Pulmonary O₂ uptake on-kinetics in rowing and cycle ergometer exercise

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Abstract

The purpose of this study was to characterise, for the first time, the pulmonary O₂ uptake (\(\dot{V}_O_2\)) on-kinetic responses to step transitions to moderate and heavy intensity rowing ergometer exercise, and to compare the responses to those observed during upright cycle ergometer exercise. We hypothesised that the recruitment of a greater muscle mass in rowing ergometer exercise (Row) might limit muscle perfusion and result in slower Phase II \(\dot{V}_O_2\) kinetics compared to cycle exercise (Cyc). Eight healthy males (aged 28 ± 5 years) performed a series of step transitions to moderate (90% of the mode-specific gas exchange threshold, GET) and heavy (50% of the difference between the mode-specific GET and \(\dot{V}_O_2\) max) work rates, for both Row and Cyc exercise. Pulmonary \(\dot{V}_O_2\) was measured breath-by-breath and the \(\dot{V}_O_2\) on-kinetics were described using standard non-linear regression techniques. With the exception of \(\Delta \dot{V}_O_2/\Delta W_R\) which was ~12% greater for Row, the \(\dot{V}_O_2\) kinetic responses were similar between the exercise modes. There was no significant difference in the time constant describing the Phase II \(\dot{V}_O_2\) kinetics between the exercise modes for either moderate (rowing: 25.9 ± 6.8 s versus cycling: 25.7 ± 8.6 s) or heavy (rowing: 26.5 ± 3.0 s versus cycling: 27.8 ± 5.1 s) exercise. Furthermore, there was no significant difference in the amplitude of the \(\dot{V}_O_2\) slow component between the exercise modes (rowing: 0.34 ± 0.13 L min⁻¹ versus cycling: 0.35 ± 0.12 L min⁻¹). These data suggest that muscle \(\dot{V}_O_2\) increases towards the anticipated steady-state requirement at essentially the same rate following a step increase in ATP turnover in the myocytes, irrespective of the mode of exercise, at least in subjects with no particular sport specialism. The recruitment of a greater muscle mass in rowing compared to cycling apparently did not compromise muscle perfusion sufficiently to result either in slower Phase II \(\dot{V}_O_2\) kinetics or a greater \(\dot{V}_O_2\) slow component amplitude during heavy exercise.

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1. Introduction

The response kinetics of pulmonary oxygen uptake (\(\dot{V}_O_2\)) to a step change in work rate have been described for a variety of modes of exercise including cycling.
Arm cranking reveals that the time constant ($\tau$) is appreciably longer (i.e., the kinetics are slower) and the amplitude of the $\dot{V}O_2$ slow component is greater during arm cranking (Casaburi et al., 1992; Koga et al., 1996; Koppo et al., 2002), swimming (Oemarzie et al., 2001), and leg extension exercise (Koga et al., 2004; Mac Donald et al., 1998). However, perhaps surprisingly given its popularity as an exercise modality, no studies to date have characterised the $\dot{V}O_2$ kinetic response to step exercise in rowing ergometry.

It has been suggested that similarities and differences in $\dot{V}O_2$ kinetics between exercise modalities provide insight into the physiological mechanisms responsible for the control of, and the limitations to, $\dot{V}O_2$ kinetics following the onset of exercise (Jones and Burnley, 2005). For example, for the same relative exercise intensity (i.e., when the work rate is normalised for differences in the lactate threshold (LT) and $\dot{V}O_2$ max between exercise modalities), the magnitude of the so-called $\dot{V}O_2$ slow component, which becomes evident at work rates exceeding the LT, is appreciably less during treadmill running than during cycle exercise (Billat et al., 1998; Carter et al., 2000; Jones and McConnell, 1999). Differences in muscle contraction regimen (i.e., proportional contribution of concentric and eccentric muscle action to muscle force production) between these exercise modes appears to be at least partly responsible for the differences in the $\dot{V}O_2$ kinetics observed (Perry et al., 2001; Pringle et al., 2002). Also, comparisons of upright cycling with arm cranking reveal that the time constant ($\tau_p$) describing the fundamental (Phase II) increase in $\dot{V}O_2$ is appreciably longer (i.e., the kinetics are slower) and the amplitude of the $\dot{V}O_2$ slow component is greater during arm cranking (Casaburi et al., 1992; Cerretelli et al., 1977; Koga et al., 1996; Koppo et al., 2002).

Given that muscle perfusion is theoretically greater in exercise engaging a small muscle mass compared to a large muscle mass (Clausen, 1976), these results have been interpreted to suggest that the metabolic characteristics of the motor units contributing to force production play an important role in determining the $\dot{V}O_2$ kinetics (Koppo et al., 2002; Schneider et al., 2002), although differences in the relative training status of the recruited muscles will also be important. The primary purpose of the present study was to characterise $\dot{V}O_2$ kinetics during cycle ergometer exercise for the first time, and to compare the responses to $\dot{V}O_2$ kinetics during cycle ergometer exercise. This comparison also provided the opportunity to assess the influence of the volume of muscle recruited on the $\dot{V}O_2$ kinetic response to step exercise. Rowing exercise engages most of the principal muscle groups of the upper and lower body (Secher, 1993) such that a much larger fraction of the total muscle mass is recruited when compared to two-legged cycle exercise (~30 kg versus ~15 kg in a 70 kg male). The recruitment of a greater muscle mass could potentially compromise muscle perfusion, particularly during heavy exercise where a larger fraction of the maximal cardiac output is utilised (Secher et al., 1977; Volianitis and Secher, 2002). If muscle perfusion represents an important limitation to $\dot{V}O_2$ kinetics as has been suggested (Hughson et al., 2001), then one might predict that the Phase II $\tau$ would be longer when a greater muscle mass is recruited. Additionally, if $O_2$ availability influences the development of the $\dot{V}O_2$ slow component (Gerbino et al., 1996; Koga et al., 1999), then the recruitment of a greater muscle mass might be expected to increase the relative amplitude of the $\dot{V}O_2$ slow component. Therefore, we hypothesised that, compared to two-legged cycle exercise, rowing ergometer exercise would be associated with: (1) a greater $\tau_p$, reflecting slower Phase II $\dot{V}O_2$ kinetics, during heavy, but not moderate, exercise; and (2) a greater relative contribution of the $\dot{V}O_2$ slow component to the total increase in $\dot{V}O_2$ during heavy exercise.

2. Methods

2.1. Subjects

Eight healthy males (aged 28 ± 5 years, height 1.78 ± 0.04 m, body mass 77.6 ± 5.1 kg) volunteered to participate in this study which was approved by the Human Subjects Ethics Committee. The subjects were physically active and familiar with laboratory exercise testing procedures, but were not highly trained and none were specialist cyclists or rowers. Each subject gave their written informed consent after receiving a detailed explanation of the procedures, benefits and possible risks of participation. Subjects were asked to arrive at the laboratory having avoided the consumption of foodstuffs, alcohol and caffeine in the 4 h prior to exercise. They were also requested to refrain from...
undertaking strenuous physical activity in the 24 h prior to attending the laboratory.

2.2. Procedures

The subjects attended the laboratory on seven occasions, with each laboratory visit separated by a minimum of 48 h. On the first occasion, they were familiarised with the laboratory, the cycling (Jaeger Ergoline E800, Mindjhaart, The Netherlands) and rowing (Concept II, Cranleigh, UK) ergometers, and the procedures for gas analysis and blood sampling. On the next two visits to the laboratory, the subjects completed incremental exercise tests to exhaustion on the cycling and rowing ergometers for the assessment of the mode-specific GET and $\dot{V}O_2$ max (see below). The order in which these tests were presented to subjects was counter-balanced. On the four remaining visits to the laboratory (twice for cycling and twice for rowing), the subjects completed a series of ‘square-wave’ exercise tests to work rates that were classified as either moderate (\(<\)GET) or heavy (\(\geq\)GET but \(<\dot{V}O_2 \max\)), (see below). The order in which these tests were presented to subjects was counter-balanced. On the four remaining visits to the laboratory (twice for cycling and twice for rowing), the subjects completed a series of ‘square-wave’ exercise tests to work rates that were classified as either moderate (\(<\)GET) or heavy (\(\geq\)GET but \(<\dot{V}O_2 \max\)), (see below). Specifically, on each visit to the laboratory, the subjects performed two step transitions from ‘unloaded’ exercise to moderate intensity exercise followed by one step transition from ‘unloaded’ exercise to moderate intensity exercise followed by one step transition from ‘unloaded’ exercise to heavy intensity exercise. The duration of each of the exercise bouts was 6 min and the bouts were separated by 6 min (comprising 3 min of rest and 3 min of ‘unloaded’ exercise). The order in which the cycling and rowing test days were presented to subjects was counter-balanced to eliminate any order effects. In total, the subjects completed four bouts of moderate exercise and two bouts of heavy exercise in each of the two exercise modes. Pulmonary gas exchange was measured breath-by-breath throughout all exercise tests and data were averaged over consecutive 10 s periods. The $\dot{V}O_2$ at the GET was estimated using standard procedures, namely: (i) a disproportionate increase in $VCO_2$ from visual inspection of individual plots of $\dot{V}CO_2$ versus $\dot{V}O_2$; and (ii) an increase in $\dot{V}E/\dot{V}O_2$ with no increase in $\dot{V}E/\dot{V}CO_2$. The $\dot{V}O_2 \max$ was taken to be the highest 10-s $\dot{V}O_2$ value attained within the last 30 s of the test. The work rates corresponding to 90% GET and 50% of the difference between the GET and $\dot{V}O_2 \max$ (i.e. 50% $\Delta$) were subsequently calculated for each of the exercise modes with account taken of the ‘lag’ in $\dot{V}O_2$ relative to the instantaneous work rate that exists during incremental exercise (Whipp et al., 1982).

2.4. Step tests

The step tests commenced with 3 min of baseline exercise. For cycle period, the baseline exercise involved the subjects turning their legs at 70 rev min$^{-1}$ against the lowest work rate available on the ergometer (20 W). After 3 min, the work rate was abruptly increased to the target work rate and maintained for 6 min. For rowing exercise, the baseline period involved the subjects sliding backward and forward on the ergometer monorail at a rate of 25–30 rev min$^{-1}$ without holding onto the hand grips. For the last 20 s of the 3 min baseline exercise period, the subjects passively held the hand grips while two experimenters, who stood either side of the ergometer, pulled on the hand grips to accelerate the flywheel to the target work rate. When 3 min had elapsed, the experimenters let go of the hand grips, and the subject was responsible for maintaining the imposed work rate for 6 min.

Pulmonary gas exchange and minute ventilation were continuously measured breath-by-breath during all exercise tests. Subjects wore a nose-clip and breathed through a low dead space (35 mL), low resistance mouthpiece and volume sensor assembly. Gases were continuously drawn from the mouthpiece through a capillary line and analysed for $O_2$ and $CO_2$ concentrations by a fast-response metabolic analyser ($O_2$: differential paramagnetic; $CO_2$: infra-red absorption; Oxycon Alpha, Jaeger, The Netherlands). The system was calibrated prior to each test with gases of known concentration. Expiratory volumes were determined using a Triple V turbine volume sensor (Jaeger,
V and A

2.5. Analysis of blood samples from subjects during rowing exercise.

blood sample was collected from a fingertip into a capillary tube immediately before the onset of unloaded exercise and as soon as possible following the completion of each of the exercise bouts, and was subsequently analysed for blood [lactate] (YSI 1500, Yellow Springs, USA). Blood was sampled immediately before the step transition to the target work rate in both exercise modes because of difficulties in obtaining blood samples from subjects during rowing exercise.

2.5. Analysis of VO2 kinetics

The breath-by-breath VO2 data from each step test were linearly interpolated to give 1-s values. For each subject and each exercise modality, the repeat transitions to moderate and heavy exercise were time aligned to the start of exercise, superimposed and ensemble averaged to reduce the breath-to-breath noise and enhance the underlying physiological response characteristics. The baseline VO2 was defined as the average VO2 measured during unloaded cycling between 150 and 30 s before the start of exercise. A single exponential model was used to analyse the VO2 responses to moderate exercise whereas a double exponential model was used for the heavy exercise transitions. The first 20 s of data following the onset of exercise (containing the initial ‘cardiodynamic’ phase) were not included in the model fits. Subsequently, each averaged response was described using one of the following equations:

\[ VO_2(t) = VO_{2\, baseline} + A_p (1 - e^{-t/Td_p/s}) \] (90\% GET)

\[ VO_2(t) = VO_{2\, baseline} + A_p (1 - e^{-t/Td_p/s}) + A_s (1 - e^{-t/Td_s/s}) \] (50\% Δ)

The exponential models include amplitudes (\(A_p\) and \(A_s\)), time constants (\(T_d\) and \(T_d\)) and time delays (\(T_d\) and \(T_d\)) that were determined using a non-linear least-square algorithm in which minimizing the sum of squared error was the criterion for convergence. \(A_p\), \(T_d\) and \(T_d\) describe the parameters related to the VO2 primary component, while \(A_s\), \(T_d\) and \(T_d\) describe the parameters related to the VO2 slow component. Because the asymptotic value \(A_s\) can represent a higher value than actually reached at the end of the exercise, the value of the VO2 slow exponential term at the end of exercise was defined as \(A_s'\). The functional gain of the primary component (\(G_p\)) and end-exercise (\(G_e\)) VO2 responses were calculated as the appropriate amplitude of VO2 above that at baseline (i.e. \(A_p\) and \(A_p + A_s'\), respectively) divided by the increase in work rate above that at baseline and expressed in units of mL.min\(^{-1}\)W\(^{-1}\).

2.6. Statistics

Data are presented as mean ± S.D. The significance of differences in the parameters of the VO2 on-kinetics between rowing and cycle ergometer exercise, and between moderate and heavy exercise within the same exercise modality, was evaluated using repeated measures analysis of variance with post hoc Bonferroni-adjusted paired t-tests (SPSS for Windows, version 11.5). A P-value <0.05 was accepted as representing a significant difference.

3. Results

3.1. Incremental exercise

The physiological responses of the subjects to incremental rowing and cycling are summarised in Table 1. There was no significant difference in VO2 max or the VO2 at GET between the two modes of exercise. However, the peak work rate attained and the work rate at the GET were significantly higher for cycling compared to rowing.

3.2. Step exercise

The work rates corresponding to 90\% GET were 96 ± 12 and 126 ± 24 W for rowing and cycling, respectively, and the work rates corresponding to 90\% Δ
Table 1
Mean ± S.D. physiological responses to incremental rowing and cycle ergometer exercise

<table>
<thead>
<tr>
<th></th>
<th>Rowing</th>
<th>Cycling</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2 max (L min⁻¹)</td>
<td>3.48 ± 0.34</td>
<td>3.38 ± 0.42</td>
</tr>
<tr>
<td>VO2 max (ml kg⁻¹ min⁻¹)</td>
<td>44.1 ± 6.3</td>
<td>43.9 ± 6.9</td>
</tr>
<tr>
<td>HR max (b min⁻¹)</td>
<td>177 ± 11</td>
<td>181 ± 14</td>
</tr>
<tr>
<td>Peak work rate (W)</td>
<td>179 ± 25</td>
<td>211 ± 35</td>
</tr>
<tr>
<td>VO2 at GET (L min⁻¹)</td>
<td>1.73 ± 0.17</td>
<td>1.62 ± 0.21</td>
</tr>
<tr>
<td>Work rate at GET (W)</td>
<td>76 ± 12</td>
<td>106 ± 24</td>
</tr>
</tbody>
</table>

HR, Heart rate; GET, gas exchange threshold.

There was also no significant difference in the %HR max 70 ± 12 between the exercise modes (rowing: 126 ± 68 versus cycling: 120 ± 72).

Table 2
Mean ± S.D. blood lactate and heart rate responses to step transitions to moderate and heavy exercise in rowing and cycle ergometer exercise

<table>
<thead>
<tr>
<th></th>
<th>Rowing moderate</th>
<th>Cycling moderate</th>
<th>Rowing heavy</th>
<th>Cycling heavy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ Blood lactate (Mm)</td>
<td>0.9 ± 0.6</td>
<td>0.6 ± 0.4</td>
<td>5.5 ± 2.2</td>
<td>4.5 ± 1.8</td>
</tr>
<tr>
<td>Exercise HR (b min⁻¹)</td>
<td>124 ± 10</td>
<td>118 ± 12</td>
<td>166 ± 12</td>
<td>158 ± 10</td>
</tr>
<tr>
<td>Δ HR max</td>
<td>70 ± 6</td>
<td>65 ± 6</td>
<td>93 ± 7</td>
<td>87 ± 5</td>
</tr>
</tbody>
</table>

HR, heart rate.

The blood lactate and heart rate responses to the moderate and heavy intensity step exercise tests are summarised in Table 2. There were no significant differences in either Δ blood lactate or the percentage of HR max utilised between rowing and cycling for either moderate or heavy exercise, demonstrating that exercise intensities were appropriately normalised between the exercise modes.

The VO2 response kinetics to moderate intensity cycling and rowing are summarised in Table 3 and illustrated in Fig. 1. There was no significant difference between the modes of exercise for baseline VO2, the amplitudes of the primary or slow components of VO2, or the VO2 attained at the end of exercise. Furthermore, τγ was not significantly different between the exercise modes (rowing: 25.9 ± 8.8 s versus cycling: 27.8 ± 5.1 s). The mean 95% confidence intervals surrounding the estimate of τγ were 2.0 and 1.5 s for rowing and cycling, respectively. As for moderate exercise, there was a significant difference in Gp, rowing: 10.5 ± 0.8 mL min⁻¹ W⁻¹ versus cycling: 9.7 ± 1.0 mL min⁻¹ W⁻¹, P < 0.05). This higher O2 cost of exercise per unit increase in work rate in rowing was also evident at the end of exercise (Gp, rowing: 11.3 ± 1.6 mL min⁻¹ W⁻¹, P < 0.05).

When the VO2 kinetic responses to moderate and heavy exercise were compared for cycling, there was found to be no significant difference in any of the comparable parameters of interest including τγ (moderate: 25.7 ± 8.6 s versus heavy: 27.8 ± 5.1 s). There was, however, a tendency for Gp, to be lower at the higher work rate (moderate: 10.5 ± 1.0 mL min⁻¹ W⁻¹ versus heavy: 9.7 ± 1.0 mL min⁻¹ W⁻¹, P < 0.10).

Table 3
Mean ± S.D. VO2 kinetic responses to a step transition to a ‘moderate’ work rate for rowing and cycle ergometer exercise

<table>
<thead>
<tr>
<th></th>
<th>Rowing</th>
<th>Cycling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ Work rate (W)</td>
<td>96 ± 12</td>
<td>106 ± 24</td>
</tr>
<tr>
<td>Baseline VO2 (L min⁻¹)</td>
<td>0.71 ± 0.08</td>
<td>0.67 ± 0.09</td>
</tr>
<tr>
<td>Phase II time constant (s)</td>
<td>25.9 ± 6.8</td>
<td>25.7 ± 6.6</td>
</tr>
<tr>
<td>Primary amplitude (L min⁻¹)</td>
<td>1.13 ± 0.13</td>
<td>1.10 ± 0.23</td>
</tr>
<tr>
<td>Primary gain (mL min⁻¹ W⁻¹)</td>
<td>11.4 ± 1.1</td>
<td>10.5 ± 1.0</td>
</tr>
<tr>
<td>End-exercise VO2 (L min⁻¹)</td>
<td>1.82 ± 0.13</td>
<td>2.17 ± 0.36</td>
</tr>
</tbody>
</table>

* Denotes the existence of a significant difference (P < 0.05).
Fig. 1. Oxygen uptake responses following the onset of moderate (upper panel) and heavy (lower panel) rowing and cycle ergometer exercise in a representative subject. Moderate rowing, open circles; moderate cycling, closed grey circles; heavy rowing, open circles; heavy cycling, closed grey circles. The solid lines represent the model fits. Note the greater O2 cost of exercise for rowing compared to cycling, the attainment of a steady-state after ∼2 min during moderate exercise for both modes of exercise, and the emergence of the $\dot{V}O_2$ slow component after 2–3 min of heavy exercise for both modes of exercise.

Table 4

<table>
<thead>
<tr>
<th></th>
<th>Rowing</th>
<th>Cycling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ Work rate (W)</td>
<td>199 ± 25</td>
<td>214 ± 35</td>
</tr>
<tr>
<td>Baseline VO2 (L min$^{-1}$)</td>
<td>0.71 ± 0.09</td>
<td>0.66 ± 0.06</td>
</tr>
<tr>
<td>Phase II constant (s)</td>
<td>26.5 ± 3.0</td>
<td>27.4 ± 3.1</td>
</tr>
<tr>
<td>Primary amplitude (L min$^{-1}$)</td>
<td>2.14 ± 0.28</td>
<td>2.03 ± 0.33</td>
</tr>
<tr>
<td>Primary gain (L min$^{-1}$ W$^{-1}$)</td>
<td>10.8 ± 0.8</td>
<td>9.7 ± 1.0</td>
</tr>
<tr>
<td>SC time delay (s)</td>
<td>135 ± 35</td>
<td>129 ± 26</td>
</tr>
<tr>
<td>SC amplitude (L min$^{-1}$)</td>
<td>0.34 ± 0.13</td>
<td>0.35 ± 0.12</td>
</tr>
<tr>
<td>SC amplitude (%)</td>
<td>14 ± 1</td>
<td>13 ± 1</td>
</tr>
<tr>
<td>(% of end-exercise VO2)</td>
<td>3.15 ± 0.32</td>
<td>2.94 ± 0.37</td>
</tr>
<tr>
<td>End-exercise VO2 (L min$^{-1}$)</td>
<td>12.5 ± 1.0</td>
<td>11.3 ± 1.6</td>
</tr>
</tbody>
</table>

SC, VO2 slow component.
* Denotes the existence of a significant difference ($P < 0.05$).

4. Discussion

To our knowledge, this is the first study to characterise VO2 kinetics during rowing ergometer exercise. Our principal finding was that the VO2 kinetic responses to moderate and heavy intensity rowing ergometer exercise cohered well with the responses to cycle ergometer exercise in our subjects, who were familiar with but not specifically trained for either mode of exercise. Specifically, following the onset of moderate intensity rowing, VO2 rose exponentially in Phase II with a similar time constant to that determined for moderate exercise but this fundamental response was supplemented by a secondary (slow) component of VO2 that emerged after 2–3 min of exercise. One notable difference between the exercise modes was the significantly greater O2 cost of exercise (i.e. greater $\Delta V/\Delta WR$) during rowing compared to cycle exercise.

The similarity of the $\tau_p$ values for both moderate and heavy work rates between the exercise modes did not support our hypothesis. Rowing exercise engages almost all the principal muscle groups of the upper and lower body (Secher, 1993) and the recruitment

However, the development of the VO2 slow component as heavy exercise continued meant that the end-exercise gain ($G_T$) tended to be greater for heavy exercise compared to moderate exercise (moderate: $10.5 ± 1.0$ mL min$^{-1}$ W$^{-1}$ versus heavy: $11.3 ± 1.6$ mL min$^{-1}$ W$^{-1}$; $P < 0.10$). For rowing, there was also no significant difference in $\tau_p$ between the two exercise intensities (moderate: $25.9 ± 6.8$ versus heavy: $26.5 ± 3.0$). However, in this mode of exercise, $G_T$ was significantly lower at the higher work rate (moderate: $11.8 ± 1.1$ mL min$^{-1}$ W$^{-1}$ versus heavy: $10.8 ± 0.8$ mL min$^{-1}$ W$^{-1}$; $P < 0.05$). Again, however, the development of the VO2 slow component at the higher work rate meant that $G_T$ tended to be greater for heavy exercise compared to moderate exercise (moderate: $11.8 ± 1.1$ mL min$^{-1}$ W$^{-1}$ versus heavy: $12.5 ± 1.0$ mL min$^{-1}$ W$^{-1}$; $P < 0.10$). The influence of work rate on the primary component, slow component, and end-exercise gain terms for cycling and rowing exercise is shown in a schematic in Fig. 2.
of a large muscle mass could theoretically challenge the appropriate distribution of cardiac output to the exercising muscles and reduce muscle O$_2$ availability, especially during heavy exercise (Secher et al., 1977; Volianitis and Secher, 2002; but see also Richardson et al., 1995). However, if muscle O$_2$ availability was reduced during rowing compared to cycling because of the greater muscle mass recruited in the former, then this did not significantly impact upon $\tau_p$. Consistent with this, Koga et al. (2001) reported that there was no significant difference in $\tau_p$ between one-legged and two-legged cycle ergometry for either moderate or heavy exercise. Our results confirm and extend the results of Koga et al. (2001) by demonstrating that $\tau_p$ is not significantly altered by the recruitment of a greater muscle mass, even when this is greater than that recruited during two-legged cycle ergometry. However, while these data could be interpreted to suggest that O$_2$ availability does not limit VO$_2$ kinetics even during heavy exercise involving a large muscle mass, it should be acknowledged that we have no direct evidence that muscle O$_2$ availability was compromised during rowing ergometry. It is possible, for example, that because the work rates we selected were sub-maximal, cardiac output could be increased during rowing exercise to ensure that muscle perfusion was adequate. It remains to be determined whether VO$_2$ kinetics are slower during rowing compared to cycling at higher (peri-maximal) work rates where the potential for a compensatory increase in cardiac output would be reduced.

The similarity of the group mean $\tau_p$ for both moderate exercise (cycling: 25.7 s versus rowing: 25.9 s) and heavy exercise (cycling: 27.8 s versus rowing: 26.5 s) is striking, particularly when one considers that the two exercise modes are quite different with regard to muscle mass recruited, use of the upper body musculature, body position, and duty cycle. It should be emphasised that our subjects were familiar with...
both exercise modalities, but were not specifically trained in either. The value of $\tau_p$ for cycle exercise (i.e. $\sim 25–28$ s) that we observed in the present study is similar to that reported previously in subjects of similar training status (Koga et al., 1999; Scheuermann and Barstow, 2003). However, it is known that endurance training in a specific mode of exercise results in faster $\dot{V}O_2$ kinetics in that same exercise mode (Cerretelli et al., 1977, 1979; Phillips et al., 1995; see Jones and Koppo, 2005 for review), presumably due to the increased mitochondrial density and capacity for muscle perfusion that accompanies such training (Jones and Carter, 2000). For this reason, it is important that the training status of the subjects studied is considered in ‘comparison’ studies of this type.

There is evidence that the rate at which muscle mitochondrial $\dot{V}O_2$ consumption increases following the onset of exercise is principally under ‘feedback’ control, that is, it is functionally linked to the splitting of high-energy phosphates in the cytosol (Chance and Williams, 1955; Mahler, 1985; Rosièr et al., 1999).

The similarity of $\tau_p$ for cycling and rowing exercise in the present study therefore suggests that muscle $\dot{V}O_2$ increases towards the anticipated steady-state requirement at essentially the same rate following a step increase in ATP turnover in the myocytes, irrespective of the mode of exercise and the volume of muscle recruited, at least in subjects with no particular sport specialism. Consistent with this interpretation, Carter et al. (2000) reported that there was no significant difference in $\tau_p$ when recreationally-active subjects completed step transitions to the same relative moderate and heavy exercise intensities during cycle exercise and treadmill running. Also, Chilibeck et al. (1996) reported that there was no significant difference in $\tau_p$ between treadmill walking and cycle exercise, or between plantar flexion exercise and cycle exercise, in two separate groups of young subjects.

The $\Delta \dot{V}O_2/\Delta WR$ (the reciprocal of ‘delta’ efficiency) was $11–12\%$ greater in rowing compared to cycling both at moderate and heavy work rates, indicating that our subjects were less economical during rowing compared to cycling. Reduced rowing economy (relative to cycle exercise) has been reported previously in non-elite subjects (Cunningham et al., 1975; Steenacker et al., 1986), but the reason for this difference is not clear. One possibility is that our subjects had not fully mastered the appropriate technique required to optimise economy during rowing ergometer exercise. Although our subjects were not specifically cycle trained and were familiar with rowing ergometer exercise, it is still likely that they had greater experience of cycling. An inappropriate rowing technique could certainly increase the $O_2$ cost of exercising at a particular work rate. It is also possible that the much greater use of the upper body musculature in rowing increased the $O_2$ cost of exercise. For example, it is known that $\Delta \dot{V}O_2/\Delta WR$ is appreciably higher in arm cranking compared to cycling ($\sim 12$ mL min$^{-1}$ W$^{-1}$ versus 10 mL min$^{-1}$ W$^{-1}$; e.g. Koppo et al., 2002).

One explanation for this is that the muscles of the upper body generally contain a larger fraction of ‘less efficient’ type II muscle fibres (Gollnick et al., 1972; Johnson et al., 1973) and are generally less well conditioned compared to the muscles of the lower body. Finally, it is possible that rowing ergometry is inherently less efficient than cycle ergometry, due to differences in duty cycle, contraction frequency, or to differences in the synchronisation of muscle contraction (synchronous in rowing, asynchronous in cycling), and the requirement for overcoming inertia at the beginning of each stroke in rowing. Indeed, the isometric contraction of the arms and back muscles during the ‘catch’ phase is likely to elevate the $O_2$ cost of exercise above that observed for the same work rate in cycling.

There was no significant difference in the amplitude of the $\dot{V}O_2$ slow component during heavy cycling and rowing, either when expressed in absolute ($\sim 340–350$ mL min$^{-1}$) or in relative ($\sim 13–14\%$ contribution to end-exercise $\dot{V}O_2$) terms. Again, this similarity is striking when one considers the substantial differences in the volume and pattern of muscle recruitment in the two exercise modes. Indeed, since the absolute amplitude of the $\dot{V}O_2$ slow component was similar between the exercise modes despite the utilisation of a greater muscle mass during rowing, it is likely that the ‘relative’ $\dot{V}O_2$ slow component expressed in terms of mL.kg muscle mass$^{-1}$ min$^{-1}$ was actually lower during rowing. The mechanistic basis for the $\dot{V}O_2$ slow component remains obscure (Poole and Jones, 2005). However, it appears that the $\dot{V}O_2$ slow component is influenced by muscle perfusion pressure and $O_2$ availability: experimental interventions designed to enhance muscle perfusion and $O_2$ availability generally result in a reduced slow component amplitude (Burnley et al., 2000; Gerbino et al., 1996; MacDonald et al., 1997;
Haseler et al., 2004) whereas interventions designed to reduce muscle perfusion and O$_2$ availability can result in an increased slow component amplitude (Koga et al., 1999; Knight et al., 2004). On the other hand, the reduced VO$_2$ slow component observed following “priming” exercise might equally be explained by alterations in motor unit recruitment patterns (Burnley et al., 2002), and the inspiration of hypoxic gas was reported to have no significant effect on the amplitude of the VO$_2$ slow component (Engelen et al., 1996). Our data could therefore be interpreted to indicate either that muscle O$_2$ availability was well preserved during heavy rowing exercise despite the greater demand for muscle perfusion (see earlier discussion), or that any reduction in O$_2$ availability did not measurably impact the amplitude of the VO$_2$ slow component. Several studies suggest a relationship between the VO$_2$ slow component and the recruitment of type II muscle fibres (Barstow et al., 1996; Pringle et al., 2003a,b; Krstrup et al., 2004). The greater involvement of the upper body musculature in force production during rowing might be expected to increase the proportional recruitment of type II fibres during such exercise (see above). However, if this did occur, it clearly did not significantly influence the amplitude of the VO$_2$ slow component. Alternatively, it is possible that there were no appreciable differences in the respective contributions of type I and type II fibres to force production during heavy rowing and cycle exercise. In this respect, it is pertinent to point out that the use of the “%Δ” method to normalise exercise intensity resulted in there being no significant difference in Δ blood [lactate] (e.g. Roston et al., 1987), although this relationship is not believed to be causal (Gaesser et al., 1994). Therefore, since the metabolic stress invoked during heavy rowing and cycle exercise (as reflected by Δ blood [lactate]) was equivalent, it is perhaps not surprising that there was no significant difference in Δ blood [lactate] between rowing and cycle exercise. The amplitude of the slow component has been shown previously to be positively correlated with Δ blood [lactate] (e.g. Roston et al., 1987), although this relationship is not believed to be causal (Gaesser et al., 1994). Therefore, since the metabolic stress invoked during heavy rowing and cycle exercise (as reflected by Δ blood [lactate]) was equivalent, it is perhaps not surprising that there was no significant difference in the amplitude of the VO$_2$ slow component. In this context, it should be considered that although the recruitment of a greater muscle mass in rowing compared to cycling for equivalent work rates might have important cardiovascular consequences, a “sharing out” of the requisite force generation across more active muscle fibres could be beneficial in reducing the metabolic perturbation in the fibres. For example, Astrand and Saltin (1961) demonstrated that time to exhaustion was significantly extended when the same absolute work rate was shared between the arms and the legs compared to the legs alone, despite there being a similar absolute VO$_2$. This greater “sharing out” of the power output during rowing compared to cycling in the present study should therefore be considered as an important factor in the similar VO$_2$ slow component amplitude observed in the two exercise modes.

Another interesting feature of our data was the lower primary component gain ($G_p$) during heavy exercise compared to moderate exercise both for rowing (where $G_p$ fell significantly from 11.8 to 10.8 mL min$^{-1}$ W$^{-1}$; $P < 0.05$) and cycling (where $G_p$ fell from 10.5 to 9.7 mL min$^{-1}$ W$^{-1}$; $P < 0.10$). However, despite the reduced $G_p$ during heavy exercise, the development of the VO$_2$ slow component meant that the end-exercise gain ($G_T$) was greater for heavy compared to moderate exercise for both modes of exercise. These data differ slightly from general descriptions of the characteristics of VO$_2$ kinetics during moderate and heavy cycle exercise which suggest that the $G_p$ is essentially constant across a wide range of work rates (at ~10 mL min$^{-1}$ W$^{-1}$) and that the VO$_2$ slow component, which is superimposed on this response, subsequently elevates the $G_T$ above 10 mL min$^{-1}$ W$^{-1}$ (Barstow and Mølø, 1991; Paterson and Whipp, 1991; Whipp and Ward, 1990). Our data are subtly different in that they suggest that the VO$_2$ slow component initially ‘compensates’ for the reduced $G_p$ at higher work rates (i.e. the slow component brings VO$_2$ closer to the value that would be ‘expected’ for the work rate as calculated from an extrapolation of the VO$_2$-work rate relationship for sub-LT exercise) before increasing to a value that exceeds 10 mL min$^{-1}$ W$^{-1}$ at the end of exercise.

The significant reduction in the $G_p$ during heavy compared to moderate rowing ergometer exercise reported in the present study is consistent with previous observations during both treadmill running (Carter et al., 2002) and cycle ergometry (Jones et al., 2002; Pringle et al., 2003a; Scheuermann and Barstow, 2003; Wilkerson et al., 2004). An explanation for this phenomenon is presently elusive, but it is unlikely that this reflects an improved exercise economy at higher work rates. Rather, it has been suggested that the reduced $G_p$ might reflect an obligatory contribution to energy demand from anaerobic ATP production and/or
a constraint on the rate of $\dot{V}_O_2$ consumption (Jones et al., 2002; Pringle et al., 2003a; Scheuermann and Barstow, 2003; Wilkerson et al., 2004), particularly in the type II muscle fibres that will be recruited in greater proportion (Kreutz et al., 2004; Vollestad and Blom, 1985), at these higher work rates. Specifically, an obligatory increase in anaerobic glycolysis would ‘spare’ the aerobic demand of heavy exercise, although whether this might be related to the propensity of higher-order fibres to meet a proportion of the energy requirement through anaerobic pathways or to slower blood flow dynamics and a reduced $\dot{V}_O_2$ gradient in these same fibres (Behnke et al., 2003) is presently unclear.

Since this is, to our knowledge, the first study to investigate $\dot{V}_O_2$ kinetics during rowing ergometer exercise, it is appropriate to comment on the fidelity of the data collected and their suitability for mathematical modelling. There is evidence of locomotor-respiratory coupling or ‘entrainment’ of the breathing rate with the stroke rate during rowing (Mahler et al., 1991; Steinacker et al., 1993), and it is possible that this could impact on the extent of the variability in the $\dot{V}_O_2$ data.

We did not investigate the incidence of entrainment of the respiratory and stroke rates in the present study. However, we did notice that the $\dot{V}_O_2$ data were somewhat “noisier” during rowing compared to cycling, although the mean 95% confidence intervals surrounding the estimation of the $\tau_1$ were small in all cases (2.5 s for moderate intensity rowing, 1.4 s for moderate intensity cycling, 2.0 s for heavy intensity rowing, and 1.5 s for heavy intensity cycling). It appears, therefore, that $\dot{V}_O_2$ data collected during rowing ergometer exercise is amenable to mathematical modelling for the purpose of $\dot{V}_O_2$ kinetics analysis provided that a sufficient number of repeat transitions are averaged. In the present study, the average of four moderate bouts and two heavy bouts was satisfactory, although it should be noted that an additional repetition at each of the intensities would have been required during rowing in order to produce a similar 95% confidence interval as that achieved during cycling.

In summary, $\dot{V}_O_2$ kinetics during moderate and heavy intensity rowing ergometer exercise were generally very similar to $\dot{V}_O_2$ kinetics at the same relative exercise intensities during cycle ergometer exercise in subjects who were not specifically trained for either mode of exercise. These results indicate that the factor or factors that regulate the $\dot{V}_O_2$ response to a step increase in muscle ATP turnover are similar, at least in modes of exercise which recruit a relatively large muscle mass and which rely predominantly on concentric muscle contraction for force generation. The results also indicate that the recruitment of a larger muscle mass than that which is recruited during two-legged upright cycle exercise, and which might be hypothesised to reduce the potential for adequate muscle perfusion, does not result in a significant slowing of the Phase II $\dot{V}_O_2$ kinetics or a significant increase in the amplitude of the $\dot{V}_O_2$ slow component, at least at a work rate requiring 50% “$\Delta$”.

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