Available online at www.sciencedirect.com
SCIENCE DIRECT®
Respiratory Physiology & Neurobiology 146 (2005) 247–258
RESPIRATORY PHYSIOLOGY & NEUROBIOLOGY
www.elsevier.com/locate/resphysiol

Pulmonary O_2 uptake on-kinetics in rowing and cycle ergometer exercise

Claire L. Roberts, Daryl P. Wilkerson, Andrew M. Jones*

Department of Exercise and Sport Science, Manchester Metropolitan University, Hassall Road, Alsager, ST & 2HL, UK

Received 2 November 2004; received in revised form 17 December 2004; accepted 20 December 2004

Abstract

The purpose of this study was to characterise, for the first time, the pulmonary O_2 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 $\Delta\dot{V}O_2/\Delta$WR 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.

Keywords: $\dot{V}O_2$ dynamics; Economy; Slow component; *Phase* II; Time constant

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

* Corresponding author. Tel.: +44 161 2475656; fax: +44 161 2476375.
E-mail address: a.m.jones@mmu.ac.uk (A.M. Jones).

1569-9048/$ – see front matter © 2004 Elsevier B.V. All rights reserved.
doi:10.1016/j.resp.2004.12.012

(Barstow and Molé, 1991; Linnarsson, 1974; Özyener et al., 2001; Whipp and Wasserman, 1972), running (Carter et al., 2000, 2002; Jones and McConnell, 1999), arm cranking (Casaburi et al., 1992; Koga et al., 1996; Koppo et al., 2002), swimming (Demarie 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 (Perrey et al., 2001; Pringle et al., 2002). Also, comparisons of upright cycling with arm cranking reveal that the time constant (τ_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 rowing 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 τ 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 τ_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 (>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 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 (see below) and heart rate (HR) was recorded telemetrically using a HR monitor (Polar Accurex, Kempele, Finland).

2.3. Incremental tests

The incremental tests began at a work rate of 30 W and work rate was subsequently increased by 30 W at the end of each minute. For rowing, subjects were instructed to row at a pace for 500 m that was equivalent to the required work rate as calculated from the formula work rate = 2.8 pace^{-3} (Concept II operator's instructions). Pulmonary gas exchange was assessed breath-by-breath throughout both 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 $\dot{V}CO_2$ 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% Δ) 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 in order 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,

The Netherlands) that was calibrated before each test with a high-precision 3 L graduated gas syringe (Hans Rudolph Inc., Kansas City, USA) according to the manufacturer's instructions. The concentration and volume signals were integrated by personal computer, and pulmonary gas exchange and ventilation variables were calculated and displayed in real-time for each breath. A 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 onset of unloaded exercise rather than 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 $\dot{V}O_2$ kinetics

The breath-by-breath $\dot{V}O_2$ 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 $\dot{V}O_2$ was defined as the average $\dot{V}O_2$ measured during unloaded cycling between 150 and 30 s before the start of exercise. A single exponential model was used to analyse the $\dot{V}O_2$ 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:

$$\dot{V}O_2(t) = \dot{V}O_{2\ \text{baseline}} + A_p(1 - e^{-(t-Td_p)/\tau_p})$$

(90% GET)

$$\dot{V}O_2(t) = \dot{V}O_{2\ \text{baseline}} + A_p(1 - e^{-(t-Td_p)/\tau_p}) + A_s(1 - e^{-(t-Td_s)/\tau_s} \quad (50\%\ \Delta)$$

The exponential models include amplitudes (A_p and A_s), time constants (τ_p and τ_s) and time delays (Td_p and Td_s) that were determined using a non-linear least-square algorithm in which minimising the sum of squared error was the criterion for convergence. A_p, τ_p and Td_p describe the parameters related to the $\dot{V}O_2$ primary component, while A_s, τ_s and Td_s describe the parameters related to the $\dot{V}O_2$ 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 $\dot{V}O_2$ 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_T) $\dot{V}O_2$ responses were calculated as the appropriate amplitude of $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ max or the $\dot{V}O_2$ 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 50% Δ

Table 1
Mean ± S.D. physiological responses to incremental rowing and cycle ergometer exercise

	Rowing	Cycling
$\dot{V}O_2$ max (L min^{-1})	3.40 ± 0.34	3.38 ± 0.42
$\dot{V}O_2$ max (mL kg^{-1} min^{-1})	44.1 ± 6.3	43.9 ± 6.9
HR max (b min^{-1})	177 ± 11	181 ± 14
Peak work rate (W)	179 ± 25	211 ± 35
$\dot{V}O_2$ at GET (L min^{-1})	1.73 ± 0.17	1.62 ± 0.21
Work rate at GET (W)	76 ± 12	106 ± 24*

HR, heart rate; GET, gas exchange threshold.
* Denotes the existence of a significant difference ($P < 0.05$).

Table 3
Mean ± S.D. $\dot{V}O_2$ kinetic responses to a step transition to a 'moderate' work rate for rowing and cycle ergometer exercise

	Rowing	Cycling
Δ Work rate (W)	96 ± 12	106 ± 24*
Baseline $\dot{V}O_2$ (L min^{-1})	0.71 ± 0.08	0.67 ± 0.05
Phase II time constant (s)	25.9 ± 6.8	25.7 ± 8.6
Primary amplitude (L min^{-1})	1.13 ± 0.13	1.10 ± 0.23
Primary gain (mL min^{-1} W^{-1})	11.8 ± 1.1	10.5 ± 1.0*
End-exercise $\dot{V}O_2$ (L min^{-1})	1.82 ± 0.13	1.77 ± 0.26

* Denotes the existence of a significant difference ($P < 0.05$).

were 199 ± 25 and 231 ± 35 W for rowing and cycling, respectively.

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 $\dot{V}O_2$ 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 exercise modalities for baseline $\dot{V}O_2$, the amplitude of the $\dot{V}O_2$ response above baseline, or the percentage of the mode-specific $\dot{V}O_2$ max attained at the end of exercise. Importantly, in relation to our experimental hypotheses, there was also no significant difference in τ_p between the exercise modes (rowing: 25.9 ± 6.8 s versus cycling: 25.7 ± 8.6 s). The mean 95% confidence intervals surrounding the estimate of τ_p were 2.5 and 1.4 s for rowing and cycling, respectively. There was a significant difference in the G_p between the exercise modes (rowing: 11.8 ± 1.1 mL min^{-1} W^{-1} versus cycling: 10.5 ± 1.0 mL min^{-1} W^{-1}; $P < 0.05$), signifying a greater increase in $\dot{V}O_2$ per unit increase in work rate in rowing compared to cycling.

The $\dot{V}O_2$ response kinetics to heavy intensity cycling and rowing are summarised in Table 4 and illustrated in Fig. 1. There was no significant difference between the modes of exercise for baseline $\dot{V}O_2$, the amplitudes of the primary or slow components of $\dot{V}O_2$, or the $\dot{V}O_2$ attained at the end of exercise. Furthermore, τ_p was not significantly different between the exercise modes (rowing: 26.5 ± 3.0 s versus cycling: 27.8 ± 5.1 s). The mean 95% confidence intervals surrounding the estimate of τ_p were 2.0 and 1.5 s for rowing and cycling, respectively. As for moderate exercise, there was a significant difference in G_p (rowing: 10.8 ± 0.8 mL min^{-1} W^{-1} versus cycling: 9.7 ± 1.0 mL min^{-1} W^{-1}; $P < 0.05$). This higher O_2 cost of exercise per unit increase in work rate in rowing was also evident at the end of exercise (G_T; Row: 12.5 ± 1.0 mL min^{-1} W^{-1} versus cycling: 11.3 ± 1.6 mL min^{-1} W^{-1}; $P < 0.05$).

When the $\dot{V}O_2$ 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 τ_p (moderate: 25.7 ± 8.6 s versus heavy: 27.8 ± 5.1 s). There was, however, a tendency for G_p 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 2
Mean ± S.D. blood lactate and heart rate responses to step transitions to moderate and heavy exercise in rowing and cycle ergometer exercise

	Rowing moderate	Cycling moderate	Rowing heavy	Cycling heavy
Δ Blood [lactate] (Mm)	0.9 ± 0.6	0.6 ± 0.4	5.5 ± 2.2	4.5 ± 1.8
Exercise HR (b min^{-1})	124 ± 10	118 ± 12	166 ± 12	158 ± 10
%HR max	70 ± 6	65 ± 6	93 ± 7	87 ± 5

HR, heart rate.

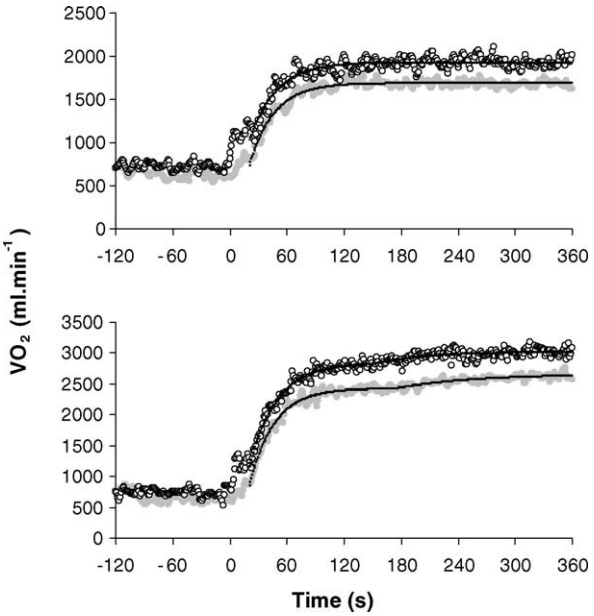

Fig. 1. Oxygen uptake responses following the onset of moderate (upper panel) and heavy (lower panel) rowing and cycle ergometer exercise in a representative subject. Moderate rowing, open circles; moderate cycling, closed grey circles; heavy rowing, open circles; heavy cycling, closed grey circles. The solid lines represent the model fits. Note the greater O_2 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
Mean ± S.D. $\dot{V}O_2$ kinetic responses to a step transition to a 'heavy' work rate for rowing and cycle ergometer exercise

	Rowing	Cycling
Δ Work rate (W)	199 ± 25	211 ± 35*
Baseline $\dot{V}O_2$ (L min^{-1})	0.71 ± 0.09	0.66 ± 0.06
Phase II time constant (s)	26.5 ± 3.0	27.8 ± 5.1
Primary amplitude (L min^{-1})	2.14 ± 0.28	2.03 ± 0.33
Primary gain (mL min^{-1} W^{-1})	10.8 ± 0.8	9.7 ± 1.0*
SC time delay (s)	155 ± 35	129 ± 26
SC amplitude (L min^{-1})	0.34 ± 0.13	0.35 ± 0.12
SC amplitude (% of end-exercise $\dot{V}O_2$)	14 ± 1	13 ± 1
End-exercise $\dot{V}O_2$ (L min^{-1})	3.15 ± 0.32	2.94 ± 0.37
End-exercise gain (mL min^{-1} W^{-1})	12.5 ± 1.0	11.3 ± 1.6

SC, $\dot{V}O_2$ slow component.
* Denotes the existence of a significant difference ($P < 0.05$).

However, the development of the $\dot{V}O_2$ 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 τ_p between the two exercise intensities (moderate: 25.9 ± 6.8 s versus heavy: 26.5 ± 3.0 s). However, in this mode of exercise, G_p 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 $\dot{V}O_2$ 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.

4. Discussion

To our knowledge, this is the first study to characterise $\dot{V}O_2$ kinetics during rowing ergometer exercise. Our principal finding was that the $\dot{V}O_2$ 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, $\dot{V}O_2$ rose exponentially in *Phase* II to attain a steady-state within ~2–3 min. Following the onset of heavy intensity rowing, $\dot{V}O_2$ rose 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 $\dot{V}O_2$ that emerged after 2–3 min of exercise. One notable difference between the exercise modes was the significantly greater O_2 cost of exercise (i.e. greater $\Delta\dot{V}O_2/\Delta WR$) during rowing compared to cycle exercise.

The similarity of the τ_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

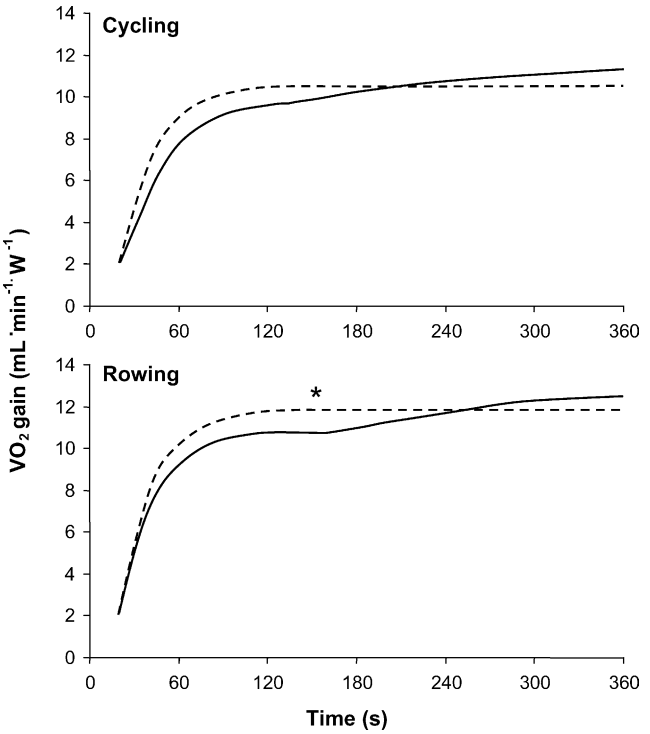

Fig. 2. Schematic illustration, based on the group mean data, of the influence of work rate on the gain ($\Delta\dot{V}O_2/\Delta WR$) of the $\dot{V}O_2$ response to moderate (dashed line) and heavy (solid line) exercise for cycling (upper panel) and rowing (lower panel) ergometer exercise. Note that the gain of the primary component is reduced at heavy compared to moderate work rates in both exercise modes. However, the emergence of the $\dot{V}O_2$ slow component during heavy exercise causes the end-exercise gain to be higher for heavy exercise compared to moderate exercise. The asterisk (*) denotes a significant difference between primary component gain in heavy and moderate rowing exercise ($P < 0.05$).

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 τ_p. Consistent with this, Koga et al. (2001) reported that there was no significant difference in τ_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 τ_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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 τ_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 τ_p for cycle exercise (i.e. ~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 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; Rossiter et al., 1999). The similarity of τ_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 τ_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 τ_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; Steinacker 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 (~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 (~340–350 mL min^{-1}) or in relative (~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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 on the amplitude of the $\dot{V}O_2$ slow component.

Several studies suggest a relationship between the $\dot{V}O_2$ slow component and the recruitment of type II muscle fibres (Barstow et al., 1996; Pringle et al., 2003a,b; Krustrup 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 $\dot{V}O_2$ 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] 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 $\dot{V}O_2$ 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 $\dot{V}O_2$. 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ slow component, which is superimposed on this response, subsequently elevates the G_T above 10 mL min^{-1} W^{-1} (Barstow and Molé, 1991; Paterson and Whipp, 1991; Whipp and Ward, 1990). Our data are subtly different in that they suggest that the $\dot{V}O_2$ slow component initially 'compensates' for the reduced G_p at higher work rates (i.e. the slow component brings $\dot{V}O_2$ closer to the value that would be 'expected' for the work rate as calculated from an extrapolation of the $\dot{V}O_2$-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 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 (Krustrup et al., 2004; Vøllestad 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 PO_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 τ_p 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% "Δ".

References

Astrand, P.-O., Saltin, B., 1961. Maximal oxygen uptake and heart rate in various types of muscular activity. J. Appl. Physiol. 16, 977–981.

Barstow, T.J., Jones, A.M., Nguyen, P.H., Casaburi, R., 1996. Influence of muscle fibre type and pedal frequency on oxygen uptake kinetics of heavy exercise. J. Appl. Physiol. 81, 472–478.

Barstow, T.J., Molé, P., 1991. Linear and non-linear characteristics of oxygen uptake kinetics during heavy exercise. J. Appl. Physiol. 71, 2099–2106.

Behnke, B.J., McDonough, P., Padilla, D.J., Musch, T.I., Poole, D.C., 2003. Oxygen exchange profile in rat muscles of contrasting fibre type. J. Physiol. 549, 597–605.

Billat, V., Binsse, V., Petit, B., Koralsztein, J.P., 1998. High level runners are able to maintain a $\dot{V}O_2$ steady-state below $\dot{V}O_2$ max in an all-out run over their critical velocity. Arch. Physiol. Biochem. 106, 38–45.

Burnley, M., Doust, J.H., Ball, D., Jones, A.M., 2002. Effects of prior heavy exercise on heavy exercise $\dot{V}O_2$ kinetics are related to changes in muscle activity. J. Appl. Physiol. 93, 167–174.

Burnley, M., Jones, A.M., Carter, H., Doust, J.H., 2000. Effects of prior heavy exercise on *phase* II pulmonary oxygen uptake kinetics during heavy exercise. J. Appl. Physiol. 89, 1387–1396.

Carter, H., Jones, A.M., Barstow, T.J., Burnley, M., Williams, C.A., Doust, J.H., 2000. Oxygen uptake kinetics in treadmill running and cycle ergometry: a comparison. J. Appl. Physiol. 89, 899–907.

Carter, H., Pringle, J.S.M., Jones, A.M., Doust, J.H., 2002. Oxygen uptake kinetics during treadmill running across exercise intensity domains. Eur. J. Appl. Physiol. 86, 347–354.

Casaburi, R., Barstow, T.J., Robinson, T., Wasserman, K., 1992. Dynamic and steady-state ventilatory and gas exchange responses to arm exercise. Med. Sci. Sports. Exerc. 24, 1365–1374.

Cerretelli, P., Shindell, D., Pendergast, D., Rennie, D.W., 1977. Oxygen uptake transients at the onset and offset of arm and leg work. Respir. Physiol. 30, 81–97.

Cerretelli, P., Pendergast, D., Paganelli, W.C., Rennie, D.W., 1979. Effects of specific muscle training on $\dot{V}O_2$ on-response and early blood lactate. J. Appl. Physiol. 47, 761–769.

Chance, B., Williams, C.M., 1955. Respiratory enzymes in oxidative phosphorylation. I. Kinetics of oxygen utilisation. J. Biol. Chem. 217, 383–393.

Chilibeck, P.D., Paterson, D.H., Smith, W.D., Cunningham, D.A., 1996. Cardiorespiratory kinetics during exercise of different muscle groups and mass in old and young. J. Appl. Physiol. 81, 1388–1394.

Clausen, J.P., 1976. Circulatory adjustments to dynamic exercise and effect of physical training in normal subjects and in patients with coronary artery disease. Prog. Cardiovasc. Dis. 18, 459–495.

Cunningham, D.A., Goode, P.B., Critz, J.B., 1975. Cardiorspiratory response to exercise on a rowing and bicycle ergometer. Med. Sci. Sports Exerc. 7, 37–43.

Demarie, S., Sardella, F., Billat, V., Magini, W., Faina, M., 2001. The $\dot{V}O_2$ slow component in swimming. Eur. J. Appl. Physiol. 84, 95–99.

Engelen, M., Porszasz, J., Riley, M., Wasserman, K., Maehara, K., Barstow, T.J., 1996. Effects of hypoxic hypoxia on O_2 uptake and heart rate kinetics during heavy exercise. J. Appl. Physiol. 81, 2500–2508.

Gaesser, G.A., Ward, S.A., Baum, V.C., Whipp, B.J., 1994. Effects of infused epinephrine on slow phase of O_2 uptake kinetics during heavy exercise in humans. J. Appl. Physiol. 77, 2413–2419.

Gerbino, A., Ward, S.A., Whipp, B.J., 1996. Effects of prior exercise on pulmonary gas exchange kinetics during high-intensity exercise in humans. J. Appl. Physiol. 80, 99–107.

Gollnick, P.D., Armstrong, R.B., Saubert, C.W., Piehl, K., Saltin, B., 1972. Enzyme activity and fiber composition of skeletal muscle of untrained and trained men. J. Appl. Physiol. 33, 312–319.

Haseler, L.J., Kindig, C.A., Richardson, R.S., Hogan, M.C., 2004. The role of oxygen in determining phosphocreatine onset kinetics in exercising humans. J. Physiol. 558, 985–992.

Hughson, R.L., Tschakovsky, M.E., Houston, M.E., 2001. Regulation of oxygen consumption at the onset of exercise. Ex. Sport. Sci. Rev. 29, 129–133.

Johnson, M.A., Polgar, J., Weightman, D., Appleton, D., 1973. Data on the distribution of fiber types in thirty-six human muscles: an autopsy study. J. Neuro. Sci. 18, 111–129.

Jones, A.M., Burnley, M., 2005. Effect of exercise modality on $\dot{V}O_2$ kinetics. In: Jones, A.M., Poole, D.C. (Eds.), Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London, pp. 95–114.

Jones, A.M., Carter, H., 2000. The effect of endurance training on parameters of aerobic fitness. Sport. Med. 29, 373–386.

Jones, A.M., Carter, H., Pringle, J.S.M., Campbell, I.T., 2002. Effect of creatine supplementation on oxygen uptake kinetics during sub-maximal cycle exercise. J. Appl. Physiol. 92, 2571–2577.

Jones, A.M., Koppo, K., 2005. Effect of training on $\dot{V}O_2$ kinetics and performance. In: Jones, A.M., Poole, D.C. (Eds.), Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London, pp. 373–397.

Jones, A.M., McConnell, A.M., 1999. Effect of exercise modality on oxygen uptake kinetics during heavy exercise. Eur. J. Appl. Physiol. 80, 213–219.

Knight, D.R., Wong, P.Y., Woodard, S., Allen, H.D., 2004. Leg tourniquets modify the on-kinetics of oxygen uptake during cycle exercise. J. Cardiopulm. Rehabil. 24, 128–135.

Koga, S., Barstow, T.J., Shiojiri, T., Takaishi, T., Fukuba, Y., Shibasaki, M., Poole, D.C., 2001. Effect of muscle mass on $\dot{V}O_2$ kinetics at the onset of work. J. Appl. Physiol. 90, 461–468.

Koga, S., Poole, D.C., Shiojiri, T., Kondo, N., Fukuba, Y., Miura, A., Barstow, T.J., 2004. A comparison of oxygen uptake kinetics during knee extension and cycle exercise. Am. J. Physiol. Regul. Integr. Comp. Physiol., [E-Pub ahead of print], in press.

Koga, S., Shiojiri, T., Shibasaki, M., Fukuba, Y., Fukuoka, Y., Kondo, N., 1996. Kinetics of oxygen uptake and cardiac output at onset of arm exercise. Respir. Physiol. 103, 195–202.

Koga, S., Shiojiri, T., Shibasaki, M., Kondo, N., Fukuba, Y., Barstow, T.J., 1999. Kinetics of oxygen uptake during supine and upright exercise. J. Appl. Physiol. 87, 253–260.

Koppo, K., Bouckaert, J., Jones, A.M., 2002. Oxygen uptake kinetics during high-intensity arm and leg exercise. Respir. Physiol. Neurobiol. 133, 241–250.

Krustrup, P., Soderlund, K., Mohr, M., Bangsbo, J., 2004. The slow component of oxygen uptake during intense, sub-maximal exercise in man is associated with additional fibre recruitment. Pflugers Arch. 447, 855–866.

Linnarsson, D., 1974. Dynamics of pulmonary gas exchange and heart rate changes at start and end of exercise. Acta Physiol. Scand. 415, 1–68.

MacDonald, M.J., Pedersen, P.K., Hughson, R.L., 1997. Acceleration of $\dot{V}O_2$ kinetics in heavy sub-maximal exercise by hyperoxia and prior high-intensity exercise. J. Appl. Physiol. 83, 1318–1325.

MacDonald, M.J., Shoemaker, J.K., Tschakovsky, M.E., Hughson, R.L., 1998. Alveolar oxygen uptake and femoral artery blood flow dynamics in upright and supine leg exercise in humans. J. Appl. Physiol. 85, 1622–1628.

Mahler, D.A., Hunter, B., Lentine, T., Ward, J., 1991. Ventilatory responses and entrainment of breathing during rowing. Med. Sci. Sport. Exerc. 23, 186–192.

Mahler, M., 1985. First order kinetics of muscle oxygen consumption, and equivalent proportionality between $\dot{V}O_2$ and phosphorylcreatine level implications for the control of respiration. J. Appl. Physiol. 86, 135–165.

Özyener, F., Rossiter, H.B., Ward, S.A., Whipp, B.J., 2001. Influence of exercise intensity on the on- and off-transient kinetics of pulmonary oxygen uptake in humans. J. Appl. Physiol. 533, 891–902.

Paterson, D.H., Whipp, B.J., 1991. Asymmetries of oxygen uptake transients at the on- and off-set of heavy exercise in humans. J. Physiol. 443, 575–586.

Perrey, S., Betik, A., Candau, R., Rouillon, J.D., Hughson, R.L., 2001. Comparison of oxygen uptake kinetics during concentric and eccentric cycle exercise. J. Appl. Physiol. 91, 2135–2142.

Phillips, S.M., Green, H.J., MacDonald, M.J., Hughson, R.L., 1995. Progressive effect of endurance training on $\dot{V}O_2$ kinetics at the onset of sub-maximal exercise. J. Appl. Physiol. 79, 1914–1920.

Poole, D.C., Jones, A.M., 2005. Towards an understanding of the mechanistic bases of $\dot{V}O_2$ kinetics. In: Jones, A.M., Poole, D.C. (Eds.), Oxygen Uptake Kinetics in Sport, Exercise and Medicine. Routledge, London, pp. 294–328.

Pringle, J.S.M., Carter, H., Doust, J.H., Jones, A.M., 2002. Oxygen uptake kinetics during horizontal and uphill treadmill running. Eur. J. Appl. Physiol. 88, 163–169.

Pringle, J.S.M., Doust, J.H., Carter, H., Tolfrey, K., Campbell, I.T., Sakkas, G.K., Jones, A.M., 2003a. Oxygen uptake kinetics during constant-load 'sub-maximal' exercise in humans: the influence of muscle fibre type and capillarisation. Eur. J. Appl. Physiol. 89, 289–300.

Pringle, J.S.M., Doust, J.H., Carter, H., Tolfrey, K., Jones, A.M., 2003b. Effect of pedal rate on primary and slow component oxygen uptake responses during heavy cycle exercise. J. Appl. Physiol. 94, 1501–1507.

Richardson, R.S., Kennedy, B., Knight, D.R., Wagner, P.D., 1995. High muscle blood flows are not attenuated by recruitment of additional muscle mass. Am. J. Physiol. 269, H1545–H1552.

Rossiter, H.B., Ward, S.A., Doyle, V.L., Howe, F.A., Griffiths, J.R., Whipp, B.J., 1999. Inferences from pulmonary O_2 uptake with respect to intramuscular [PCr] kinetics during moderate exercise in humans. J. Physiol. 518, 921–932.

Roston, W.L., Whipp, B.J., Davis, J.A., Cunningham, D.A., Effros, R.M., Wasserman, K., 1987. Oxygen uptake kinetics and lactate concentration during exercise in humans. Am. Rev. Respir. Dis. 135, 1080–1084.

Schneider, D.A., Wing, A.N., Morris, N.R., 2002. Oxygen uptake and heart rate kinetics during heavy exercise: a comparison between arm cranking and leg cycling. Eur. J. Appl. Physiol. 88, 100–106.

Scheuermann, B.W., Barstow, T.J., 2003. O_2 uptake kinetics during exercise at peak O_2 uptake. J. Appl. Physiol. 95, 2014–2022.

Secher, N.H., 1993. Physiological and biomechanical aspects of rowing. J. Sport. Sci. 15, 24–42.

Secher, N.H., Clausen, J.P., Klausen, K., Noer, I., Trap-Jensen, J., 1977. Central and regional circulatory effects of adding arm exercise to leg exercise. Acta Physiol. Scand. 100:, 288–297.

Steinacker, J.M., Both, M., Whipp, B.J., 1993. Pulmonary mechanics and entrainment of respiration and stroke rate during rowing. Int. J. Sport. Med. 14, S15–S19.

Steinacker, J.M., Marx, T.R., Marx, U., Lormes, W., 1986. Oxygen consumption and metabolic strain in rowing ergometer exercise. Eur. J. Appl. Physiol. 55, 240–247.

Volianitis, S., Secher, N.H., 2002. Arm blood flow and metabolism during arm and combined arm and leg exercise in humans. J. Physiol. 544, 977–984.

Vøllestad, N.K., Blom, P.C.S., 1985. Effect of varying exercise intensity on glycogen depletion in human muscle fibres. Acta Physiol. Scand. 125, 395–405.

Whipp, B.J., Ward, S.A., 1990. Physiological determinants of pulmonary gas exchange kinetics during exercise. Med. Sci. Sport. Exerc. 22, 62–71.

Whipp, B.J., Ward, S.A., Lamarra, N., Davis, J.A., Wasserman, K., 1982. Parameters of ventilatory and gas exchange dynamics during exercise. J. Appl. Physiol. 52, 1506–1513.

Whipp, B.J., Wasserman, K., 1972. Oxygen uptake kinetics for various intensities of constant load work. J. Appl. Physiol. 33, 351–356.

Wilkerson, D.P., Koppo, K., Barstow, T.J., Jones, A.M., 2004. Effect of work rate on the functional 'gain' of *Phase* II pulmonary O_2 uptake response to exercise. Respir. Physiol. Neurobiol. 142, 211–223.