded throughout all exercise tests. The bipolar signal was ampli-
fied (>1-MΩ amplifier input impedance), 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 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 50 Hz to produce a 
linear envelope. The average integrated EMG (iEMG) was calculated 
for 15-s intervals throughout exercise, with these values normalized to the 
average measured during 15–165 s of unloaded cycling before the 
initial transition. Therefore, all iEMG data are presented as a percent-
age of the initial unloaded cycling phase. Data from repeat trials were 
averaged, and iEMG at minute 2 and at end exercise were defined as 
the average from 120–135 s and the average over the last 15 s of 
exercise, respectively. ∆iEMGend−2 was calculated as the difference 
between minute 2 and end-exercise values.

Data Analysis Procedures

The breath-by-breath VO₂ data from each test were initially exam-
ined to exclude errant breaths caused by coughing, swallowing, and 
sighing, and those values lying >4 SDs from the local mean were 
removed. The breath-by-breath data were subsequently linearly inter-
polated to provide 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 (53, 54), and a nonlinear 
least-square algorithm was used to fit the data. For moderate- and 
severe-intensity exercise, single-exponential (Eq. 1) and biexponential 

\[ \text{Data Analysis Procedures} \]

\[ \text{Downloaded from http://jap.physiology.org/ by 10.220.32.247 on October 23, 2017} \]
\( (E 2) \) models, respectively, were used to characterize the \( \dot{V}O_2 \) response

\[
\dot{V}O_2(t) = \dot{V}O_2_{\text{baseline}} + A_p \left[ 1 - e^{-\left(-\Delta T_D\right)\tau_p} \right] 
\]

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; \( A_p \), \( \Delta T_D \), and \( \tau_p \) represent the amplitude, time delay, and time constant, respectively, describing the phase II increase in \( \dot{V}O_2 \) above baseline, and \( A_s \), \( \Delta T_D \), and \( \tau_s \), represent the amplitude of, time delay before the onset of, 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 cycling before each transition. The end-exercise \( \dot{V}O_2 \) was defined as the mean \( \dot{V}O_2 \) measured over the final 30 s of exercise. The fundamental component amplitude (absolute \( A_s \)) 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 may represent a higher value than is actually reached at the end of the exercise, the actual amplitude of the \( \dot{V}O_2 \) slow component at the end of exercise was defined as \( A_e \). The amplitude of the slow component was also described relative to the entire \( \dot{V}O_2 \) response (i.e., \( A_{s/e}\) and \( A_e \)) and by calculating the difference between \( \dot{V}O_2 \) at minute 1 (average from 105–135 s) and end-exercise (\( \Delta \dot{V}O_2_{26–21} \)). In addition, the functional “gain” of the fundamental \( \dot{V}O_2 \) response (\( G_p \)) was computed by dividing \( \dot{A}_p \) by \( \Delta \text{work} \); the functional gain of the entire response (i.e., end-exercise gain) was calculated in a similar manner. We also fitted a single-exponential curve without time delay from the onset to the end of severe-intensity exercise. The MRT so derived was used to provide information on the “overall” \( \dot{V}O_2 \) kinetics during severe-intensity exercise, with no distinction made for the various phases of the response.

To provide information on muscle oxygenation, we also modeled the \([HHb] \) response to exercise. The single- and biexponential models (\( E 1 \) and \( E 2 \)) were also used to fit the \([HHb] \) data for moderate- and severe-intensity cycling, respectively. However, in this case, the fitting window commenced from the first data point that was 1 SD above the baseline mean after initiation of the transition. For moderate exercise, \([HHb] \) \( \tau \) and \( TD \) were summed to provide information on the overall \([HHb] \) response dynamics (\( [HHb] \) \( \tau \) and \( TD \)), and the ratio of \([HHb]-modeled \) amplitude to \( \dot{V}O_2 \)-modeled amplitude was calculated as an index of \( O_2 \) extraction (\( \Delta[HHb]/\Delta\dot{V}O_2 \)). For severe exercise, \([HHb] \) \( \tau \) and \( TD \) was determined for the fundamental response phase, \( \Delta[HHb]/\Delta\dot{V}O_2 \) was calculated for the fundamental and overall response phases, and the \([HHb] \) slow component at end-exercise was defined relative to the overall \([HHb] \) response. We also fitted a single-exponential curve without time delay to the severe-intensity data from the onset to the end of exercise to provide information on the overall \([HHb] \) kinetics, with no distinction made for the various phases of the response (\([HHb] \) MRT). Finally, \([HbO_2] \) responses do not approximate an exponential \( (12) \) and were, therefore, not modeled; however, we did assess total blood volume by summing the \([HbO_2] \) and \([HHb] \) signals to provide an estimate of \([HHb]\text{tot} \) in the area under investigation. Specifically, we determined the mean value at baseline (30 s preceding each transition), at 60-s intervals throughout exercise (15-s bins centered on each point), and at end exercise (final 30 s) to facilitate comparisons between conditions.

**Statistics**

The parameters derived from the modeling of the \( \dot{V}O_2 \) and \([HHb] \) data and the HR and blood [lactate] data were analyzed using paired \( t \)-tests or one-way repeated-measures analysis of variance with Fisher’s least significant difference tests, as appropriate, to identify the location of statistically significant differences. Significance was accepted at \( P < 0.05 \). Results are reported as means ± SD.

**RESULTS**

The subjects’ \( \dot{V}O_2 \text{peak} \) and peak work rates were significantly lower during supine than upright cycling \( (51 ± 8 \text{ ml·kg}^{-1}·\text{min}^{-1} \) and \( 389 ± 54 \text{ W} \) for upright cycling vs. \( 47 ± 6 \text{ ml·kg}^{-1}·\text{min}^{-1} \) and \( 342 ± 39 \text{ W} \) for supine cycling, \( P < 0.05 \) in both cases). However, GET, which occurred at \( ~45\% \) \( \dot{V}O_2 \text{peak} \), was not significantly different between conditions. The moderate-intensity work rates were \( 111 ± 16 \) and \( 103 ± 20 \text{ W} \) for upright and supine cycling, respectively \( (P < 0.05) \). The severe-intensity work rates were \( 293 ± 41 \) and \( 258 ± 31 \text{ W} \) for upright and supine cycling, respectively \( (P < 0.05) \).

**Effect of Posture on \( \dot{V}O_2 \) Kinetics during Moderate-Intensity, Severe-Intensity, and Work-to-Work Exercise**

**Moderate-intensity cycling.** The \( \dot{V}O_2 \) responses to upright and unprimed supine moderate-intensity cycling are illustrated for a representative subject in Fig. 2, and the \( \dot{V}O_2 \) response parameters are presented in Table 1. The phase II \( \dot{V}O_2 \) \( \tau \) and \( G \) were not significantly different between conditions. The \([HHb] \) \( \tau + TD \) was not significantly different between conditions; however, the \([HHb] \) amplitude and \( \Delta[HHb]/\Delta\dot{V}O_2 \) (Fig. 3) were significantly greater for supine cycling. The \([HHb] \), HR, and blood [lactate] data for moderate-intensity cycling transitions are presented in Table 2.

**Severe-intensity cycling.** During baseline cycling, \( \dot{V}O_2 \) was not significantly different in the supine and upright positions. In absolute terms, end-exercise \( \dot{V}O_2 \) was significantly lower for supine than upright cycling; however, when expressed relative to posture-specific \( \dot{V}O_2 \text{peak} \), there was no significant difference between conditions. The phase II \( \dot{V}O_2 \) \( \tau \) \( (32 ± 11 \) and \( 33 ± 5 \) s for upright and supine, respectively) and \( G \) and the amplitude of the \( \dot{V}O_2 \) slow component \( (0.58 ± 0.23 \) and \( 0.66 ± 0.27 \text{ l/min} \) for upright and supine, respectively) were not significantly different between conditions. However, the MRT for \( \dot{V}O_2 \) was significantly greater for supine than upright exercise \( (64 ± 16 \) and \( 78 ± 19 \) s for upright and supine, respectively, \( P < 0.05) \).
Table 1. *O₂* uptake kinetics during moderate-intensity upright, supine unprimed, and supine primed cycling

<table>
<thead>
<tr>
<th></th>
<th>Upright</th>
<th>Unprimed</th>
<th>Primed</th>
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<tbody>
<tr>
<td><strong>Baseline O₂ uptake, l/min</strong></td>
<td>0.90 ± 0.99</td>
<td>1.03 ± 0.52*</td>
<td>1.20 ± 0.85†</td>
</tr>
<tr>
<td><strong>Phase II τ, s</strong></td>
<td>26 ± 7</td>
<td>35 ± 15</td>
<td>30 ± 12</td>
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<tr>
<td><strong>Phase II amplitude, l/min</strong></td>
<td>0.81 ± 0.20</td>
<td>0.67 ± 0.18*</td>
<td>0.69 ± 0.16*</td>
</tr>
<tr>
<td><strong>Phase II gain, ml-min⁻¹-W⁻¹</strong></td>
<td>8.8 ± 0.8</td>
<td>8.0 ± 0.9</td>
<td>8.3 ± 0.6</td>
</tr>
<tr>
<td><strong>End-exercise O₂ uptake</strong></td>
<td>1.71 ± 0.25</td>
<td>1.70 ± 0.20</td>
<td>1.88 ± 0.21†</td>
</tr>
<tr>
<td><strong>%peak O₂ uptake</strong></td>
<td>64 ± 5</td>
<td>65 ± 0</td>
<td>65 ± 0</td>
</tr>
<tr>
<td><strong>%peak O₂ uptake</strong></td>
<td>0.20 ± 0.16*</td>
<td>0.52* †</td>
<td>0.85* †</td>
</tr>
<tr>
<td><strong>End-exercise V˙O₂</strong></td>
<td>0.99 ± 0.67</td>
<td>1.03 ± 0.95</td>
<td>1.03 ± 0.95</td>
</tr>
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Values are means ± SD, τ, time constant. *Significantly different from upright (P < 0.05). †Significantly different from unprimed (P < 0.05).

Blood [lactate] and HR were similar between the upright and supine conditions. The [HHb] τ + TD (14 ± 2 and 15 ± 4 s for upright and supine, respectively) and the [HHb] amplitude [259 ± 119 and 291 ± 106 arbitrary units (AU) for upright and supine, respectively] were not significantly different between conditions; however, ∆[HHb]/∆V˙O₂ was significantly greater for supine cycling (105 ± 52 and 158 ± 76 AU·min⁻¹ for upright and supine, respectively, P < 0.05).

Work-to-work (moderate- to severe-intensity) cycling. The V˙O₂ responses to upright and unprimed supine work-to-work cycling are illustrated for a representative subject in Fig. 4, and the V˙O₂ response parameters are presented in Table 3. The phase II V˙O₂ τ was significantly longer for supine cycling (38 ± 10 vs. 54 ± 19 s, P < 0.05), but G was not significantly different between conditions. In absolute terms, the V˙O₂ slow component was less during supine work-to-work cycling; however, when expressed in relative terms, there was no significant difference between conditions. The MRT for V˙O₂ was significantly greater for supine than upright exercise. The fundamental [HHb] τ + TD and the ∆[HHb]/∆V˙O₂ during the fundamental and overall response phases (Fig. 3) were not significantly different for upright and supine work-to-work cycling; however, the [HHb] MRT was significantly shorter for the supine transition, and the [HHb] slow component was significantly reduced. The [HHb], HR, and blood [lactate] data for severe work-to-work cycling transitions are presented in Table 4.
Effect of Priming on Moderate and Work-to-Work Cycling Exercise in the Supine Position

The V\textsubscript{O}\textsubscript{2} responses to unprimed and primed moderate-intensity and work-to-work supine cycling are illustrated for a representative subject in Figs. 2 and 4, respectively. Group mean V\textsubscript{O}\textsubscript{2} response parameters after priming are presented in Tables 1 and 3, and group mean [H\textsubscript{Hbtot}], HR, and blood [lactate] data after priming are presented in Tables 2 and 4. Baseline V\textsubscript{O}\textsubscript{2}, HR, and blood [lactate] were significantly elevated before moderate-intensity supine cycling after priming, as was [H\textsubscript{bto}] (Fig. 5), and all these elevations were present at end exercise. Consequently, baseline V\textsubscript{O}\textsubscript{2}, HR, blood [lactate], and [H\textsubscript{bto}] were significantly elevated before the work-to-work severe-intensity cycling transition.

The phase II V\textsubscript{O}\textsubscript{2} \(\tau\), amplitude, and G were not significantly different during moderate-intensity supine cycling after priming. However, during severe-intensity work-to-work cycling, phase II V\textsubscript{O}\textsubscript{2} \(\tau\) was significantly reduced (54 ± 19 vs. 34 ± 9 s, \(P < 0.05\)). Furthermore, after priming, \(\tau\) \textsubscript{p} was not significantly different from the upright work-to-work control condition. The extent of the reduction of \(\tau\) \textsubscript{p} for supine work-to-work cycling after priming was significantly correlated with the difference in \(\tau\) between the upright and unprimed supine work-to-work values (\(r = 0.88, P < 0.01\)). Similar to \(\tau\) \textsubscript{p}, V\textsubscript{O}\textsubscript{2} MRT was significantly shorter and was not significantly different from the upright unprimed control condition for work-to-work supine cycling after priming. No significant difference in V\textsubscript{O}\textsubscript{2} fundamental absolute or slow component (absolute or relative) amplitude was observed, although \(\Delta V\textsubscript{O2(26-2)}\) was significantly reduced. There were no significant differences in fundamental [H\textsubscript{Hbtot}] \(\tau\) + TD, fundamental and overall \(\Delta[H\textsubscript{Hbtot}]/\Delta V\textsubscript{O2}\), or [H\textsubscript{Hbtot}] MRT for work-to-work severe-intensity supine cycling after priming. However, the [H\textsubscript{Hbtot}] slow component tended to be lower across the group (\(P = 0.07\)) and was completely eliminated in three subjects during the primed work-to-work supine transition.

For unprimed and primed moderate-intensity supine cycling, the mean iEMG at the end of exercise was not significantly different from \(\tau\) \textsubscript{p} at minute 2, and \(\Delta\text{EMG}_{(end-2)}\) was not affected by priming. In contrast, for unprimed and primed work-to-work supine cycling, the mean iEMG at the end of exercise was significantly greater than that measured at minute 2. The \(\Delta\text{EMG}_{(end-2)}\) for unprimed and primed work-to-work cycling was not significantly affected by priming. The group mean iEMG responses at minute 2 and end exercise for moderate-intensity and work-to-work severe-intensity supine cycling in the unprimed and primed states are depicted in Fig. 6.

Fig. 5. Total Hb concentration ([H\textsubscript{bto}]\textsubscript{tot}) at 60-s intervals during moderate-intensity and work-to-work supine cycling in the unprimed (○) and primed (■) states. Arrows, abrupt transitions to the higher work rates. AU, arbitrary units. Values are means ± SD. *Significantly different from unprimed (\(P < 0.05\)). A significant elevation indicative of hyperemia is present throughout the 4-min moderate-intensity bout and for the first 3 min of the 6-min severe-intensity bout after priming.
During work-to-work cycling and was not altered by priming. An increase in iEMG from baseline (slowed by \( \sim 50\% \)) was necessary to compensate for a blunted circulatory response. These effects (a lengthened \( \tau_p \) and faster [HHb] time course with no change in the amplitude of estimated O2 extraction) during supine compared with upright work-to-work exercise are in contrast to our observations during moderate-intensity exercise (see above). These data suggest that muscle O2 extraction could not be increased sufficiently to compensate for reduced muscle blood flow during work-to-work exercise in the supine position, resulting in slower phase II \( \dot{V}_O_2 \) kinetics. This is consistent with what would be predicted for the population of higher-order muscle fibers that would be expected to predominantly contribute to power production across the work-to-work transition (24, 36). These fibers are known to exhibit a faster and more pronounced decrease in microvascular Po2 at the onset of contractions, suggesting a greater reliance on fractional O2 extraction to maintain a given oxidative flux (3, 40).

**DISCUSSION**

The principal finding of this investigation was that the characteristic slowing of phase II \( \dot{V}_O_2 \) kinetics that is observed for severe-intensity upright cycling transitions initiated from an elevated baseline was amplified when the same relative intensity transition was performed in the supine position. Specifically, unlike moderate-intensity supine cycling, where muscle fractional O2 extraction was increased to preserve \( \dot{V}_O_2 \) kinetics, O2 extraction during work-to-work supine cycling was unchanged compared with the upright control condition and the \( \dot{V}_O_2 \) \( \tau_p \) was thus lengthened by \( \sim 50\% \). However, the performance of prior high-intensity exercise shortened \( \tau_p \) during supine work-to-work severe-intensity cycling, so that it was not significantly different from \( \tau_p \) in the upright position.

**Effect of Posture on \( \dot{V}_O_2 \) Kinetics During Moderate-Intensity and Work-to-Work Cycle Exercise**

Consistent with our hypothesis, \( \tau_p \) was not significantly different for supine compared with upright moderate-intensity cycling. However, NIRS data indicated a marked difference in the degree of Hb/Mb desaturation that was required to maintain an unimpaired \( \dot{V}_O_2 \) response in the supine position. Specifically, \( \Delta[Hb]/\Delta\dot{V}_O_2 \) was significantly greater during moderate-intensity supine than moderate-intensity upright cycling, which indicates that fractional O2 extraction was enhanced throughout the bout (Fig. 3). The gravitational assist to muscle blood flow is absent during supine exercise, and MacDonald et al. (39) showed slower kinetics of femoral artery blood flow during low-intensity knee extension/flexion exercise in the supine than upright position. Therefore, it is likely that increased O2 extraction during moderate-intensity supine cycling was necessary to compensate for a blunted circulatory response.

Also consistent with our hypothesis, \( \tau_p \) was significantly lengthened for severe-intensity supine cycling transitions initiated from a moderate-intensity baseline, in relation to transitions to severe-intensity supine cycling from an unloaded baseline (slowed by \( \sim 65\% \)) and severe-intensity upright cycling from a moderate-intensity baseline (slowed by \( \sim 50\% \)). Estimated muscle fractional O2 extraction (as \( \Delta[Hb]/\Delta\dot{V}_O_2 \)) was not different between work-to-work transitions in the supine and upright positions, but faster [HHb] kinetics (a reduced [HHb] slow component and shortened [HHb] MRT) were apparent for supine exercise. These effects (a lengthened \( \dot{V}_O_2 \) \( \tau_p \) and faster [HHb] time course with no change in the amplitude of estimated O2 extraction) during supine compared with upright work-to-work exercise are in contrast to our observations during moderate-intensity exercise (see above). These data suggest that muscle O2 extraction could not be increased sufficiently to compensate for reduced muscle blood flow during work-to-work exercise in the supine position, resulting in slower phase II \( \dot{V}_O_2 \) kinetics. This is consistent with what would be predicted for the population of higher-order muscle fibers that would be expected to predominantly contribute to power production across the work-to-work transition (24, 36). These fibers are known to exhibit a faster and more pronounced decrease in microvascular Po2 at the onset of contractions, suggesting a greater reliance on fractional O2 extraction to maintain a given oxidative flux (3, 40).

**Effect of Priming on Moderate-Intensity and Work-to-Work Cycle Exercise in the Supine Position**

Consistent with our hypothesis, the performance of prior high-intensity exercise resulted in a significant speeding of phase II \( \dot{V}_O_2 \) kinetics during work-to-work, but not moderate-intensity, supine cycling. Specifically, after priming, \( \tau_p \) during work-to-work supine cycling was reduced by \( \sim 33\% \) and was no longer significantly different from the upright work-to-work value. Furthermore, the extent of the reduction in \( \tau_p \) with priming was significantly correlated with the difference in \( \tau_p \) between the upright and supine conditions. Collectively, these findings suggest that priming counteracted the adverse effects of the supine body posture during work-to-work cycling.

It is well documented that prior high-intensity exercise that results in residual metabolic acidosis facilitates convective and diffusive components of muscle O2 delivery, increases muscle oxidative enzyme activity, and alters motor unit recruitment profiles during subsequent exercise (1, 6, 10, 14, 16, 20, 25, 27, 34, 47, 48, 51). Collectively, these changes accelerate the overall \( \dot{V}_O_2 \) kinetics during subsequent high-intensity exercise (21, 27), due predominantly to a marked reduction of the \( \dot{V}_O_2 \) slow component with increased fundamental phase absolute amplitude, but no change in \( \tau_p \) (1, 6–9, 20, 27, 32, 47–49, 57). We recently showed that priming exercise results in these same effects when severe-intensity upright cycle exercise is initiated from a moderate-intensity baseline (16). However, in other circumstances where \( \tau_p \) is rather long in the control condition (i.e., more than \( \sim 30–35 \) s), a reduction of \( \tau_p \) has been reported (14, 23, 25, 33). The priming effect that we observed for supine work-to-work cycling (reduced \( \tau_p \) with unchanged fundamental and slow component amplitudes) in the present study is different from the effect we reported previously for upright work-to-work cycling (altered fundamental and slow component amplitudes with unchanged \( \tau_p \)) (16). This difference is consistent with the findings of Jones et al. (25), who reported that, during upright cycling, priming altered the amplitudes of the fundamental and slow components without changing \( \tau_p \), whereas during supine cycling, priming reduced \( \tau_p \) without
changing the response phase amplitudes. Whether priming exercise alters the response phase amplitudes or the \( \tau_p \) during subsequent exercise, therefore, appears to be related to the adequacy of \( O_2 \) delivery relative to metabolic rate in the control condition.

A model that explains why \( \tau_p \) might or might not be influenced by altered \( O_2 \) delivery has been advanced by Poole et al. (43, 44). In this model, it is proposed that there is an “\( O_2 \) delivery-independent” zone within which changing \( O_2 \) delivery will not substantially impact \( \tau_p \) (e.g., during moderate-intensity exercise in healthy young subjects) and, beyond the so-called “tipping point,” an “\( O_2 \) delivery-dependent” zone within which enhancing or reducing \( O_2 \) delivery will shorten or lengthen the \( \tau_p \), respectively (43, 44). Application of this model to the present study might suggest that the muscle fibers predominantly involved in moderate-intensity cycling lie to the right of the tipping point, whereas the fibers involved in a moderate-to-severe work-to-work transition operate at or close to the tipping point, with the supine position placing them firmly in the \( O_2 \) delivery-dependent zone.

We observed a significant difference between iEMG at minute 2 and end exercise during work-to-work supine cycling in the unprimed and primed states (Fig. 6), and the magnitude of this difference [i.e., \( \Delta iEMG_{end-2} \)] was unaffected by priming. These results contrast with those that we reported previously for primed work-to-work cycling in the upright position, which was characterized by a significant reduction of \( \Delta iEMG_{end-2} \), such that the end-exercise value was no longer different from the minute 2 value after priming (16). One explanation for the increased \( VO_2 \) fundamental component amplitude and reduced \( VO_2 \) slow component amplitude after priming in the upright position is that motor unit recruitment is increased during the early stages of high-intensity exercise, such that the requirement for additional fiber activation as exercise proceeds and the associated \( VO_2 \) cost of that activation are reduced (1, 6, 10). The iEMG results in the present study suggest that the characteristic slow component reduction that is present under “normal” circumstances after priming might be absent in the supine posture, because fiber activation is not altered in a similar manner. Why the effect of prior exercise on subsequent fiber activation would be different for supine compared with upright cycling is unclear but might be linked to the unusual nature of cycling in the supine position. In contrast to our previous study (25) in which subjects exercised at the same absolute work rate in the supine and upright positions, the subjects in the present study cycled at the same relative intensity (70% \( \Delta \)) in the supine and upright positions. This was done to better match the physiological demands of exercise in the different postures. However, a lack of familiarity for most subjects with this form of exercise could mandate an altered fiber activation pattern as exercise proceeds that is independent of favorable metabolic alterations induced by priming.

It is also possible that, despite increased \( O_2 \) availability at the onset of primed cycling, the supine posture alters perfusion sufficiently to accelerate removal of this effect as the bout proceeds. Previous research indicates that, for upright cycling, the characteristic prior-exercise effect declines in a time-dependent manner but is preserved for \( \geq 20–30 \) min (1, 8). Although it has yet to be established which specific residual physiological alteration(s) underpins this phenomenon, it is interesting to note that the increase in \( [Hb_{sat}] \) that we observed at the onset of primed work-to-work supine cycling in the present study (presumably reflecting hyperemia) was abolished after 3 min of exercise (Fig. 5). It is well established that \( O_2 \) availability exerts a profound influence on motor unit recruitment (41, 42), particularly in high-threshold motor units comprising fast-twitch fibers, which have been implicated in the development of the \( VO_2 \) slow component (2, 35, 36, 45, 46). Therefore, it is possible that, despite facilitation of the initial \( VO_2 \) response during supine exercise after priming, the elevated tissue oxygenation is relatively short-lived, resulting in a more rapid development of fatigue and a continued drive for motor unit recruitment similar to that observed in the unprimed state.

Although priming did not reduce the \( VO_2 \) slow component in the present study, it did shorten the time delay before its emergence. Jones et al. (25) also reported a significant reduction in the slow component time delay for primed supine cycling with no similar effect for primed upright cycling. The \( VO_2 \) slow component time delay is not altered by priming during upright cycle exercise (6, 7, 9, 16, 17, 20, 49). The reason for this disparity is unclear. However, if the \( VO_2 \) slow component is related, at least in part, to the protracted response profiles of initially recruited fibers with extremely slow \( VO_2 \) kinetics (56), a reduced time delay during supine exercise might reflect an accelerated phase II \( VO_2 \) response in these fibers. We previously speculated that such a speeding might be indistinguishable from reciprocal changes in the amplitudes of the \( VO_2 \) fundamental and slow components during upright cycling (17).

Other than an elevated baseline and end-exercise \( VO_2 \), we observed no significant differences in \( VO_2 \) kinetics during moderate-intensity supine cycling after priming. Specifically, even though HR, blood [lactate], and [\( Hb_{sat} \)] were elevated at the onset of and throughout the moderate-intensity primed supine bout, \( \tau_p \) was not altered. Prior research indicates that moderate-intensity cycling in the upright position is unaffected by moderate- or high-intensity prior exercise (6, 19, 21; cf. Ref. 23). Therefore, given that the supine posture did not compromise \( VO_2 \) kinetics for moderate-intensity cycling in the present study (i.e., \( \tau_p \) was similar in the supine and upright positions), this result is not surprising. Moreover, \( \Delta [Hb] \Delta VO_2 \) was unaffected during moderate-intensity supine cycling after priming, which supports the notion that \( O_2 \) extraction by the involved muscle fibers had already increased sufficiently to counteract the supine posture. In this regard, it is interesting to note that priming also did not enhance \( \Delta [Hb] \Delta VO_2 \) during work-to-work supine cycling (Fig. 3). This indicates that the speeding of \( VO_2 \) kinetics after priming during supine work-to-work exercise was related to an increased bulk muscle blood flow and/or better local matching of perfusion to metabolic rate, rather than any changes in muscle fractional \( O_2 \) extraction, which might have been close to maximal in the control condition.

Methodological Considerations

It should be cautioned that our NIRS measurements were made at only one site (the vastus lateralis), and we cannot be certain that the conclusions reached from the [\( Hb \)] response measured at that site hold true for other regions of the quadriceps. Indeed, there is some evidence that the vastus lateralis has a higher fraction of type II fibers and lower blood flow than these other regions (29). Recent studies showed that the pattern...
of quadriceps muscle deoxygenation following the onset of heavy exercise displays significant intersite heterogeneity (30) and that this heterogeneity is reduced after a priming bout of heavy exercise (48). Although the reduced heterogeneity of muscle deoxygenation following priming was not correlated with changes in VO₂ kinetics (i.e., reduced VO₂ slow component) during upright cycle exercise (48), it remains to be established whether a more homogenous distribution of blood flow might be, in part, responsible for the faster phase II VO₂ kinetics observed after priming in the supine position (25; present study).

Boone et al. (4) recently proposed that, because of the existence of an additional amount of unmeasured (negative) internal work, the measured VO₂ at very low baseline work rates is higher than the value that would be expected from backextrapolation of the VO₂ response to moderate-intensity exercise. The authors argued that this could influence VO₂ kinetics and might, in part, explain the greater functional gain of the VO₂ response that is measured during cycling when the baseline work rate is above compared with below ~50 W (5, 56). However, although it is possible that the influence of internal work could contribute to the differences in the VO₂ gain between moderate-intensity and work-to-work exercise in the present study, it should be stressed that this would not influence our within-condition comparisons (i.e., the effects of body position and priming on VO₂ kinetics during moderate-intensity or work-to-work severe-intensity transitions). It is also important to note that muscle phosphocreatine kinetics, which closely reflect muscle VO₂ kinetics (43), provide evidence of slower dynamics and an increased gain for transitions from moderate- to heavy-intensity exercise compared with transitions from rest to moderate-intensity exercise, where no similar internal work disparity would be expected (28).

In conclusion, we have shown notable differences in the capacities for the recruited fractions of the motor unit pool to adapt to altered muscle perfusion during supine cycle exercise. Specifically, by dividing transitions to severe-intensity cycling into two discrete steps, we attempted to isolate the response characteristics of fibers that are positioned “lower” and “higher” in the recruitment hierarchy. During moderate-intensity supine exercise, the results indicate that muscle fractional VO₂ extraction was increased in the recruited fibers, such that VO₂ kinetics were preserved. Conversely, during transitions from moderate- to severe-intensity work-to-work exercise, which would oblige the recruitment of a different population of fibers situated higher in the recruitment order, muscle fractional VO₂ extraction was unchanged and VO₂ kinetics were markedly slowed. The fiber type specificity in the susceptibility to reduced perfusion pressure suggested by our results is consistent with previous findings of a faster and more pronounced fall in microvascular PO₂ at the onset of contractions (reflecting a greater reliance on fractional O₂ extraction) in fast-twitch muscle (3, 40). Furthermore, priming exercise did not alter VO₂ kinetics during moderate-intensity supine cycling but did accelerate the VO₂ response during work-to-work transitions in the supine position, restoring τ₀ to the value that was observed during upright work-to-work exercise. This latter effect occurred in the absence of increased muscle fractional O₂ extraction, indicating that the priming-induced facilitation of blood flow matched increased O₂ utilization in the involved fibers and resulted in faster VO₂ kinetics. Collectively, these findings suggest that, during supine cycling, priming speeds VO₂ kinetics by enhancing perfusion in the higher-order (i.e., type II) fibers, which are known to be especially sensitive to limitations in O₂ supply.

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

REFERENCES


