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.

posture; gas exchange kinetics; oxygen transport; slow component of oxygen uptake

PULMONARY OXYGEN UPTAKE (VO2) has been reported to adjust more slowly at the onset of exercise in the supine position for both moderate- (8, 15, 18, 19, 22, 25, 34) and heavy-intensity exercise (8, 9, 23). Despite greater cardiac output (15, 18, 23), it appears that effective blood flow to the working leg muscles is less in the supine posture (11, 13, 25, 33), presumably as a consequence of lower arterial pressure in the legs when the effect of gravity (hydrostatic gradient effect) is removed. These results imply that the slowing of VO2 kinetics in the supine position may be the result of a blunted cardiovascular response to exercise.

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 exacerbated 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 for the predominant component of VO2 and a larger slow component compared with the upright position. In addition, we characterized the off-kineti c 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 ~50% of the LT determined for the upright position, whereas the heavy 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 using 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

\[
V_{O2}(t) = V_{O2}(b) + A_1(1 - e^{-t/\tau_1}) + A_2(1 - e^{-t/\tau_2}) + A_3(1 - e^{-t/\tau_3})
\]

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 \(\tau_1\) and \(\tau_2\) are the time delays. The phase 1 term was terminated at the start of phase 2 (i.e., at \(\tau_1\)) and assigned the value for that time (\(A_1\))

\[A_0' = A_0 + (1 - e^{-\tau_1/\tau_1})\]

The physiologically relevant amplitude of the fast primary exponential component during phase 2 (\(A_2\)) was defined as the sum of \(A_1 + A_2\). 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 \(A_2'\). 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 (\(A_2'/ (A_0' + A_2)\)) were calculated.

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

<table>
<thead>
<tr>
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<th>Upright</th>
<th>Supine</th>
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<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>
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</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.
Fig. 2. Response of \( V\dot{O}_2 \) for transition from unloaded cycling to moderate exercise (A) and heavy exercise (B) in representative subject during conditions of upright (solid lines) and supine position (dashed lines). During moderate exercise, \( V\dot{O}_2 \) kinetics were slower in supine position. During heavy exercise, rate of adjustment of \( V\dot{O}_2 \) to 6-min value was also slower in supine position but was characterized by a significant reduction in amplitude of fast component of \( V\dot{O}_2 \), without significant slowing of fast component time constant. However, amplitude of slow component was significantly increased, such that end-exercise \( V\dot{O}_2 \) was the same for 2 positions.

The overall kinetics of the response was determined from mean response time (MRT). It was calculated by fitting the response data of \( V\dot{O}_2 \) 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 \( V\dot{O}_2 \) responses, the baseline, 2-min, and end-exercise values of HR and oxygen pulse (\( V\dot{O}_2 \)-to-HR ratio \( V\dot{O}_2/HR \)) 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 \( \pm \) 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 \( V\dot{O}_2 \), estimated LT, and peak HR compared with upright posture (Table 1).

Moderate Constant Work Rate Exercise

The response for \( V\dot{O}_2 \) 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 \( V\dot{O}_2 \) (as \( A_1 \)) and the kinetics in phase 2 (as \( \tau_2 \)) were similar for the two positions, but the overall \( V\dot{O}_2 \) kinetics (as MRT) were slower in supine compared with the upright position (Table 2).

The \( O_2 \) deficit was calculated for the two positions, on the assumption that the end-exercise \( V\dot{O}_2 \) (at 6 min) represented the \( O_2 \) requirement for the exercise. The \( O_2 \) deficit was similar between the supine (0.54 \( \pm \) 0.20 liter) and the upright position (0.42 \( \pm \) 0.27 liter).

Heavy Constant Work Rate Exercise

Associated with the decrease in peak \( V\dot{O}_2 \) and the LT, supine posture resulted in an increase for the relative intensity of the heavy work rate, as denoted by \%\( \Delta \), compared with that seen in the upright position (Table 1). The response for \( V\dot{O}_2 \) during heavy exercise in a representative subject is shown for the two conditions in Fig. 2B. The primary time constant (\( \tau_1 \)) was not significantly longer, but instead the amplitude (\( A_1 \)) and the gain (\( G_1 \)) of the fast component of \( V\dot{O}_2 \) 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 (\( A'_1 \)) and the

| Table 2. \( \dot{V}O_2 \) response parameters for moderate exercise |
|---------------------------------|-------|-------|
| On-response                     |       |
| BL, l/min                       | 0.53 \( \pm \) 0.07 | 0.50 \( \pm \) 0.09 |
| \( A_0 \), l/min                | 0.27 \( \pm \) 0.11 | 0.16 \( \pm \) 0.12* |
| \( \tau_0 \), s                  | 60.2 \( \pm \) 113.3 | 43.0 \( \pm \) 68.6 |
| \( A_1 \), l/min                | 0.76 \( \pm \) 0.09 | 0.76 \( \pm \) 0.08 |
| \( \tau_1 \), s                  | 21.2 \( \pm \) 8.2  | 27.7 \( \pm \) 13.4 |
| TD, s                           | 25.6 \( \pm \) 6.4  | 22.1 \( \pm \) 8.4  |
| \( G_1 \), ml·min\(^{-1}\)·W\(^{-1}\) | 9.7 \( \pm \) 0.6 | 9.7 \( \pm \) 0.6 |
| MRT, s                          | 35.7 \( \pm \) 0.8  | 43.0 \( \pm \) 12.4* |
| Off-response                    |       |
| BL, l/min                       | 1.27 \( \pm \) 0.12 | 1.25 \( \pm \) 0.14 |
| \( A_0 \), l/min                | 0.17 \( \pm \) 0.07†| 0.09 \( \pm \) 0.05* |
| \( \tau_0 \), s                  | 4.6 \( \pm \) 10.2  | 0.6 \( \pm \) 0.6   |
| \( A_1 \), l/min                | 0.78 \( \pm \) 0.10 | 0.74 \( \pm \) 0.09†|
| \( \tau_1 \), s                  | 33.0 \( \pm \) 6.4  | 34.0 \( \pm \) 6.3  |
| TD, s                           | 18.0 \( \pm \) 5.7† | 16.4 \( \pm \) 4.3  |
| \( G_1 \), ml·min\(^{-1}\)·W\(^{-1}\) | 9.9 \( \pm \) 0.8 | 9.5 \( \pm \) 0.8† |
| MRT, s                          | 39.3 \( \pm \) 7.8  | 45.7 \( \pm \) 10.3 |

Values are means \( \pm \) SD; \( n = 9 \) subjects. BL, baseline; \( A_0 \) and \( A_1 \), amplitudes of response; \( \tau_0 \) and \( \tau_1 \), time constants; TD, time delay; \( G_1 \), gain of response \( \dot{A}/work \) rate; MRT, mean response time. *Significantly different from upright, \( P < 0.05 \). †Significantly different from on-response, \( P < 0.05 \).
Table 3. $\dot{V}O_2$ on-response parameters for heavy exercise

<table>
<thead>
<tr>
<th></th>
<th>Upright</th>
<th>Supine</th>
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<tbody>
<tr>
<td>BL, l/min</td>
<td>0.51 ± 0.05</td>
<td>0.51 ± 0.09</td>
</tr>
<tr>
<td>$\Delta\dot{V}O_2$, l/min</td>
<td>2.14 ± 0.30</td>
<td>2.16 ± 0.31</td>
</tr>
<tr>
<td>$A_0$, l/min</td>
<td>0.58 ± 0.17</td>
<td>0.39 ± 0.15*</td>
</tr>
<tr>
<td>$\tau_0$, s</td>
<td>34.6 ± 57.3</td>
<td>38.5 ± 39.1</td>
</tr>
<tr>
<td>$A_1$, l/min</td>
<td>1.83 ± 0.36</td>
<td>1.62 ± 0.31*</td>
</tr>
<tr>
<td>$\tau_1$, s</td>
<td>26.9 ± 9.2</td>
<td>24.5 ± 9.8</td>
</tr>
<tr>
<td>$T_D$, s</td>
<td>21.8 ± 5.2</td>
<td>23.1 ± 5.9</td>
</tr>
<tr>
<td>$A_2$, l/min</td>
<td>0.30 ± 0.17</td>
<td>0.55 ± 0.17*</td>
</tr>
<tr>
<td>$\tau_2$, s</td>
<td>114.3 ± 44.0</td>
<td>133.5 ± 144.8</td>
</tr>
<tr>
<td>$T_D$, s</td>
<td>126.7 ± 48.4</td>
<td>96.1 ± 28.6*</td>
</tr>
<tr>
<td>$G_1$, ml·min$^{-1}$·W$^{-1}$</td>
<td>10.4 ± 1.0</td>
<td>9.2 ± 1.0*</td>
</tr>
<tr>
<td>$A_2(A_1 + A_2)$</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. $\Delta\dot{V}O_2$, increase above baseline in $\dot{V}O_2$ at end exercise. $A_2$/($A_1 + A_2$), relative contribution of slow component to net increase in $\dot{V}O_2$ at end exercise. *Significantly different from upright, $P < 0.05$.

relative magnitude of the slow component of $\dot{V}O_2$ ($A_2$), such that the end-exercise $\dot{V}O_2$ was the same for the two positions. The overall $\dot{V}O_2$ kinetics (MRT) were slower in supine compared with the upright position.

When Eq. 1 was used to model the recovery kinetics of $\dot{V}O_2$ after heavy exercise in both the supine and upright positions, $T_D$ converged back to a value that, on average, was not significantly different from $T_D$ (upright: $T_D = 25.7 ± 16.3$, $T_D = 15.4 ± 2.9$ s, $P > 0.05$; supine: $T_D = 29.5 ± 19.4$, $T_D = 17.4 ± 3.4$ s, $P > 0.05$). Given these results, we fit the recovery $\dot{V}O_2$ response for each condition and subject with a modified version of Eq. 1, where $T_D$ was set equal to $T_D$ (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 $\dot{V}O_2$. Thus, the relative contribution of the slow component to the overall $\dot{V}O_2$ response was retained during recovery from heavy-intensity exercise, irrespective of positions. Therefore, the amplitudes for both the fast ($A_1$) and slow components ($A_2$), and the time constant for the fast component ($\tau_1$), were similar between exercise and recovery responses for each position. These results suggest a symmetry between the exercise and recovery responses of $\dot{V}O_2$ for this relative intensity (56–80% $\Delta$) of heavy exercise, especially with regard to the amplitude and time constant of the fast exponential component.

The $O_2$ 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, $\dot{V}O_2$/HR, $\dot{V}E$, 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 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 heavy exercise is shown in a representative subject under conditions of upright (solid lines) and supine position (dashed lines). During moderate exercise, end-exercise HR value was significantly lower in supine compared with upright position. During heavy exercise, 2-min value of HR was significantly lower in the supine compared with the upright position.
supine heavy exercise likely contributed to the lower \( \dot{V}O_2 \) 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 \( V_e \) and \( R \) for moderate exercise between the two conditions. The baseline and end-exercise values of \( V_e \) were similar for supine and upright heavy exercise. The end-exercise values of \( R \) for heavy exercise in the supine condition (1.07 \pm 0.03) were significantly greater than for the upright position (1.03 \pm 0.05, \( P < 0.05 \)).

**DISCUSSION**

In the present study, supine exercise at a moderate intensity resulted in a significant slowing of the overall \( \dot{V}O_2 \) response (longer MRT) compared with the upright condition, but the exercise steady-state amplitude (\( A_{t1} \)) and the kinetics in phase 2 (\( \tau_2 \)) were unaltered. During heavy-intensity exercise in the supine position, the primary time constant (\( \tau_1 \)) was not significantly longer, but instead there was a significant reduction in the initial \( \dot{V}O_2 \) amplitude as \( A_{t1} \). This was compensated for by an increase in the magnitude of the slow component of \( \dot{V}O_2 \) such that, by the end of exercise (6 min), there was no significant net effect of body position on \( \dot{V}O_2 \). The MRT was slower in the supine compared with the upright position. These results represent the first quantitative comparison of \( \dot{V}O_2 \) kinetics during heavy exercise between the supine and upright positions.

These results are consistent with previous observations of overall slower \( \dot{V}O_2 \) kinetics during moderate supine exercise with both lower (15, 18, 25) and upper body (16) exercise. \( \dot{V}O_2 \) responses during presumably \( \geq \)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 \( \dot{V}O_2 \) (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 \( \dot{V}O_2 \) component (\( A_{t1} \)) as the mechanism by which the responses appeared slowed in the supine position. In a somewhat similar study in which the hydrostatic contribution to perfusion pressure was manipulated, Hughson et al. (17) found that forearm exercise with the arm above the heart led to an estimated arm \( \dot{V}O_2 \) 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 \( A_{t1} \), but not a statistically significant slowing of the time constant \( \tau_1 \), during heavy supine exercise was contrary to our hypothesis. It has been proposed that slower kinetics during upright heavy exercise reflect a relative inadequate perfusion and \( O_2 \) delivery to the working muscles (14, 24). The data from this study suggest that, during heavy exercise in the supine position, \( O_2 \) delivery to and utilization by the working muscles are further compromised, resulting in a consistently reduced amplitude of the fast component of \( \dot{V}O_2 \) 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 \( O_2 \) delivery than was the associated time constant \( \tau_1 \). This illustrates that both the time constant and the amplitude of the primary \( \dot{V}O_2 \) response need to be considered when the effects of an intervention on \( \dot{V}O_2 \) kinetics during heavy exercise are being evaluated.

The amplitude of the \( \dot{V}O_2 \) slow component was significantly 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 \( \dot{V}O_2 \) slow component may be attributable primarily to motor unit recruitment of lower efficiency, fast-twitch fibers that have a higher \( O_2 \) 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 \( O_2 \) 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 comparison with the previous work of Barstow et al. (3). In that study, the amplitude of the primary, fast component of \( \dot{V}O_2 \) (\( A_{t1} \)) was significantly, but inversely, related to the percentage of type II fibers, whereas the end-exercise increase in \( \dot{V}O_2 \) was not different as a function of fiber
response in the supine position was a blunted rise in $\dot{V}O_2$.

$\dot{V}O_2$ kinetics during supine heavy exercise. The heavy exercise is predictive of (consistent with) an

conclusion that a reduced oxidative capacity (for review, see Ref. 21), it may be

explained by the similar results in both studies. It could be

argued that, in the present study, the primary mecha-

nism producing the attenuated and slowed $\dot{V}O_2$

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 $\dot{V}O_2$ components and fitness (as maximal $\dot{V}O_2$, $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) that paralleled those observed with type I fiber composition. Given the known better perfu-

sion 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 compo-
nent must also consider the underlying physiological processes reflected by the primary exponential rise in $\dot{V}O_2$.

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 counteracted somewhat by improved venous return, such that perfusion pressure (arteriove-

nous) 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 hydro-

static column keeps leg venous pressure low irrespec-
tive 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 $\dot{V}O_2$

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 re-

sponses (11, 15).

The observation of a reduction in both the HR and

$\dot{V}O_2$ responses to a similar degree at 2 min during heavy exercise in the supine position suggests that the $\dot{V}O_2$

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 $\dot{V}O_2$/HR reached a

constant value by 2 min. Because $\dot{V}O_2$/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 $\dot{V}O_2$ was

accomplished by an increase in HR. These data thus

suggest that a primary mechanism for the slowed $\dot{V}O_2$

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 $\dot{V}O_2$ 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 $\dot{V}O_2$ ($t_1$), 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 con-

stant ($t_1$) for exercise and recovery responses. In con-

trast, Paterson and Whipp (27) found asymmetry of $\dot{V}O_2$

kinetics, with a greater amplitude and a faster time con-

stant 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 I 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 the exercise and recovery kinetics for VO₂ suggests that, irrespective of the metabolic process(es) responsible for the slow component during exercise, in recovery these metabolic processes remain kinetically distinguishable from those associated with the fast VO₂ component. It is presently unclear what the explanation(s) might be for the differences in findings between the present study and those of Paterson and Whipp (27).

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 those involved in the upright position.

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