Kinetics of oxygen uptake during supine and upright heavy exercise

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1Applied Physiology Laboratory, Kobe Design University, Kobe 651–2196; 2Laboratory of Exercise and Sports Science, Yokohama City University, Yokohama 236–0027; 3Faculty of Human Development, Kobe University, Kobe 657–0011; 4Department of Exercise Science and Physiology, Hiroshima Women’s University, Hiroshima 734–8558 Japan; and 5Department of Kinesiology, Kansas State University, Manhattan, Kansas 66506-0302

Koga, Shunsaku, Tomoyuki Shiojiri, Manabu Shibasaki, Narihiko Kondo, Yoshiyuki Fukuba, and Thomas J. Barstow. Kinetics of oxygen uptake during supine and upright heavy exercise. J. Appl. Physiol. 87(1): 253–260, 1999.—It is presently unclear how the fast and slow components of pulmonary oxygen uptake (VO2) kinetics would be altered by body posture during heavy exercise [i.e., above the lactate threshold (LT)]. Nine subjects performed transitions from unloaded cycling to work rates representing moderate (below the estimated LT) and heavy exercise (VO2 equal to 50% of the difference between LT and peak VO2) under conditions of upright and supine positions. During moderate exercise, the steady-state increase in VO2 was similar in the two positions, but VO2 kinetics were slower in the supine position. During heavy exercise, the rate of adjustment of VO2 to the 6-min value was also slower in the supine position but was characterized by a significant reduction in the amplitude of the fast component of VO2, without a significant slowing of the phase 2 time constant. However, the amplitude of the slow component was significantly increased, such that the end-exercise VO2 was the same in the two positions. The changes in VO2 kinetics for the supine vs. upright position were paralleled by a blunted response of heart rate at 2 min into exercise during supine compared with upright heavy exercise. Thus the supine position was associated with not only a greater amplitude of the slow component for VO2 but also, concomitantly, with a reduced amplitude of the fast component; this latter effect may be due, at least in part, to an attenuated early rise in heart rate in the supine position.

None of the previous studies (8, 9, 23), however, partitioned VO2 kinetics during supine-heavy-intensity exercise [i.e., above the lactate threshold (LT)] into discrete components, so as to elucidate the mechanism by which the kinetics appeared slowed in the supine position. Furthermore, the previous studies (8, 23) did not repeat each exercise test to improve the dynamic resolution of VO2 kinetics during supine heavy exercise. It has been proposed that the slower VO2 kinetics and the presence of a slow component during heavy exercise in the upright position reflect inadequate perfusion and O2 delivery to the working muscles (14, 24), which results in lactic acidosis (7, 30, 32). Therefore, if supine exercise is associated with a relative perfusion inadequacy to the working muscles, this should be exaggerated during heavy exercise. On the basis of the above findings, we hypothesized that heavy exercise in the supine position would be associated with a slower adjustment of the predominant component of VO2 and a larger slow component compared with the upright position. In addition, we characterized the off-kinetics for VO2 in the two positions to investigate whether any alterations in kinetics seen during exercise would also be translated into parallel differences in recovery.

METHODS

Subjects

Nine healthy subjects (8 men and 1 woman, age 23.8 ± 9.2 yr, height 172.8 ± 6.7 cm, and weight 65.8 ± 10.6 kg) volunteered for this study. After a detailed explanation of the study, informed consent was obtained. The study was approved by the Human Subjects Committee of Kobe Design University.

Protocol

Incremental-exercise tests. A ramp exercise protocol (25 W/min), preceded by 4-min unloaded cycling on a cycle ergometer, was utilized to estimate each individual’s LT and peak VO2 (the highest VO2 achieved during exercise). Responses to upright and supine posture conditions were tested on separate days. In the supine position, the crank shaft was positioned 30 cm above the level of the back. Handgrips were available for support. The VO2 at the LT was estimated as the break point in the plot of CO2 output (VCO2) against a function of VO2 (V-slope method) (6). The break point was determined by a computer program that defined the VO2 above which VCO2 increased faster than VO2, without hyperventilation.

Constant work rate tests. Exercise transition tests were conducted under the two posture conditions on separate days. Each constant work rate exercise test was performed for 6
min. The steady-state work rate that corresponded to the LT was defined as the work rate that occurred 45 s before the LT was actually exceeded during the ramp test. The 45-s offset represents an average correction for the delay in the VO2 response relative to the ramp forcing function. The moderate work rate used for both posture conditions corresponded to a VO2 of ~80% of the LT determined for the upright position, whereas the heavy exercise work rate was estimated to require a VO2 equal to ~50% of the difference (Δ) between the subject’s LT and peak VO2, i.e., a value of (LT + 0.50Δ), on the basis of the initial VO2-to-work rate ratio observed during the ramp exercise in the upright position (Table 1). The exercise was preceded by 3 min, and was followed by 6 min, of unloaded cycling at a pedal frequency of 60 rpm. To minimize random noise and enhance the underlying response patterns for the moderate work rate tests, subjects performed a total of four to six repetitions of the exercise transition under each posture condition. Subjects performed two to three exercise transitions under each posture condition for the heavy work rate.

**Measurements**

Subjects breathed through a low-resistance valve (Hans-Rudolph) connected to two pneumotachographs for measurement of inspiratory and expiratory flows, as previously described (20). Each system was calibrated repeatedly by inputting known volumes of room air at various mean flows and flow profiles. Respired gases were analyzed by mass spectrometry (model MGA-1100, Perkin-Elmer) from a sample drawn continuously from the mouthpiece. Precision-analyzed gas mixtures were used for calibration. Alveolar gas exchange variables were calculated breath by breath according to the algorithms of Beaver et al. (5). Heart rate (HR) was continuously monitored via a three-lead electrocardiogram.

**Analysis**

Individual responses during the baseline-to-exercise transitions were time interpolated to 1-s intervals. Responses to exercise were further averaged across all transitions for each subject and condition. To further reduce the breath-to-breath noise to enhance the underlying characteristics, each average response was smoothed with a five-point moving average filter. For both the on- and off-transients, the response curve of VO2 was fit by a three-term exponential function that included amplitudes, time constants, and time delays, by nonlinear least squares regression techniques (Fig. 1) (3, 12). The computation of best-fit parameters was chosen by the program to minimize the sum of the squared differences between the fitted function and the observed response. The first exponential term started with the onset of exercise, and the second and third terms began after independent time delays

\[
\begin{align*}
V_{O2}(t) & = V_{O2}(b) + A_0 \left(1 - e^{-t/\tau_1}\right) \quad \text{Phase 1 (initial component)} \\
& + A_1 \left(1 - e^{-t/(\tau_2-\Delta)}\right) \quad \text{Phase 2 (fast primary component)} \\
& + A_2 \left(1 - e^{-t/(\tau_2-\Delta)}\right) \quad \text{Phase 3 (slow component)}
\end{align*}
\]

where \(V_{O2}(b)\) is the unloaded cycling baseline value; \(A_0, A_1,\) and \(A_2\) are the asymptotic values for the exponential terms; \(\tau_1, \tau_2,\) and \(\tau_3\) are the time constants; and \(\tau_2\) and \(\tau_3\) are the time delays. The phase 1 term was terminated at the start of the slow exponential term, \(\Delta\) and assigned the value for that time \(A_0\).

Between the fitted function and the observed response. The first exponential term started with the onset of exercise, and the second and third terms began after independent time delays

\[
A_0 = A_0 \left(1 - e^{-t/\tau_{D1}}\right)
\]

The physiologically relevant amplitude of the fast primary exponential component during phase 2 \(A_1\) was defined as the sum of \(A_0 + A_1\). Because of concerns regarding the validity of using the extrapolated asymptotic value for the slow component \(A_2\) for comparisons, we used the value of the slow exponential function at the end of exercise, defined as \(A_2^E\). Because the VO2 response during moderate-intensity exercise (LT) reaches a new steady state within 3 min after the onset of exercise in normal subjects, the slow exponential term invariably dropped out during the iterative-fitting procedure. In addition, to facilitate comparison across the subjects and different absolute work rates, the gain of the fast primary response \(G_1 = A_1/\text{work rate} \) and relative contribution of slow component to the overall increase in VO2 at end exercise \(A_2^E / (A_1 + A_2)\) were calculated.

**Recovery kinetics for VO2 were initially analyzed with Eq. 1. However, preliminary findings (see RESULTS) demonstrated that, for both the supine and upright heavy-exercise conditions, \(\tau_{D1}\) in recovery converged back to a value that was not significantly different from that found for \(\tau_2\). Thus we subsequently fit each of the recovery VO2 curves with a model similar to Eq. 1, except that after phase 1 both the primary and slow exponential terms shared the same time delay \(\tau_{D1}\), equivalent to the duration of phase 1 in recovery (3, 12).**

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**Table 1. Peak and submaximal responses to upright and supine exercise**

<table>
<thead>
<tr>
<th></th>
<th>Upright</th>
<th>Supine</th>
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</thead>
<tbody>
<tr>
<td>Peak work rate, W</td>
<td>283.9 ± 35.0</td>
<td>244.7 ± 26.4*</td>
</tr>
<tr>
<td>Peak VO2, l/min</td>
<td>3.29 ± 0.51</td>
<td>2.90 ± 0.40*</td>
</tr>
<tr>
<td>Peak HR, beats/min</td>
<td>184.5 ± 3.1</td>
<td>167.7 ± 12.5*</td>
</tr>
<tr>
<td>VO2 at LT, l/min</td>
<td>1.68 ± 0.40</td>
<td>1.50 ± 0.39*</td>
</tr>
<tr>
<td>Moderate work (&lt;LT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work rate, W</td>
<td>78 ± 5</td>
<td>78 ± 5</td>
</tr>
<tr>
<td>%LT</td>
<td>77.4 ± 13.7</td>
<td>87.0 ± 18.7*</td>
</tr>
<tr>
<td>Heavy work (&gt;LT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work rate, W</td>
<td>175 ± 26</td>
<td>175 ± 26</td>
</tr>
<tr>
<td>%Δ</td>
<td>55.8 ± 13.6</td>
<td>79.3 ± 15.3*</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 9 subjects. VO2 = O2 uptake; HR, heart rate; LT, estimated lactate threshold; %LT, relative intensity calculated as steady-state VO2 = 100/LT; %Δ, relative intensity calculated as (end-exercise VO2 - LT)/100/(peak VO2 - LT). *Significantly different from upright, P < 0.05.
The overall kinetics of the response was determined from mean response time (MRT). It was calculated by fitting the response data of VO₂ to a monoexponential function that included a single amplitude and time constant, starting from the onset of the transition.

For the comparison with the associated VO₂ responses, the baseline, 2-min, and end-exercise values of HR and oxygen pulse (VO₂-to-HR ratio (VO₂/HR)) during exercise were calculated. Furthermore, the kinetics of HR (half time) were determined in terms of the response time to achieve 50% of change in HR from baseline to end-exercise. The values of minute ventilation (Ve) and respiratory exchange ratio (R) during exercise were also calculated.

Statistics

Data are presented as means ± SD. The data were analyzed by using a repeated-measures analysis of variance design. Significant results were further analyzed by Scheffé’s post hoc test. Significance was declared at P < 0.05.

RESULTS

Incremental Exercise

Supine posture led to significant reductions in peak work rate, peak VO₂, estimated LT, and peak HR compared with upright posture (Table 1).

Moderate Constant Work Rate Exercise

The response for VO₂ from baseline to moderate exercise is shown in a representative subject for the two conditions in Fig. 2A. During moderate exercise, the steady-state increase in VO₂ (as A₁) and the kinetics in phase 2 (as τ₁) were similar for the two positions, but the overall VO₂ kinetics (as MRT) were slower in supine compared with the upright position (Table 2).

The O₂ deficit was calculated for the two positions, on the assumption that the end-exercise VO₂ (at 6 min) represented the O₂ requirement for the exercise. The O₂ deficit was similar between the supine (0.54 ± 0.20 liter) and the upright position (0.42 ± 0.27 liter).

Heavy Constant Work Rate Exercise

Associated with the decrease in peak VO₂ and the LT, supine posture resulted in an increase for the relative intensity of the heavy work rate, as denoted by %Δ, compared with that seen in the upright position (Table 1). The response for VO₂ during heavy exercise in a representative subject is shown for the two conditions in Fig. 2B. The primary time constant (τ₁) was not significantly longer, but instead the amplitude (A₁) and the gain (G₁) of the fast component of VO₂ during heavy exercise were significantly reduced in supine compared with upright position (Table 3). This was compensated for by an increase in both the absolute (A₂) and the

Table 2. VO₂ response parameters for moderate exercise

<table>
<thead>
<tr>
<th></th>
<th>Upright</th>
<th>Supine</th>
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</thead>
<tbody>
<tr>
<td>On-response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL, l/min</td>
<td>0.53 ± 0.07</td>
<td>0.50 ± 0.09</td>
</tr>
<tr>
<td>A₀, l/min</td>
<td>0.27 ± 0.11</td>
<td>0.16 ± 0.12*</td>
</tr>
<tr>
<td>τ₀, s</td>
<td>60.2 ± 113.3</td>
<td>43.0 ± 68.6</td>
</tr>
<tr>
<td>A₁, l/min</td>
<td>0.76 ± 0.09</td>
<td>0.76 ± 0.08</td>
</tr>
<tr>
<td>τ₁, s</td>
<td>21.2 ± 8.2</td>
<td>27.7 ± 13.4</td>
</tr>
<tr>
<td>TD, s</td>
<td>25.6 ± 6.4</td>
<td>22.1 ± 8.4</td>
</tr>
<tr>
<td>G₁, ml·min⁻¹·W⁻¹</td>
<td>9.7 ± 0.6</td>
<td>9.7 ± 0.6</td>
</tr>
<tr>
<td>MRT, s</td>
<td>35.7 ± 8.8</td>
<td>44.4 ± 12.4*</td>
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<tr>
<td>Off-response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL, l/min</td>
<td>1.27 ± 0.12</td>
<td>1.25 ± 0.14</td>
</tr>
<tr>
<td>A₀, l/min</td>
<td>0.17 ± 0.07†</td>
<td>0.09 ± 0.05*</td>
</tr>
<tr>
<td>τ₀, s</td>
<td>4.6 ± 10.2</td>
<td>0.6 ± 0.6</td>
</tr>
<tr>
<td>A₁, l/min</td>
<td>0.78 ± 0.10</td>
<td>0.74 ± 0.09†</td>
</tr>
<tr>
<td>τ₁, s</td>
<td>33.0 ± 6.4</td>
<td>34.0 ± 6.3</td>
</tr>
<tr>
<td>TD, s</td>
<td>18.0 ± 5.7†</td>
<td>16.4 ± 4.3</td>
</tr>
<tr>
<td>G₁, ml·min⁻¹·W⁻¹</td>
<td>9.9 ± 0.8</td>
<td>9.5 ± 0.8†</td>
</tr>
<tr>
<td>MRT, s</td>
<td>39.3 ± 7.8</td>
<td>45.7 ± 10.3</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 9 subjects. BL, baseline; A₀ and A₁, amplitudes of response; τ₀ and τ₁, time constants; TD, time delay; G₁, gain of response (A₁/work rate); MRT, mean response time. *Significantly different from upright, P < 0.05. †Significantly different from on-response, P < 0.05.
slow components. A significant effect of posture on either the fast or slow terms). The results are given in Table 4. There was no common time delay for the fast and slow exponential for the two positions. The overall VO₂ kinetics (MRT) were slower in supine compared with the upright position. When Eq. 1 was used to model the recovery kinetics of VO₂ after heavy exercise in both the supine and upright positions, TD₂ converged back to a value that, on average, was not significantly different from TD₁ (upright: TD₂ = 25.7 ± 16.3, TD₁ = 15.4 ± 2.9 s, P > 0.05; supine: TD₂ = 29.5 ± 19.4, TD₁ = 17.4 ± 3.4 s, P > 0.05). Given these results, we fit the recovery VO₂ response for each condition and subject with a modified version of Eq. 1, where TD₂ was set equal to TD₁ (i.e., a common time delay for the fast and slow exponential terms). The results are given in Table 4. There was no significant effect of posture on either the fast or slow component of the off-transient response of VO₂. Thus the relative contribution of the slow component to the overall VO₂ response was retained during recovery from heavy-intensity exercise, irrespective of positions. Therefore, the amplitudes for both the fast (A₁) and slow components (A₂), and the time constant for the fast component (τ₁), were similar between exercise and recovery responses for each position. These results suggest a symmetry between the exercise and recovery responses of VO₂ for this relative intensity (56–80%Δ) of heavy exercise, especially with regard to the amplitude and time constant of the fast exponential component.

The O₂ deficit was significantly greater in the supine (2.32 ± 0.42 liters) compared with upright position (1.86 ± 0.52 liters, P < 0.05).

HR, VO₂/HR, VE, and R Responses

The response for HR from baseline to moderate exercise is shown in a representative subject for the two conditions in Fig. 3A. The end-exercise HR value was significantly lower in the supine compared with the upright position (Table 5).

The response for HR from baseline to heavy exercise is shown in a representative subject for the two conditions in Fig. 3B. The 2-min value was significantly lower in the supine compared with the upright position (Table 5). The reduced early response of HR during

### Table 3. VO₂ on-response parameters for heavy exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Upright</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL, l/min</td>
<td>0.51 ± 0.05</td>
<td>0.51 ± 0.09</td>
</tr>
<tr>
<td>∆EE VO₂, l/min</td>
<td>2.14 ± 0.30</td>
<td>2.16 ± 0.31</td>
</tr>
<tr>
<td>A₀, l/min</td>
<td>0.58 ± 0.17</td>
<td>0.39 ± 0.15*</td>
</tr>
<tr>
<td>τ₂, s</td>
<td>34.6 ± 57.3</td>
<td>38.5 ± 39.1</td>
</tr>
<tr>
<td>A₁, l/min</td>
<td>1.83 ± 0.36</td>
<td>1.62 ± 0.31*</td>
</tr>
<tr>
<td>τ₁, s</td>
<td>26.9 ± 9.2</td>
<td>24.5 ± 9.8</td>
</tr>
<tr>
<td>TD₁, s</td>
<td>21.8 ± 5.2</td>
<td>23.1 ± 5.9</td>
</tr>
<tr>
<td>A₂, l/min</td>
<td>0.30 ± 0.17</td>
<td>0.55 ± 0.17*</td>
</tr>
<tr>
<td>τ₂, s</td>
<td>114.3 ± 44.0</td>
<td>133.5 ± 144.8</td>
</tr>
<tr>
<td>TD₂, s</td>
<td>126.7 ± 48.4</td>
<td>96.1 ± 28.6*</td>
</tr>
<tr>
<td>G₁ ml·min⁻¹·W⁻¹</td>
<td>10.4 ± 1.0</td>
<td>9.2 ± 1.0*</td>
</tr>
<tr>
<td>A₂/(A₁ + A₂)</td>
<td>0.15 ± 0.09</td>
<td>0.25 ± 0.08*</td>
</tr>
<tr>
<td>MRT, s</td>
<td>49.6 ± 12.9</td>
<td>65.5 ± 12.2*</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 9 subjects. ∆EEVO₂: increase above baseline in VO₂ at end exercise. A₂/(A₁ + A₂), relative contribution of slow component to net increase in VO₂ at end exercise. *Significantly different from upright, P < 0.05.

### Table 4. VO₂ off-response parameters for heavy exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Upright</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL, l/min</td>
<td>2.52 ± 0.36</td>
<td>2.59 ± 0.39</td>
</tr>
<tr>
<td>A₀, l/min</td>
<td>0.40 ± 0.12†</td>
<td>0.22 ± 0.10*</td>
</tr>
<tr>
<td>τ₀, s</td>
<td>1.9 ± 2.6</td>
<td>5.3 ± 7.9</td>
</tr>
<tr>
<td>A₁, l/min</td>
<td>1.72 ± 0.35</td>
<td>1.55 ± 0.53</td>
</tr>
<tr>
<td>τ₁, s</td>
<td>28.5 ± 5.9</td>
<td>37.9 ± 28.7</td>
</tr>
<tr>
<td>TD₁, s</td>
<td>15.5 ± 2.7†</td>
<td>16.9 ± 3.6†</td>
</tr>
<tr>
<td>A₂, l/min</td>
<td>0.23 ± 0.32</td>
<td>0.41 ± 0.50</td>
</tr>
<tr>
<td>τ₂, s</td>
<td>136.7 ± 139.5</td>
<td>149.8 ± 133.5</td>
</tr>
<tr>
<td>G₁ ml·min⁻¹·W⁻¹</td>
<td>10.0 ± 1.8</td>
<td>8.8 ± 2.5</td>
</tr>
<tr>
<td>End Rec VO₂, l/min</td>
<td>0.57 ± 0.07</td>
<td>0.63 ± 0.11</td>
</tr>
<tr>
<td>MRT, s</td>
<td>40.2 ± 4.6†</td>
<td>46.6 ± 8.6†</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 9 subjects. End Rec VO₂: VO₂ at end of recovery (6 min). *Significantly different from upright, P < 0.05. †Significantly different from on-response, P < 0.05.
supine heavy exercise likely contributed to the lower 
VO₂ rise, because the oxygen pulse was similar for 
supine and upright heavy exercise.

There were no significant differences in the baseline 
and end-exercise values of VE and R for moderate 
exercise between the two conditions. The baseline 
and end-exercise values of VE were similar for supine 
and upright heavy exercise. The end-exercise values of R for 
heavy exercise in the supine condition (1.07 ± 0.03) 
were significantly greater than for the upright position 
(1.03 ± 0.05, P < 0.05).

**DISCUSSION**

In the present study, supine exercise at a moderate 
intensity resulted in a significant slowing of the overall 
VO₂ response (longer MRT) compared with the upright 
condition, but the exercise steady-state amplitude (A₁) 
and the kinetics in phase 2 (τ₂) were unaltered. During 
heavy-intensity exercise in the supine position, the 
primary time constant (τ₁) was not significantly longer, 
but instead there was a significant reduction in the 
initial VO₂ amplitude as A₁. This was compensated for 
by an increase in the magnitude of the slow component 
of VO₂ such that, by the end of exercise (6 min), there 
was no significant net effect of body position on VO₂. The 
MRT was slower in the supine compared with the 
upright position. These results represent the first quan-
titative comparison of VO₂ kinetics during heavy exercise 
between the supine and upright positions.

These results are consistent with previous observa-
tions of overall slower VO₂ kinetics during moderate 
supine exercise with both lower (15, 18, 25) and upper 
body (16) exercise. VO₂ responses during presumably 
>LT exercise have been reported only qualitatively (23) 
or semiquantitatively (half time only) (8, 9) as being 
slower in the supine position. In these studies, end-
exercise VO₂ (at 5–10 min) was not affected by posture, 
similar to the results in the present study. In neither of 
these previous studies, however, was the potential 
effect of a longer time constant distinguished from that 
of a transiently lower amplitude of the primary VO₂ 
component (A₁) as the mechanism by which the re-
sponses appeared slowed in the supine position. In a 
somewhat similar study in which the hydrostatic contribu-
tion to perfusion pressure was manipulated, Hugh-
son et al. (17) found that forearm exercise with the arm 
above the heart led to an estimated arm VO₂ response 
that was attenuated early and augmented later into 
exercise, compared with identical exercise with the arm 
below the heart. These results are similar to the 
present findings.

In the present study, the finding of a reduced A₁, but 
not a statistically significant slowing of the time con-
stant τ₁, during heavy supine exercise was contrary to 
our hypothesis. It has been proposed that slower kinet-
ics during upright heavy exercise reflect a relative 
inadequate perfusion and O₂ delivery to the working 
muscles (14, 24). The data from this study suggest that, 
during heavy exercise in the supine position, O₂ deliv-
ery to and utilization by the working muscles are 
further compromised, resulting in a consistently 
reduced amplitude of the fast component of VO₂ and a 
slowing of the overall kinetics response, compared with 
the upright position. Under these circumstances, then, 
the amplitude of the fast component was more sensitive 
to a limitation in O₂ delivery than was the associated 
time constant τ₁. This illustrates that both the time 
constant and the amplitude of the primary VO₂ re-
sponse need to be considered when the effects of an 
intervention on VO₂ kinetics during heavy exercise are 
being evaluated.

The amplitude of the VO₂ slow component was signifi-
cantly increased in supine compared with upright 
heavy exercise. Although the mechanisms underlying 
the slow component remain speculative, the primary 
origin appears to be the working muscles (1, 3, 29, 32, 
35). It has been suggested that the VO₂ slow component 
may be attributable primarily to motor unit recruit-
ment of lower efficiency, fast-twitch fibers that have a 
higher O₂ cost per tension development and a longer 
time constant (1–4, 10, 28). Consistent with this, 
Barstow et al. (3) found that the amplitude of the slow 
component during upright heavy exercise, comparable 
to that performed here, was directly related to the 
percentage of fast-twitch (type II) fibers of the vastus 
lateralis. It has been suggested that availability of O₂ 
plays an important role in regulating the recruitment 
of high-threshold motor units, because there is a close 
link between state of energy supply and types of muscle 
fibers being recruited (26). Thus one interpretation of 
the present data would suggest greater recruitment of 
type II fibers in the supine position during heavy 
exercise compared with in the upright position.

An alternative interpretation may arise from compari-
on with the previous work of Barstow et al. (3). In that 
study, the amplitude of the primary, fast component of 
VO₂ (A₁) was significantly, but inversely, related to the 
percentage of type II fibers, whereas the end-exercise 
increase in VO₂ was not different as a function of fiber

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**Table 5. HR and VO₂/HR exercise responses**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Upright</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>80.3 ± 9.8</td>
<td>79.0 ± 4.9</td>
</tr>
<tr>
<td>HR at 2 min</td>
<td>107.2 ± 6.4</td>
<td>104.7 ± 3.9</td>
</tr>
<tr>
<td>End-exercise</td>
<td>110.3 ± 7.7</td>
<td>105.6 ± 4.6*</td>
</tr>
<tr>
<td>Half time</td>
<td>24.7 ± 13.4</td>
<td>29.6 ± 11.0</td>
</tr>
<tr>
<td>VO₂/HR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL</td>
<td>6.3 ± 1.1</td>
<td>6.3 ± 1.1</td>
</tr>
<tr>
<td>VO₂/HR at 2 min</td>
<td>11.9 ± 1.4</td>
<td>11.7 ± 1.3</td>
</tr>
<tr>
<td>End-exercise</td>
<td>11.8 ± 1.5</td>
<td>11.9 ± 1.4</td>
</tr>
<tr>
<td>Heavy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>78.3 ± 9.1</td>
<td>77.9 ± 10.1</td>
</tr>
<tr>
<td>HR at 2 min</td>
<td>143.4 ± 9.0</td>
<td>138.6 ± 9.5*</td>
</tr>
<tr>
<td>End-exercise</td>
<td>155.9 ± 10.0</td>
<td>154.0 ± 11.2</td>
</tr>
<tr>
<td>Half time</td>
<td>42.5 ± 13.5</td>
<td>39.6 ± 15.4</td>
</tr>
<tr>
<td>VO₂/HR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL</td>
<td>6.5 ± 0.8</td>
<td>6.5 ± 1.0</td>
</tr>
<tr>
<td>VO₂/HR at 2 min</td>
<td>16.3 ± 2.7</td>
<td>16.2 ± 2.0</td>
</tr>
<tr>
<td>End-exercise</td>
<td>16.6 ± 2.4</td>
<td>16.8 ± 2.4</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 9 subjects. VO₂/HR, oxygen pulse.
*Significantly different from upright, P < 0.05.
type. Thus, compared with an individual with mostly type I fibers, one with mostly type II fibers had a reduced primary component \( A_1 \) and a greater slow component \( A_2 \). This pattern is similar to the supine response compared with upright in the present study. However, a significant difference between the two studies is that the results of Barstow et al. are based on intersubject comparisons across fiber type and level of fitness, whereas the present results come from intrasubject responses to a perturbation (change in body position). There may be a common mechanism that might explain the similar results in both studies. It could be argued that, in the present study, the primary mechanism producing the attenuated and slowed VO\(_2\) response in the supine position was a blunted rise in O\(_2\) delivery due to reduced perfusion pressure (and HR, see below). In the study by Barstow et al., there were similar relationships between parameters of the fast and slow VO\(_2\) components and fitness (as maximal VO\(_2\), ml·kg\(^{-1}\)·min\(^{-1}\)) that paralleled those observed with type I fiber composition. Given the known better perfusion in both the trained state and to fibers with greater oxidative capacity (for review, see Ref. 21), it may be concluded that a reduced \( A_1 \) during the adjustment to heavy exercise is predictive of (consistent with) an attenuated rise in O\(_2\) delivery early into exercise. The fact that by 6–8 min the responses were not affected by body position (present study) or fiber type (Barstow et al.) suggests that eventually the contracting muscle-circulatory complex is able to achieve a similar O\(_2\) delivery and utilization pattern. The present results, along with the previous findings regarding muscle fiber type and fitness, suggest that any interpretation of the physiological mechanisms underlying the slow component must also consider the underlying physiological processes reflected by the primary exponential rise in VO\(_2\).

In a closed circulatory system at rest, one would predict that any reduction in arterial pressure to a tissue bed in the supine compared with the upright position would be countered somewhat by improved venous return, such that perfusion pressure (arteriovenous) might be similar. However, in the lower limbs, the combination of muscle contractions during exercise (pump) providing energy for venous return and the presence of venous valves to break the venous hydrostatic column keeps leg venous pressure low irrespective of body position. Thus, in the supine posture, the reduction in arterial pressure in the legs is not matched by a similar improvement in venous return, which is already facilitated. The resultant fall in perfusion pressure leads to reduced exercise tolerance and slower VO\(_2\) kinetics (11, 15, 18, 23). Consistent with this view, lower body negative pressure, which increases the pressure gradient from the heart to the working muscles of the lower limbs, partially or fully reverses the detrimental effects of supine position on exercise responses (11, 15).

The observation of a reduction in both the HR and VO\(_2\) responses to a similar degree at 2 min during heavy exercise in the supine position suggests that the VO\(_2\) response was matched to the HR response and that this matching became evident by 2 min into exercise. This is reinforced by the observation that VO\(_2\)/HR reached a constant value by 2 min. Because VO\(_2\)/HR is equal to the product of stroke volume and the arteriovenous O\(_2\) content difference, the simplest interpretation is that both of these responses reached their exercise levels by 2 min and that any further increase in VO\(_2\) was accomplished by an increase in HR. These data thus suggest that a primary mechanism for the slowed VO\(_2\) kinetics during heavy exercise in the supine position was an attenuated HR, and presumably cardiac output, response. This conclusion is also supported by the recent work of MacDonald et al. (25), who found slower response kinetics for femoral artery blood flow after the onset of knee extension and flexion exercise in the supine compared with the upright position.

The responses to supine exercise found herein, with the presumably compromised adjustment of leg blood flow, can be contrasted with those reported for heavy exercise in hypoxic conditions (inspired O\(_2\) fraction = 0.12), in which arterial O\(_2\) content was reduced (12). Under those hypoxic conditions, peak VO\(_2\) was reduced ~25%, twice the reduction seen with supine exercise in the present study. However, in the hypoxic condition, the integrated cardiopulmonary system was able to compensate for the reduced arterial O\(_2\) content by increasing HR, and possibly leg blood flow (but, see Ref. 36). The net effect was a relatively small increase in the time constant for the primary rise in VO\(_2\) \( t_1 \), with no effect either on the amplitude of the fast component or on any aspect of the slow component. It is interesting to note that, for both hypoxia (12) and supine exercise (present study), end-exercise VO\(_2\) at 6–8 min was not different from the control, upright condition, suggesting that the integrated muscle-circulatory system was ultimately able to adjust to the metabolic demand for O\(_2\) delivery and utilization under both conditions.

Recovery kinetics for VO\(_2\) after the heavy-exercise bouts were initially described with the same model as was used for the exercise responses (Eq. 1), which contained separate time delays for the fast and slow exponential terms. However, the second time delay (TD\(_2\)) converged to a value similar to that for the fast component (TD\(_1\)), implying that both the fast and slow exponential processes decayed together during phase 2 of recovery. This finding of a common time delay in recovery for the fast and slow exponential processes has also been recently reported by Scheuermann et al. (31), using a similar approach. Furthermore, in the present study, symmetry was found between the exercise and recovery kinetics for VO\(_2\) for the heavy-exercise intensities for both supine and upright body positions, i.e., similar relative contributions (amplitudes) of the fast and slow components and similar fast time constant \( t_1 \) for exercise and recovery responses. In contrast, Paterson and Whipp (27) found asymmetry of VO\(_2\) kinetics, with a greater amplitude and a faster time constant for the fast component, and less contribution of the slow component, during recovery compared with exercise. Their results could be interpreted to suggest
that the slow component of VO₂ during heavy exercise includes metabolism from motor units with essentially fast VO₂ kinetic characteristics (e.g., type 1 motor units) but which are recruited progressively over time during the exercise. In this case, these units would be predicted to exhibit fast kinetics during recovery and thus contribute to a faster time constant and a greater amplitude for the fast exponential response and less contribution of a slow exponential term. The present findings are not consistent with this interpretation, however. Symmetry between exercise and recovery responses for similar intensities of heavy exercise has also been reported for hypoxic exercise (12) and for different pedal rates (3). The observation of symmetry between exercise and recovery kinetics for VO₂ uptake and heart rate kinetics during heavy exercise. J. Appl. Physiol. 81: 2500–2508, 1996.

In conclusion, during moderate (<LT) exercise, the VO₂ kinetics are slowed, but the steady-state increase is unchanged for supine compared with upright cycle ergometer exercise. During heavy (>LT) exercise, the supine position is associated with a reduction in the amplitude of the primary VO₂ exponential component without slowing of the fast component time constant, and a concomitant increase in the slow component, such that the 6-min value is no different from that seen during upright exercise at the same work rate and a concomitant increase in the slow component, without slowing of the fast component time constant.

In conclusion, during moderate (<LT) exercise, the VO₂ kinetics are slowed, but the steady-state increase is unchanged for supine compared with upright cycle ergometer exercise. During heavy (>LT) exercise, the supine position is associated with a reduction in the amplitude of the primary VO₂ exponential component without slowing of the fast component time constant, and a concomitant increase in the slow component, such that the 6-min value is no different from that seen during upright exercise at the same work rate. The reduced early response of VO₂ in the supine position is associated with a proportionately lower HR rise. These data suggest that, during heavy exercise in the supine position, O₂ delivery to and utilization by the working muscles are further compromised, compared with in the upright position.

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