The response characteristics of oxygen uptake (\( \dot{V}_{O_2} \)) to step changes in power output have been well documented (3). During the transition from rest or unloaded cycling to constant-load exercise of moderate intensity [i.e., below the lactate threshold (LT)], after the cardiodynamic phase (phase I), \( \dot{V}_{O_2} \) rises in an approximately monoexponential fashion (phase II) to attain a new steady state (phase III) within 2–3 min. However, the \( \dot{V}_{O_2} \) response to constant-load exercise of heavy intensity (i.e., >LT) is complicated by the development of an additional component of \( \dot{V}_{O_2} \) that causes \( \dot{V}_{O_2} \) to rise above the predicted value (39).

The cause of this slow rise in \( \dot{V}_{O_2} \) over time during heavy exercise, i.e., the \( \dot{V}_{O_2} \) slow component, is an issue of great experimental interest. Putative mechanisms for the phenomenon include elevation of plasma catecholamines, increased rates of pulmonary ventilation, and increases in whole body and muscle temperature (6, 34). However, simultaneous measurement of pulmonary and leg \( \dot{V}_{O_2} \) demonstrated that ~86% of the excess \( \dot{V}_{O_2} \) seen with high-intensity exercise originates from within the exercising limb (33). The close correlation between the rate of blood lactate accumulation and the development of the \( \dot{V}_{O_2} \) slow component has led to the suggestion that the catabolism of lactate as an exercise substrate, or its use in gluconeogenesis, might increase exercise \( \dot{V}_{O_2} \). However, infusion of adrenaline and consequent elevation of blood lactate concentration was not found to affect \( \dot{V}_{O_2} \) in exercising humans (16, 43).

The recruitment of type II muscle fibers during heavy exercise is, perhaps, the most plausible explanation for the slow component phenomenon (2, 39). Barstow et al. (2) demonstrated that the contribution made by the slow component to the total \( \dot{V}_{O_2} \) response to 8 min of heavy, constant-load cycling was greater in subjects with a high proportion of type II fibers. This is in keeping with the observation that the contraction of type II muscle fibers is less efficient than that of type I fibers [i.e., the high-energy phosphate produced per \( O_2 \) molecule consumed (P:O) is lower in type II fibers] (12). Furthermore, glycogen depletion (38) and electromyo-

graphic (28, 36) studies have demonstrated that type II motor units are recruited at the exercise intensities at which the slow component is observed.

Relatively few studies have examined \( \dot{V}_{O_2} \) kinetics in modes of exercise other than cycling. However, two recent studies have attempted to characterize the \( \dot{V}_{O_2} \)
slow component during treadmill running (4, 23). In both studies, the magnitude of the slow component was notably smaller than has been described for cycle exercise at the same relative intensity. Only two studies have directly compared the \( \dot{V}O_2 \) slow component response in running and cycling in the same subjects. Billat et al. (5) reported that the slow component, defined as the increase in \( \dot{V}O_2 \) over 3 min and the end of exercise, was significantly greater during cycling than during treadmill running (~270 vs. 200 ml/min, respectively) when a group of elite triathletes exercised to exhaustion at 90% of their mode-specific \( V_{\text{O}2\text{ max}} \). Similarly, Jones and McConnell (22) reported that the increase in \( \dot{V}O_2 \) at 3 and 6 min of heavy exercise at 50%\( \Delta \) (i.e., the running speed or power output calculated to require 50% of the difference between the \( \dot{V}O_2 \) at LT and \( \dot{V}O_2\text{ max} \)) was significantly greater in cycling than in running (~290 vs. 200 ml/min). However, it should be noted that the slow component for running in the study of Jones and McConnell (22) was 10-fold higher than that reported by Billat et al. (5).

The existence of the \( \dot{V}O_2 \) slow component during treadmill running appears to be controversial, with three studies clearly demonstrating the phenomenon during high-intensity running (22, 23) and two studies suggesting that it is effectively nonexistent (4, 5). The demonstration of differences in the \( \dot{V}O_2 \) slow component response between exercise modes might shed light on the physiological mechanisms underpinning the phenomenon. A limitation to the studies mentioned above (4, 5, 22, 23) was the characterization of the slow component, which involved the simple calculation of the difference between \( \dot{V}O_2 \) at 3 min and \( \dot{V}O_2 \) at the end of exercise. In addition, no previous study has examined possible differences in the characteristics of the primary (fast) component between exercise modalities.

Therefore, the purpose of the present study was to compare the fast and slow component responses of \( \dot{V}O_2 \) during cycling and running exercise using the mathematical modeling procedures validated by Barstow and colleagues (2, 3). To further our understanding of any differences between the exercise modes, we studied the responses across a wide range of exercise intensities, including exercise of moderate intensity (<LT) and several exercise intensities above LT.

**METHODS**

**Subjects.** Seven recreationally active subjects [three men, four women; age 27 ± 5 (SD) yr; height 1.74 ± 0.08 m; body mass 69.3 ± 9.3 kg] volunteered to take part in this study. The subjects gave written, informed consent after the experimental procedures, the associated risks, and the benefits of participation were explained. The procedures used in this study were approved by the Chelsea School Ethics Committee, University of Brighton. The subjects were all fully familiar with laboratory exercise testing procedures.

The subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3-h postprandial, and to avoid strenuous exercise in the 48 h preceding a test session. For each subject, tests took place at the same time of day (~±2 h) to minimize the effects of diurnal biological variation on the results.

**Experimental design.** The subjects were required to visit the laboratory on 10 occasions. The first two visits were used to determine LT and \( \dot{V}O_2\text{ max} \) for both running and cycling exercise. During the remaining eight sessions, the subjects performed 2–3 repetitions of square-wave transitions from rest to one of four exercise intensities: 80% LT and 25, 50, and 75%\( \Delta \) for both treadmill and cycle exercise. On a given day, a subject would complete two or three transitions of the same exercise intensity using the same mode of exercise. The transitions were separated by 1 h of recovery. The transitions performed on a given day were determined at random, and the study was completed within 3 wk for all subjects.

**Procedures.** All running tests were performed on a motorized treadmill (Woodway, Cardiokinetics, Salford, UK) with the grade set at 1% (19). Cycle tests were conducted on an electrically braked cycle ergometer (Jaeger ER800), with seat and handlebar height kept constant over the sessions for each subject. Pedal frequency was maintained at 80 ± 5 rpm for all cycle tests. During the exercise tests, pulmonary gas exchange was determined breath by breath. We chose not to apply an alveolar algorithm to correct for possible changes in pulmonary \( \dot{V}O_2 \) stores between consecutive breaths. Subjects breathed through a low-dead space (90 ml), low-resistance (0.65 mmHg·l⁻¹·s at 8 l/s) mouthpiece and turbine assembly. Gases were continuously drawn from the mouthpiece through a 2-m capillary line of small bore (0.5 mm) at a rate of 60 ml/min, and analyzed for \( O_2, CO_2, \) and \( N_2 \) concentrations by a quadripole mass spectrometer (CaSE QP9000, Gillingham, Kent, UK) that was calibrated before each test using gases of known concentration. Expiratory volumes were determined using a turbine volume transducer (Interface Associates). The volume and concentration signals were integrated by computer after analog-to-digital conversion, taking the gas transit delay through the capillary into account. Respiratory gas exchange variables [\( \dot{V}O_2, CO_2 \) uptake (\( \dot{V}CO_2 \)), and minute ventilation] were calculated and displayed for every breath. Heart rate was recorded telemetrically throughout the exercise tests (Polar Electro Oy, Kempele, Finland).

Subjects performed incremental exercise to volitional exhaustion to determine LT and \( \dot{V}O_2\text{ max} \) during both treadmill and cycle ergometry. For the treadmill test, the initial running speed was 6.0–7.0 km/h for the women and 8.0–9.0 km/h for the men. Subjects completed 6–8 submaximal stages of 4-min duration, with running speed increased by 1.0 km/h between stages (20). At the end of each stage, subjects supported their weight with their hands and moved their feet to the sides of the treadmill belt. Finger-tip capillary blood samples (~25 \( \mu l \)) were collected in capillary tubes and subsequently analyzed for lactate concentration using an automated analyzer (YSI 2300, Yellow Springs). Subjects recommenced running within 10–15 s.

When blood lactate concentration exceeded 4 mM or heart rate exceeded 90% of the known or age-predicted maximum heart rate, the running speed was increased by 1.0 km/h every minute until the subject reached volitional exhaustion. We chose to measure LT directly rather than estimate it from gas exchange responses because we wished to equate exercise intensity as accurately as possible in our comparisons of the \( \dot{V}O_2 \) responses in the two exercise modes. In a previous study, Jones and Doust (18) demonstrated that the \( \dot{V}O_2\text{ max} \) measured after a 25-min LT determination was not significantly different from the \( \dot{V}O_2\text{ max} \) measured with a conventional 10-min incremental protocol.
A similar procedure was used in the incremental cycle test. All subjects began the test at 50 W, with increases in power output of 25 W every 4 min, and brief (10–15 s) pauses in exercise to facilitate fingertip capillary blood sampling between stages. As in the treadmill test, when blood lactate concentration exceeded 4 mM or heart rate exceeded 90% of the known or age-predicted maximum, the incremental rate was increased 25 W per minute until the subject reached volitional exhaustion.

Plots of blood lactate against running speed or power output and $\dot{V}O_2$ were provided to two independent reviewers, who determined LT was the first sudden and sustained increase in blood lactate above resting concentrations. The breath-by-breath gas exchange data collected during the incremental tests were averaged over consecutive 30-s periods. $\dot{V}O_2$ max was defined as the average $\dot{V}O_2$ attained in the last 30 s of the tests. The running speed and power output at $\dot{V}O_2$ max were estimated by extrapolation of the sub-LT relationship between $\dot{V}O_2$ and running speed/power output. The running speeds and power output at $\dot{V}O_2$ max were calculated to require 50% of $\dot{V}O_2$ at LT (moderate-intensity exercise), and 25, 50, and 75%LT (heavy-intensity exercise) were determined [e.g., 50%LT = LT + 0.5 × ($\dot{V}O_2$ max – LT)].

Subsequently, subjects performed a series of square-wave transitions of 6-min duration at the four exercise intensities for both cycling and running on separate days. The exercise protocol began with 2 min of seated rest (cycle ergometer) or standing rest with feet astride the moving treadmill belt and hands holding the guard rails (treadmill). At the start of cycling exercise, the experimenters accelerated the flywheel, while the subjects’ legs moved passively, until a pedal cadence of 80 rpm was reached, and the resistance was applied. We chose this approach because it most closely reflects the situation for the running trials, in which the subjects supported their body mass with their hands on the guard rail until leg speed matched treadmill belt speed, after which, they let go of the guard rails and began running. For both exercise modes, the transition from rest to exercise took ~5 s. Fingertip capillary blood samples were taken immediately before and after the 6-min exercise period. The difference between the end-exercise lactate and the resting lactate concentration was expressed as a delta value ($\Delta$[lactate concentration]). For the running trials, stride frequency was calculated by timing the completion of 10 strides at 2 min and again at 5 min of exercise. After a 1-h recovery period, another blood sample was taken to ensure that blood lactate had returned to resting levels. The subjects then performed an identical square-wave transition using the same mode of exercise as for the first test. For the moderate exercise trial (80% LT), the subjects performed a total of three transitions for each exercise mode, whereas for the heavy exercise trials (25, 50, and 75%LT), the subjects performed two transitions for each exercise mode.

Data analysis. For each exercise transition, the breath-by-breath data were interpolated to give second-by-second values. The transitions for each intensity were then time aligned to the start of exercise and averaged to enhance the underlying response characteristics.

Nonlinear regression techniques were used to fit $\dot{V}O_2$ data after the onset of exercise with an exponential function. An iterative process ensured the sum of squared error was minimized. The mathematical model consisted of two (moderate exercise) or three (heavy exercise) exponential terms, each representing one phase of the response (2, 3). On the basis of previous literature (2), the model was constrained to aid in identification of the key parameters. The first exponential term started with the onset of exercise (time zero), whereas the other terms began after independent time delays $\dot{V}O_2(t) = \dot{V}O_2(b) + A_0 \times (1 - e^{-t/\tau_0})$

(Phase 1: cardiodynamic component)

$+ A_1 \times (1 - e^{-t/\tau_1})$

(Phase 2: primary component)

$+ A_2 \times (1 - e^{-t/\tau_2})$

(Phase 3: slow component)

where $\dot{V}O_2(b)$ is the average value over 2 min of resting baseline; $A_0$, $A_1$, and $A_2$ are the asymptotic amplitudes for the exponential terms; $\tau_0$, $\tau_1$, and $\tau_2$ are the time constants; and $TD_1$ and $TD_2$ are the time delays. The phase 1 term was terminated at the start of phase 2 (i.e., at $TD_1$) and was assigned the value for that time ($A_0^\prime$)

$$A_0^\prime = A_0 \times (1 - e^{-TD_1/\tau_0})$$

$\dot{V}O_2$ at the end of phase 1 ($A_0^\prime$) and the amplitude of phase 2 ($A_1$) were summed to calculate the amplitude of the fast primary component ($A_1^\prime$). The slow component at the end of exercise ($A_2^\prime$) was calculated and is used in preference to $A_2$ (2).

The $\dot{V}O_2$ kinetics in recovery were analyzed in a similar way to those in exercise, with one exception. After phase 1, both the primary and slow component shared $TD_1$ (2). With the use of the procedures outlined by Motulsky and Ransnas (29), fitting the recovery data with a more complex model (separate $TD_1$ and $TD_2$) did not lead to a significantly better fit ($t_{51} = -0.46, P = 0.65$); therefore, the simpler model was adopted.

Statistical analysis. Paired t-tests were used to determine the significance of differences between running and cycling trials. The effects of exercise intensity on $\dot{V}O_2$, blood lactate concentration, and stride frequency responses were tested using one-way ANOVA with Tukey’s post hoc tests where appropriate. Pearson product moment coefficients ($r$) were used to assess the significance of relationships between the slow component and the increase in blood lactate. Statistical significance was accepted at 5%. Results are presented as means ± SD.

RESULTS

Incremental tests. Both $\dot{V}O_2$ max and LT were significantly higher for treadmill than for cycle ergometry. The $\dot{V}O_2$ max for running was 50.7 ± 13 ml·kg$^{-1}$·min$^{-1}$ vs. 43.1 ± 11 ml·kg$^{-1}$·min$^{-1}$ for cycling ($t_6 = 6.5, P = 0.001$). $\dot{V}O_2$ at LT was 36.8 ± 7 and 23.8 ± 8 ml·kg$^{-1}$·min$^{-1}$ for running and cycling, respectively ($t_6 = 12.8, P < 0.001$). Consequently, the LT occurred at a significantly higher percentage of $\dot{V}O_2$ max in running (73.4 ± 5.8%) than in cycling (54.7 ± 6.7%; $t_6 = 6.4, P = 0.001$).

Square-wave transitions. Despite the significant differences between the exercise modes in terms of $\dot{V}O_2$ max and the absolute and relative $\dot{V}O_2$ at LT attained by the subjects, there were no significant differences in exercise stress between modalities, as evidenced by the blood lactate concentration and percentage of maximum heart rate achieved (Table 1). The relative exercise intensities of the square-wave
transitions (calculated as %Δ) were not significantly different between exercise modes.

As expected, given the higher absolute VO₂ max and VO₂ at LT during running, the end-exercise VO₂ and A₁ was significantly higher in running than in cycling during both moderate and heavy exercise (Table 2). Other parameters of the VO₂ response were similar across modalities, with the exception of the VO₂ slow component (A₂). The absolute magnitude of A₂ was significantly higher for cycling than for running at both 50%Δ (334 ± 183 vs. 205 ± 84.3 ml/min, respectively; tₓ = -2.65, P = 0.038) and 75%Δ (430 ± 159 vs. 302 ± 154 ml/min, respectively; tₓ = -3.47, P = 0.001). Likewise, the proportion of the total VO₂ response attributable to the slow component (relative A₂) was significantly greater in cycling than in running at both 50 and 75%Δ (15.3 ± 3.6 vs. 7.3 ± 1.4%, tₓ = -6.01, P = 0.001; and 16.9 ± 2 vs. 9.6 ± 3.1%, tₓ = -6.46, P < 0.001). The VO₂ response of a typical subject to the four different intensities across exercise modes is shown in Fig. 1.

The magnitude of both the VO₂ slow component and the degree of blood lactate accumulation during the exercise transition increased with exercise intensity in both running (F₂,20 = 10.1, P = 0.001 and F₂,20 = 8.1, P = 0.003, respectively) and cycling (F₂,20 = 10.3, P = 0.001 and F₂,20 = 63.4, P < 0.001, respectively). The VO₂ slow component was significantly correlated with lactate concentration for both cycling (r = 0.56) and running (r = 0.81).

The parameters for the response of VO₂ during recovery are given in Table 3. As with the on-transient responses, only A₁ and A₂ varied significantly with exercise mode. For cycling, a comparison of the off-transient with the on-transient revealed parameters to be generally similar in recovery, suggesting symmetry between the exercise and recovery VO₂ kinetics. However, for running, there was a significant trend for τ₁ to be greater during recovery than during exercise at moderate intensity (11.6 ± 7.9 vs. 39.3 ± 15.9 s, tₓ = -5.9, P = 0.001), at 25%Δ (19.4 ± 8.0 vs. 34.7 ± 8 s, tₓ = -4.3, P = 0.005), at 50%Δ (20.1 ± 5.2 vs. 31.5 ± 5.9 s, tₓ = -3.6, P = 0.012) and at 75%Δ (15.9 ± 5.8 vs. 27.6 ± 8.9 s, tₓ = -2.9, P = 0.027).

Pedal frequency during cycle ergometry was successfully maintained at 80 ± 5 rpm for each exercise transition in all subjects. Stride frequency during treadmill exercise tended to increase with exercise intensity from moderate (78.6 ± 4.7 strides/min), to 25%Δ (81.6 ± 4.2 strides/min), to 50%Δ (83.7 ± 4.1 strides/min), and to 75%Δ (86.1 ± 4.8 strides/min), with only the increase from moderate to 75%Δ exercise being significant (Δ₂₃₂₇ = 3.6, P = 0.03). There were no significant changes in stride frequency between min- utes 3 and 6 of exercise.

Table 1. Blood lactate and oxygen uptake responses to the four square-wave transitions

<table>
<thead>
<tr>
<th>Desired Intensity</th>
<th>Run</th>
<th>Cycle</th>
<th>Run</th>
<th>Cycle</th>
<th>Run</th>
<th>Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>80% LT</td>
<td>76.2 ± 2.2</td>
<td>79.5 ± 1.6</td>
<td>0.0 ± 0.1</td>
<td>0.0 ± 0.1</td>
<td>61.5 ± 2</td>
<td>61.7 ± 1.4</td>
</tr>
<tr>
<td>25%Δ</td>
<td>24.1 ± 1.6</td>
<td>25.1 ± 1.8</td>
<td>1.4 ± 0.3</td>
<td>1.6 ± 0.1</td>
<td>79.5 ± 4.5</td>
<td>79.4 ± 1.9</td>
</tr>
<tr>
<td>50%Δ</td>
<td>43.8 ± 2.2</td>
<td>43.2 ± 3.5</td>
<td>3.3 ± 0.5</td>
<td>2.7 ± 0.2</td>
<td>87.6 ± 2.9</td>
<td>86.8 ± 2.4</td>
</tr>
<tr>
<td>75%Δ</td>
<td>73.2 ± 3.8</td>
<td>70.4 ± 2.3</td>
<td>4.0 ± 0.5</td>
<td>5.3 ± 0.4</td>
<td>95.2 ± 2.1</td>
<td>95.8 ± 2.1</td>
</tr>
</tbody>
</table>

Values are means ± SE. Blood lactate concentration change (Δ[Lactate]) and heart rate during running and cycling protocols at varying intensities. LT, lactate threshold; Δ, difference between LT and maximal oxygen uptake (VO₂ max). Intensity achieved was calculated using A₁ from Eq. 1; end-exercise heart rate is expressed as percent of mode-specific maximum heart rate.

Table 2. Parameters of oxygen uptake response during heavy exercise as functions of exercise modality and intensity

<table>
<thead>
<tr>
<th>Desired Intensity</th>
<th>Run</th>
<th>Cycle</th>
<th>Run</th>
<th>Cycle</th>
<th>Run</th>
<th>Cycle</th>
<th>Run</th>
<th>Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>80% LT</td>
<td>388 ± 39</td>
<td>439 ± 24</td>
<td>422 ± 28.4</td>
<td>447 ± 11</td>
<td>411 ± 32.4</td>
<td>442 ± 11.2</td>
<td>413 ± 75</td>
<td>470 ± 22</td>
</tr>
<tr>
<td>A₂, ml/min</td>
<td>720 ± 96</td>
<td>384 ± 69*</td>
<td>930 ± 68</td>
<td>619 ± 62*</td>
<td>1,064 ± 172</td>
<td>656 ± 101*</td>
<td>1,181 ± 151</td>
<td>861 ± 154*</td>
</tr>
<tr>
<td>TD, s</td>
<td>25.5 ± 4.1</td>
<td>23.0 ± 3.0</td>
<td>22.6 ± 1.9</td>
<td>22.3 ± 2.9</td>
<td>16.6 ± 1.6</td>
<td>21.2 ± 1.7</td>
<td>17.9 ± 1.4</td>
<td>21.8 ± 1.5</td>
</tr>
<tr>
<td>A₁, ml/min</td>
<td>1,570 ± 177</td>
<td>858 ± 142*</td>
<td>2,347 ± 230</td>
<td>1,522 ± 219*</td>
<td>2,559 ± 276</td>
<td>1,773 ± 231*</td>
<td>2,736 ± 326</td>
<td>2,110 ± 267*</td>
</tr>
<tr>
<td>τ₁, s</td>
<td>15.0 ± 2.0</td>
<td>18.0 ± 4.0</td>
<td>19.4 ± 3.0</td>
<td>21.6 ± 2.2</td>
<td>20.1 ± 2.0</td>
<td>22.4 ± 3.4</td>
<td>15.9 ± 2.2</td>
<td>22.6 ± 5.4</td>
</tr>
<tr>
<td>TD₂, s</td>
<td>120.1 ± 12.9</td>
<td>131.3 ± 8.8</td>
<td>111.6 ± 9.9</td>
<td>116.8 ± 16.3</td>
<td>105.2 ± 8.9</td>
<td>119 ± 14.9</td>
<td>105.2 ± 58.3</td>
<td>450 ± 605</td>
</tr>
<tr>
<td>A₂, ml/min</td>
<td>73.5 ± 20.9</td>
<td>102 ± 9.8</td>
<td>204.8 ± 31.9</td>
<td>334 ± 68*</td>
<td>301.5 ± 58.3</td>
<td>450 ± 605</td>
<td>301.5 ± 58.3</td>
<td>450 ± 605</td>
</tr>
<tr>
<td>τ₂, s</td>
<td>207.6 ± 22.5</td>
<td>232.5 ± 14.5</td>
<td>234.3 ± 23.3</td>
<td>229.5 ± 20.9</td>
<td>256.9 ± 24.2</td>
<td>254.7 ± 21.1</td>
<td>256.9 ± 24.2</td>
<td>254.7 ± 21.1</td>
</tr>
<tr>
<td>Relative A₂, % EE VO₂</td>
<td>3.2 ± 1.1</td>
<td>6.6 ± 0.5</td>
<td>7.3 ± 0.5</td>
<td>15.3 ± 1.4*</td>
<td>9.6 ± 1.2</td>
<td>16.9 ± 0.8*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EE VO₂, ml/min</td>
<td>2,420 ± 232</td>
<td>1,624 ± 228*</td>
<td>2,764 ± 306</td>
<td>2,107 ± 290*</td>
<td>3,037 ± 367</td>
<td>2,540 ± 322*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE. BL, baseline; TD₁ and TD₂ time delays from Eq. 1; τ₁ and τ₂ time constants from Eq. 1; A₁, amplitude at end of phase 1; A₂, sum of A₁ in Eq. 1 and A₂ (equal to amplitude of fast primary component); A₂, value of slow component at end of exercise; Relative A₂ [A₂/(A₁ + A₂)], relative contribution of slow component to net increase in oxygen consumption (VO₂) at end exercise; EE VO₂, increase in VO₂ above baseline at end of exercise. *Significantly lower in cycling than in running, P < 0.05; †significantly higher in cycling than in running, P < 0.05.
DISCUSSION

To our knowledge, this study is the first to describe and compare VO2 kinetics for treadmill and cycle exercise and recovery in the same subjects using a comprehensive mathematical modeling procedure.

As would be expected for subjects who are not specifically cycle trained, both VO2 max and LT (in l/min and as a percentage of VO2 max) were higher for running than for cycling. The difference in the percentage of VO2 max utilized at LT in running and cycling exercise is intriguing, but it appears to be inevitable unless elite athletes, equally well-trained in cycling and running exercise, are studied (5). The mechanisms underpinning this difference are not clear but may be similar to those responsible for the difference in the magnitude of the slow component between exercise modes (14).

To make a valid comparison of VO2 kinetics across exercise modes, we chose to normalize the exercise intensity with reference to both the LT and VO2 max determined for the two exercise modes (i.e., we used

Table 3. Parameters of oxygen uptake response during recovery from heavy exercise

<table>
<thead>
<tr>
<th></th>
<th>80% LT</th>
<th></th>
<th>25% Δ</th>
<th></th>
<th>50% Δ</th>
<th></th>
<th>75% Δ</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Run</td>
<td>Cycle</td>
<td>Run</td>
<td>Cycle</td>
<td>Run</td>
<td>Cycle</td>
<td>Run</td>
<td>Cycle</td>
</tr>
<tr>
<td>A1, ml/min</td>
<td>884 ± 114</td>
<td>387 ± 61.7</td>
<td>914 ± 181</td>
<td>507 ± 93.1</td>
<td>876 ± 97</td>
<td>450 ± 96.8</td>
<td>755 ± 85.6</td>
<td>652 ± 204</td>
</tr>
<tr>
<td>TD1, s</td>
<td>35.2 ± 5.2</td>
<td>25.9 ± 5.2</td>
<td>21.9 ± 4.2</td>
<td>22.7 ± 2.3</td>
<td>25.0 ± 2.5</td>
<td>17.3 ± 1.8</td>
<td>26.6 ± 3.2</td>
<td>20.6 ± 2.5</td>
</tr>
<tr>
<td>A2, ml/min</td>
<td>1,531 ± 213</td>
<td>876 ± 123*</td>
<td>2,285 ± 250</td>
<td>1,575 ± 191*</td>
<td>2,300 ± 262</td>
<td>1,862 ± 210*</td>
<td>2,369 ± 252</td>
<td>2,138 ± 225.5</td>
</tr>
<tr>
<td>t1, s</td>
<td>39.3 ± 3‡</td>
<td>35.9 ± 4.2‡</td>
<td>34.7 ± 3.0‡</td>
<td>25.1 ± 1.6</td>
<td>31.5 ± 2.2†</td>
<td>32.6 ± 2.7</td>
<td>27.6 ± 3.4*</td>
<td>28.8 ± 3.8</td>
</tr>
<tr>
<td>A2, ml/min</td>
<td>64 ± 32.4</td>
<td>138 ± 11.8†</td>
<td>369 ± 89</td>
<td>98 ± 37.6‡</td>
<td>509 ± 160</td>
<td>198 ± 70.9*</td>
<td>212.6 ± 19.4</td>
<td>255 ± 23.1</td>
</tr>
<tr>
<td>t2, s</td>
<td>241.6 ± 19.9</td>
<td>243.0 ± 15.4</td>
<td>215.5 ± 22.4</td>
<td>249.9 ± 26.7</td>
<td>212.6 ± 19.4</td>
<td>255 ± 23.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative A2, ml/min</td>
<td>2.5 ± 1.6</td>
<td>7.1 ± 1.1†</td>
<td>12.7 ± 3.3‡</td>
<td>5.7 ± 2.3*</td>
<td>16.7 ± 3.5</td>
<td>8.1 ± 2.5*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER VO2, ml/min</td>
<td>2,339 ± 230</td>
<td>1,676 ± 168*</td>
<td>2,646 ± 250</td>
<td>1,961 ± 190*</td>
<td>2,864 ± 346</td>
<td>2,335 ± 148</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. ER VO2 decreases in VO2 from end of exercise to end of 6-min recovery. *Significantly lower in cycling, P < 0.05; †significantly higher in cycling, P < 0.05; ‡significantly different from on-transient, P < 0.05.
the “%Δ” concept). This approach is preferable to normalizing the exercise intensity by VO₂ max alone (i.e., by testing subjects at fixed percentages of the mode-specific VO₂ max), because the latter can lead to differences in metabolic and perceptual stress, depending on the proximity of the exercise intensity to the LT (24). In the present study, despite differences in the absolute VO₂, the relative intensity of each square-wave transition was successfully matched across exercise modes because the %ΔVO₂ achieved, the increase in blood lactate above baseline, and the percent of maximum heart rate achieved were not significantly different (Table 1). Therefore, our study allows a comparison of VO₂ kinetics between running and cycling when the degree of metabolic stress, reflected as blood lactate concentration, is the same.

A comparison of the VO₂ response to moderate exercise (80% LT) revealed very similar patterns in treadmill and cycle ergometry (Table 2). As can be seen in Fig. 1A, the only differences are in the amplitudes of the VO₂ response (A₀ and A₁). Steady state was attained within ~2 min and was maintained for the entire exercise period. Blood lactate concentration did not change from resting concentrations during running or cycling (mean change: 0.0 ± 0.3 and 0.0 ± 0.4 mM, respectively). These results confirm earlier descriptions of VO₂ kinetics in cycle exercise (3, 40).

For the three heavy exercise conditions, the overall dynamics of the VO₂ response were generally similar between running and cycling (Table 2). Interestingly, there were no differences in any of the time-based parameters (i.e., TD₁, TD₂, τ₁, τ₂) between the two exercise modes. Our values for the time constant of the primary component (τ₁) are somewhat faster than have been reported previously (2, 15). This is likely to be related to the fact that our subjects were young, healthy, physical education students who were active in competitive sports and who had a high level of aerobic fitness. Recent work in our laboratory has shown that endurance training results in faster phase II VO₂ kinetics (Carter, Jones, Barstow, Burnley, Williams, and Doust, unpublished observations). It should also be noted that τ₁ was not significantly different over the range of exercise intensities studied for either cycling or running (Fig. 2). Controversy surrounds the issue of whether phase II VO₂ kinetics are slowed for exercise above compared with below the LT. Our data support the position that phase II VO₂ kinetics are not slowed for exercise above the LT (3), but this issue requires further investigation with a larger number of subjects to increase statistical power.

The increase in the amplitude of the primary VO₂ component was linearly related to the increase in exercise intensity for both running and cycling (i.e., the “gain” of the primary response calculated as A₁ divided by the exercise intensity) was not significantly different between 80% LT and 75% Δ for either running (192 ± 24.5, 199 ± 23.3, 195 ± 15.6 and 192 ± 35 ml/min per km/h for 80% LT, and 25, 50, and 75%Δ, respectively) or cycling (12.1 ± 3.6, 11.4 ± 1.3, 10.4 ± 0.6, and 10.2 ± 0.6 ml/W for 80% LT, and 25, 50, and 75%Δ, respectively). These results confirm previous reports (3) that the primary VO₂ response to square-wave exercise approximates that of a linear, first-order, time-invariant system, and imply that the VO₂ slow component is a phenomenon of delayed onset that causes VO₂ to rise above the expected VO₂ for that exercise intensity.

The differences between the exercise modes were primarily related to the amplitude of response. The A₁ was significantly higher in running exercise than in cycling exercise at every intensity (Table 2), as a result of the higher VO₂ at LT and VO₂ max in running and the consequently higher absolute VO₂ requirement at each of the exercise intensities studied. Despite the significantly higher end-exercise VO₂ and A₁ in running compared with cycling for each exercise intensity, A₂ was significantly greater for cycling than for running. This was true for all of the heavy exercise conditions, and...
which demonstrated that the increase in V\dot{O}_{2} between 3 and 6 min of heavy exercise. Because the V\dot{O}_{2} slow component begins to develop at 2 min into heavy exercise, it is possible that a significant portion of the slow component was not accounted for in this analysis. It is also known that subjects of higher aerobic fitness and/or a higher proportion of type I fibers in the vastus lateralis have a greater initial gain of the primary component and a proportionately smaller slow component (2). In addition, it is not clear from these previous studies (4, 5) if the running speed selected was sufficiently above the critical velocity to elicit a substantial slow component response.

Several physiological and mechanical differences between treadmill and cycle ergometry may account for the differences in the amplitude of the slow component that we observed. During high-intensity exercise, especially in subjects not experienced in cycle exercise, an increased handlebar grip and rocking of the torso can be observed as subjects fatigue. In this situation, the muscular work is increased with no contribution to the generation of external power output. It is possible that the increased energetic cost of isometric contraction of these auxiliary muscles contributes to the development of the slow component. In support of this suggestion, Ozyener et al. (31) recently demonstrated that the rate of Hb desaturation in the arm musculature was significantly correlated with the magnitude of the V\dot{O}_{2} slow component observed in cycle exercise at 80%\Delta.

Running and cycling comprise different types of muscle contraction. In running, ~60% of the time taken to complete one stride is spent in the support phase (i.e., foot in contact with the ground) for speeds between 12 and 23 km/h (30). Approximately 34% of this time involves eccentric muscle action (27). This eccentric muscle action may have two important consequences for the oxygen cost of running. Firstly, the metabolic cost of eccentric exercise is substantially lower than that of comparable concentric exercise (37). White (41) explained this difference by contrasting the ATP-driven detachment and resetting of the actin-myosin cross-bridges during concentric work with the forcible detachment and reattachment of the cross-bridge during eccentric exercise. The latter does not require the cleaving of ATP and therefore reduces the oxygen cost of exercise. Secondly, the “preloading” of muscle during the eccentric phase of the muscle action during running may improve the efficiency of the subsequent concentric phase. Cavanagh and Kram (8) have provided evidence that the mechanical efficiency of running exceeds that predicted from simple conversion of chemical energy to kinetic energy by muscles. The stretch-shortening cycle in running allows for storage of elastic energy during the eccentric phase and its subsequent release during the concentric phase of the action, thereby enhancing force production for a given neural input (7, 13). It is possible that the greater eccentric muscle action in running may in some way offset or delay the onset of peripheral fatigue and/or reduce the recruitment of type II motor units during running compared with cycling for the same relative exercise intensity.

It has been proposed that the oxygen consumption occurring in the legs makes up a smaller proportion of the total VO_{2} response measured at the mouth during running than cycling (22). Electromyographic studies have revealed high bursts of phasic activity in the arm and trunk muscles that suggest that the upper body musculature is responsible for significant oxygen consumption during running (17). It is likely that the metabolic cost of upper body work makes a smaller contribution to the total exercise VO_{2} during cycling than running, except perhaps when auxiliary muscles are recruited as the subject becomes fatigued. Therefore, for a given whole body VO_{2}, the leg musculature may be closer to its individual maximal oxygen consumption and its maximal voluntary contraction during cycling than running. This might require a progressive recruitment of the less efficient type II muscle fibers as the initially recruited type I fibers become fatigued.

During heavy exercise, the cycling action is associated with high intramuscular tension development for the majority of the pedal revolution, peaking from 90 to 180° (10). Ahlquist et al. (1) demonstrated that the recruitment of type II motor units is closely related to the requirements for muscle force generation. The high intramuscular pressures may lead to partial occlusion of femoral arterial blood flow (14), which may reduce oxygen delivery and lead to a greater recruitment of type II motor units.

The mechanisms presented herein implicate the recruitment of type II motor units in the development of the VO_{2} slow component. Indeed, the recent research literature has focused much attention on their importance (2, 39, 42). Type II fibers are less efficient than type I fibers, possessing an 18% lower phosphate produced to oxygen consumed ratio (12, 25). Subjects with a high proportion of type I fibers have been reported to sustain a higher power output for the same VO_{2} than subjects with a lower proportion of type I fibers during prolonged cycle exercise (11). A higher proportion of type I fibers in the vastus lateralis was also associated with a larger gain (\Delta VO_{2}/\Delta work rate) of the fast component for VO_{2} and a reduced slow component as a percentage of the total VO_{2} response during exercise at
50%Δ (2). Electromyographic and glycogen depletion studies have demonstrated that type II muscle fibers are active at exercise intensities associated with the slow component (36, 38).

Previous research in this field has typically used a cadence of 60 rpm during cycle exercise bouts. In the present study, it was decided to allow subjects to pedal at 80 ± 5 rpm. We felt it important for the interpretation of the results that subjects utilize a similar movement frequency in running and cycling. In a previous study from this laboratory, it was reported that stride frequency during high-speed treadmill running was ~85 strides/min (21). In the present study, freely chosen stride frequencies of ~80 strides/min were observed across the range of exercise intensities. As expected, stride frequency increased somewhat with running speed, but it did not change between 3 and 6 min of exercise in any of the transitions. The procedures used were therefore successful in equating muscle contraction frequencies across exercise modes, thereby eliminating this as a possible explanation for the difference in the magnitude of the VO2 slow component we observed.

Several previous studies have noted a strong relationship between the accumulation of blood lactate during heavy exercise and the magnitude of the VO2 slow component (6, 35). In contrast, in two recent studies the increase in blood lactate over 6 min of exercise has been shown to be poorly correlated with the VO2 slow component for both running and cycling (4, 22). In the present study, both modes of exercise were associated with moderate to good correlations between the two variables (r = 0.56 in cycling and r = 0.81 in running). Interestingly, there was a greater slow component rise in VO2 during cycling compared with running, despite similar increases in blood lactate above baseline in the two modes of exercise. These results support the suggestion that the relationship between the rate of blood lactate accumulation and the VO2 slow component is coincidental rather than causal (16, 43).

The procedures used in this study allowed the on- and off-transient responses to be compared across exercise modes for the first time. Analysis of recovery kinetics may provide further insight into the mechanisms controlling VO2 kinetics during exercise. In moderate exercise, there appeared to be reasonable symmetry between exercise and recovery kinetics for VO2 in the amplitude of response. However, in both running and cycling, the time constant for the primary response (τ1) was greater during recovery than during exercise, i.e., the kinetics were slower in recovery (see Table 3). The slowing of phase II kinetics was also evident when considering the recovery from heavy running exercise. It is difficult to explain this asymmetry other than through considering the subjects’ position during the recovery period. In cycling exercise, subjects completed recovery in a seated position. However, due to the nature of the exercise, recovery from running was made while standing. The present study demonstrates the presence of a slow component for recovery of VO2 similar to the magnitude found in both running and cycling exercise. This supports the suggestion that the slow component is slow to both develop and recover (15) and may indicate that similar mechanisms are responsible for both phenomena.

In conclusion, this study has shown that the oxygen uptake kinetics in running and cycling are generally similar, with the exception of the amplitude of the primary and slow components. Although a slow component does indeed develop during treadmill running, its magnitude is considerably lower than in cycling of a similar relative intensity. This difference may be related to differences in muscle contraction regimens between the exercise modes that may include: an increased isometric contraction of the upper body musculature during cycling; a higher muscle tension development during the concentric phase of the cycle action leading to rhythmic ischemia; and a greater storage and return of elastic energy during the stretch-shortening activity of running. We suggest that the latter points implicate a greater recruitment of type II muscle fibers in cycling compared with running at the same relative intensity. This may support the postulate that type II fiber recruitment is the primary mechanism responsible for the development of the slow component.

REFERENCES


