Effect of pedal rate on primary and slow-component oxygen uptake responses during heavy-cycle exercise

Jamie S. M. Pringle,1 Jonathan H. Doust,2 Helen Carter,3 Keith Tolfrey,1 and Andrew M. Jones1

1Department of Exercise and Sport Science, Manchester Metropolitan University, Alsager ST7 2HL; 2Department of Sport Science, University of Wales, Aberystwyth, Ceredigion SY23 2AX; and 3School of Sport, Exercise and Leisure, University of Surrey, Roehampton, London SW15 3SN, United Kingdom

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Pringle, Jamie S. M., Jonathan H. Doust, Helen Carter, Keith Tolfrey, and Andrew M. Jones. Effect of pedal rate on primary and slow-component oxygen uptake responses during heavy-cycle exercise. J Appl Physiol 94: 1501–1507, 2003. First published December 20, 2002; 10.1152/japplphysiol.00456.2002.—We hypothesized that a higher pedal rate (assumed to result in a greater proportional contribution of type II motor units) would be associated with an increased amplitude of the O2 uptake (V˙O2) slow component during heavy-cycle exercise. Ten subjects (mean ± SD, age 26 ± 4 yr, body mass 71.5 ± 7.9 kg) completed a series of square-wave transitions to heavy exercise at pedal rates of 35, 75, and 115 rpm. The exercise power output was set at 50% of the difference between the pedal rate-specific ventilatory threshold and peak V˙O2, and the baseline power output was adjusted to account for differences in the O2 cost of unloaded pedaling. The gain of the V˙O2 primary component was significantly higher at 35 rpm compared with 75 and 115 rpm (mean ± SE, 10.6 ± 0.3, 9.5 ± 0.2, and 8.9 ± 0.4 ml·min⁻¹·W⁻¹, respectively; P < 0.05). The amplitude of the V˙O2 slow component was significantly greater at 115 rpm (328 ± 29 ml/min) compared with 35 rpm (109 ± 30 ml/min) and 75 rpm (202 ± 38 ml/min) (P < 0.05). There were no significant differences in the time constants or time delays associated with the primary and slow components across the pedal rates. The change in blood lactate concentration was significantly greater at 115 rpm (3.7 ± 0.2 mM) and 75 rpm (2.8 ± 0.3 mM) compared with 35 rpm (1.7 ± 0.4 mM) (P < 0.05). These data indicate that pedal rate influences V˙O2 kinetics during heavy exercise at the same relative intensity, presumably by altering motor unit recruitment patterns.

increased oxygen cost associated with increased rates of pulmonary ventilation and cardiac output, lactate clearance or gluconeogenesis, and elevated core and/or muscle temperature (for review, see Ref. 42). However, neither elevation of blood lactate concentration ([La]) by infusion of epinephrine (17) nor elevation of muscle temperature by passive warming (22) increased the amplitude of the V˙O2 slow component. Furthermore, Poole et al. (29) demonstrated that extramuscular sources of increased VO2, such as the oxygen cost of additional respiratory muscle work, could contribute no more than ~15% to the V˙O2 slow component. This study indicated that the primary source of the additional O2 cost of heavy exercise was located within the exercising muscle, with the recruitment of “low-efficiency” type II muscle fibers being a strong candidate (29). Barstow et al. (2) demonstrated that the %type II fibers in the vastus lateralis was significantly correlated with the relative amplitude of the V˙O2 slow component during heavy-cycle exercise, and these observations have recently been confirmed (31). It is, therefore, possible that the V˙O2 slow component is related to the recruitment of type II muscle fibers during heavy exercise.

It is widely accepted that the recruitment of type II muscle fibers is enhanced with increases in pedal rate for the same external power output (23, 35). Therefore, altering pedal rate at the same relative power output during heavy exercise might provide a useful model for examining the effect of type II muscle fiber recruitment on the V˙O2 slow component. Barstow et al. (2) have previously reported no effect of pedal rate on the V˙O2 slow component, but the exercise power outputs used were estimated from the responses to a single ramp test at 60 rpm, and subjects only performed one exercise bout at each of the pedal rates, which might limit confidence in the parameters derived from the curve-fitting procedures. Furthermore, the range of pedal rates tested was relatively narrow (45–90 rpm), and it is possible that this did not significantly affect motor unit recruitment patterns (23, 35). The effect of more
extreme differences in pedal rate on the VO2 slow component, therefore, remains to be determined.

Previous investigations into the effect of pedal rate on the physiological response to exercise have typically studied subjects exercising at the same external power output over a number of different pedal rates and reported the gross efficiency (i.e., the absolute external power output divided by the absolute VO2) (7, 15, 37). These studies have shown that, for any given power output, gross efficiency decreases with increases in pedal rate, an effect that can be attributed largely to the “extra” O2 cost of turning the legs at the faster movement frequency. Failure to account for differences in the O2 cost of “unloaded” cycling could obscure any differences in mechanical efficiency across pedal rates (as expressed by the change (Δ) in VO2 above baseline per unit change in external power output; equivalent to “delta” efficiency). Furthermore, to allow meaningful physiological comparisons, the relative metabolic stress of the exercise must be normalized relative to any differences in the peak VO2 (VO2 peak) and the VT that might exist across pedal rates.

The purpose of the present study was to test the hypothesis that increased pedal rate (assumed to result in a greater recruitment of type II motor units) would be associated with a larger VO2 slow component. To facilitate comparisons of the VO2 response across the pedal rates, the baseline power output was adjusted to equate the baseline VO2 and the increase in power output above that at baseline was designed to result in the same relative metabolic rate (i.e., 50% of the difference between VT and VO2 peak for each pedal rate).

METHODS

Subjects. Ten healthy subjects (8 men; means ± SD, age 26 ± 4 yr, body mass 71.5 ± 7.9 kg) were briefed as to the benefits and risks of participation and gave written, informed consent to participate in the study, which was approved by the Ethics Committee of the Manchester Metropolitan University. All subjects were involved in regular exercise training and were familiar with laboratory exercise testing procedures. Subjects were instructed to avoid strenuous exercise and were familiar with laboratory exercise testing procedures. Within 2 wk of completing the ramp tests, each subject returned to the laboratory to perform constant-load exercise bouts at the different pedal rates. To enhance the signal-to-noise ratio in the VO2 signal, subjects performed three to four square-wave transitions at each pedal rate from baseline (see below) to a power output calculated to require 50% of the difference between VT and VO2 peak for each pedal rate (i.e., 50%Δ “heavy” exercise). The order of these tests was randomized. After one of the transitions at each of the three pedal rates, the subjects completed a maximal 6-s sprint test to determine the extent of posttest fatigue relative to a rested control trial. In addition, the subjects performed four to eight transitions to 80% of the VT at one pedal rate (75 rpm) to establish the primary component gain in the “control” condition of moderate exercise.

Determination of VO2 peak and VT. The ramp tests started with the subject pedaling at the required pedal rate with an applied load of 20 W for 3 min. The power output was then increased by 5 W every 12 s (25 W/min) until volitional exhaustion was reached or the required pedal rate could not be maintained. During all exercise tests, pulmonary gas exchange was measured breath by breath (see below).

The breath-by-breath VO2 data were interpolated to give second-by-second values. The highest VO2 value in any 30-s period was taken to be the VO2 peak. The VT was determined as the point at which a nonlinear increase in carbon dioxide production relative to VO2 was evident (4).

Square-wave transitions. Each subject completed three to four square-wave transitions consisting of 4 min of baseline pedaling followed by an abrupt transition to 6 min of heavy exercise at a power output calculated to require 50%Δ at each of the three pedal rates (35, 75, and 115 rpm). The subjects also performed a total of four to eight transitions to 80% VT at 75 rpm. In each laboratory visit, subjects completed a moderate-intensity exercise transition followed 10 min later by a heavy-exercise transition; this sequence was repeated after 60-min recovery. After one heavy transition at each of the three pedal rates, subjects immediately dismantled the electrically braked ergometer and mounted an adjacent friction-braked ergometer (Monark 824E, Varberg, Sweden) within 10 s. After ~3 s of zero-load cycling to raise the pedal rate to 60 rpm, they performed a maximal 6-s sprint with the loading of the ergometer set at 7.5% of the subject’s body mass. The angular velocity of the flywheel was sampled at 20 Hz by a personal computer, and power output was calculated and recorded. The peak power output in each sprint was compared with that previously obtained under resting conditions (i.e., with no prior exercise).

The power outputs at 50%Δ were calculated by extrapolation of the linear relationship between VO2 and power output in the sub-VT portion of the respective ramp tests, with correction made for the lag time in VO2, which occurs during ramp exercise. At the two lowest pedal rates, “baseline” pedaling was performed with added load to account for differences in the O2 cost of unloaded cycling that resulted from increased internal work at higher pedal rates. The exact load applied was calculated by using the ΔVO2/spower output relationship established from the sub-VT region of the corresponding ramp test. Baseline VO2 was, therefore, similar across the three pedal rates before the transitions to heavy exercise. The power output required to elicit 50%Δ for each of the pedal rates was then added to the respective baseline power outputs to produce the exercise power outputs for each of the three conditions.
A fingertip capillary blood sample was taken immediately before and after two transitions at each pedal rate to determine the Δ[La]. Approximately 20–25 μL of blood were collected into capillary tubes and analyzed for whole blood [La] by using a YSI 1500 Sport lactate analyzer (Yellow Springs Instruments). Heart rate was recorded every 5 s during all exercise tests by telemetry (Polar Electro Oy, Kempele, Finland).

**Measurement of pulmonary gas exchange and minute ventilation.** For all exercise trials, pulmonary gas exchange and minute ventilation were continuously measured by breath. Subjects wore a nose clip and breathed through a low-dead space (35 m) low-resistance (<0.1 kPa·s·L−1·min−1 at 16 L/s) mouthpiece and volume sensor assembly. Gases were continuously drawn from the mouthpiece assembly through a capillary line and analyzed for O₂ and CO₂ concentrations by fast-response analyzers (O₂: differential paramagnetic; CO₂: infrared absorption) (Oxycon Alpha, Jaeger, The Netherlands). The system was calibrated before each test with gases of known concentration. Expiratory volumes were determined by using a Triple V turbine volume sensor (Jaeger), which was calibrated before each test with a 3-liter graduated gas syringe (Hans Rudolph, Kansas City, MO), 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.

**Data analysis.** The breath-by-breath VO₂ data for each transition were interpolated to give second-by-second values and time aligned to the start of exercise. The repeat transitions for each condition were then averaged to enhance the underlying response characteristics. Nonlinear regression techniques were used to fit the first 6 min of VO₂ data after the onset of exercise with an exponential function. An iterative process was used to minimize the sum of squared error between the fitted function and the observed values. The empirical model consisted of two (moderate exercise) or three (heavy exercise) exponential terms, with each representing one phase of the response. The first exponential term started with the onset of exercise [time (t) = 0], whereas the other terms began after independent time delays

\[
VO_2(t) = VO_2(BL) + A_c \times (1 - e^{-\tau_c t}) + A_p \times (1 - e^{-\tau_p t}) + A_s \times (1 - e^{-\tau_s t})
\]

where VO₂(t) is the VO₂ at any given time point; VO₂(BL) is the baseline VO₂ value; A_c, A_p, and A_s are the asymptotic amplitudes for the exponential terms fitting the cardiodynamic, primary, and slow components, respectively; τ_c, τ_p, and τ_s are the respective time constants; and TD_p and TD_s are the time delays. The phase I term was terminated at the start of phase II (i.e., at TD_p) and assigned the value for that time (A_c). The VO₂ at the end of phase I (A_c) and the amplitude of phase II (A_p) were summed to calculate the amplitude of the primary component (A_p). The amplitude of the VO₂ slow component was determined as the increase in VO₂ from TD_s to the end of the modeled data (defined as A_s).

**Statistical analysis.** The statistical software package SPSS (version 10.0, SPSS, Chicago, IL) was used for all statistical analyses. Comparisons between responses at the different pedal rates were made by using a one-way, repeated-measures ANOVA with post hoc Bonferroni-adjusted paired t-tests. Statistical significance was accepted at 5%. Results are presented as means ± SE, unless stated otherwise.

**RESULTS**

**Ramp tests.** The VO₂ peak was not significantly different across pedal rates (3.58 ± 0.18, 3.55 ± 0.24, and 3.66 ± 0.216 l/min at 35, 75, and 115 rpm, respectively). However, the VO₂ at VT was significantly higher at 115 rpm (1.91 ± 0.10 l/min) compared with 35 (1.69 ± 0.07 l/min) and 75 (1.72 ± 0.11 l/min) rpm (P < 0.05). The %VO₂ peak at VT was not significantly different between pedal rates (~50% of VO₂ peak).

**Square-wave transitions.** The relative intensity achieved for moderate exercise at 75 rpm was 82 ± 3% VO₂ at VT. Table 1 shows the main results of the square-wave transitions to heavy exercise performed at the different pedal rates. There was no significant difference in the relative intensity achieved at the end of the primary component (BL + A_p) across the pedal rates (~55 ± 4%). The baseline VO₂ was not different across the different pedal rates; thus we were successful in accounting for differences in the O₂ cost of internal work.

Figure 1 shows the VO₂ response to heavy exercise at each pedal rate in a typical individual, whereas Fig. 2 shows the mean response across all 10 subjects. On average, there were no significant differences in the temporal aspects of the VO₂ kinetic response, although there was a tendency for TD_p and TD_s to occur earlier with increases in pedal rate and for the primary component time constant to be longer at higher pedal rates. The amplitude of the VO₂ primary component was not significantly different across pedal rates (1.466 ± 133, 1.536 ± 153, and 1.593 ± 157 ml/min at 35, 75, and 115 rpm, respectively). However, the amplitude of the VO₂ slow component increased with pedal rate and was significantly higher at 115 compared with 35 rpm, both in absolute terms and when expressed relative to the

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<tr>
<th>Exercise power, W</th>
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<th>VO₂ BL, l/min</th>
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**Table 1. Oxygen uptake response for 6 min of heavy cycle exercise at 35, 75, and 115 rpm**
end-exercise (EE) \( \dot{V}_O_2 \). The greater amplitude of the \( \dot{V}_O_2 \) slow component at 115 rpm resulted in the EE \( \dot{V}_O_2 \) being significantly higher at this pedal rate compared with 35 rpm.

Figure 3 shows the \( \dot{V}_O_2 \) response to each condition when \( \dot{V}_O_2 \) is expressed relative to the change in power output from the baseline to the exercise power output for each pedal rate, i.e., as a “gain” \( [\Delta \dot{V}_O_2/\Delta \text{work rate}] \). The gain of the primary component decreased with increases in pedal rate and was significantly lower at 75 (9.5 ± 0.2 ml·min\(^{-1}\)·W\(^{-1}\)) and 115 (8.9 ± 0.4 ml·min\(^{-1}\)·W\(^{-1}\)) compared with 35 rpm (10.6 ± 0.3 ml·min\(^{-1}\)·W\(^{-1}\)) and with moderate exercise (10.8 ± 0.5 ml·min\(^{-1}\)·W\(^{-1}\)) \((P < 0.05)\). The gain of the slow component increased as pedal rate increased and was significantly higher at 115 rpm (1.8 ± 0.2 ml·min\(^{-1}\)·W\(^{-1}\)) compared with 35 rpm (0.8 ± 0.2 ml·min\(^{-1}\)·W\(^{-1}\)) \((P < 0.05)\) but not with 75 rpm (1.3 ± 0.2 ml·min\(^{-1}\)·W\(^{-1}\)). The EE gain was not significantly different across the pedal rates (10.8 to 11.3 ml·min\(^{-1}\)·W\(^{-1}\)).

The \( \Delta \text{blood [La]} \) from baseline to EE increased with pedal rate and was significantly greater at 75 and 115 rpm compared with 35 rpm. The relative amplitude of the \( \dot{V}_O_2 \) slow component \((\Delta \dot{V}_O_2/\text{EE} \dot{V}_O_2)\) was significantly correlated with \( \Delta \text{blood [La]} \) at 35 rpm \((r = 0.65)\), 75 rpm \((r = 0.75)\), and 115 rpm \((r = 0.66)\) \((P < 0.05)\).

**Sprint tests.** Peak power output in the control condition (without prior exercise) was 1,055 ± 58 W. Prior heavy exercise led to a small but nonsignificant increase in peak power output at 35 rpm (1,090 ± 76 W) and 75 rpm (1,076 ± 88 W). However, prior heavy exercise at 115 rpm caused a 7% reduction in peak power output (983 ± 55 W) that was significantly lower than the value after prior exercise at 35 rpm \((P < 0.05)\).

**DISCUSSION**

Increasing pedal rate during constant-load heavy exercise of the same relative intensity resulted in a significant reduction in the gain of the primary component and a significant increase in the amplitude of the \( \dot{V}_O_2 \) slow component, with the latter being consistent with our hypothesis.

Barstow et al. (2) reported that differences in pedal rate between 45 and 90 rpm had no effect on the amplitude of the \( \dot{V}_O_2 \) slow component. However, it is possible that this range of pedal rates was too narrow to cause substantial differences in motor unit recruitment patterns \((23, 35)\). We used more extreme pedal rates in our study (between 35 and 115 rpm) in an attempt to evoke more substantial differences in the contribution of type II fibers to the external power output. Our study may be considered to have other advantages over the Barstow et al. study. Barstow et al. used a single ramp test at 60 rpm to estimate the power outputs required to elicit 50%\( \Delta \) across the range of pedal rates that they employed. In the present study, subjects performed separate ramp tests to determine the \( \dot{V}_O_2 \) peak, VT, and the \( \Delta \dot{V}_O_2/\Delta \text{WR} \) relationship for each of the three pedal rates used. This allowed us to
calculate more precisely the exercise power output required to elicit the target \( \dot{V}O_2 \) (i.e., 50%Δ) for each pedal rate. Furthermore, we used the \( \Delta \dot{V}O_2/\Delta WR \) relationship established from the ramp tests to calculate the additional loading that was required to account for differences in baseline \( \dot{V}O_2 \) resulting from the increased internal work at higher pedal rates. As a result, neither the baseline \( \dot{V}O_2 \) nor the relative exercise intensity (expressed as \( \% \dot{V}O_2 \) achieved) was significantly different across pedal rates. Finally, in our study, subjects completed three to four transitions at each of the pedal rates (compared with a single transition in the Barstow et al. study), providing improved confidence in the modeled parameters.

We have no direct evidence from our study that increased pedal rate resulted in increased type II fiber recruitment. However, a number of studies have indicated that the contribution of type II muscle fibers is greater at high-movement frequencies (5, 6, 32, 34, 35). Beelen et al. (6) demonstrated that, after 6 min of pedaling at 90% of the pedal rate-specific \( \dot{V}O_2 \) peak, glycogen depletion was greater in the type II fiber population at 120 rpm compared with 60 rpm. In another study, Beelen and Sargeant (5) demonstrated a greater reduction in peak power output after exercise at 120 rpm compared with 60 rpm and interpreted this to mean that prior exercise at the higher pedal rate resulted in fatigue of the type II fiber population. Similarly, in our study, heavy exercise at 115 rpm caused a 7% reduction in peak power output compared with the control condition. The differences in blood [La] observed between pedal rates might also be interpreted to indicate an enhanced recruitment of type II fibers at the highest pedal rate. However, it is difficult to separate the influence of fiber recruitment per se from the possible influence of absolute blood [La] on the slow component. Although the correlation between increases in \( \dot{V}O_2 \) and blood [La] with time during heavy exercise may be coincidental (17), it is also true that the slow component is only observed at power outputs that elicit a metabolic acidosis and that reductions in the slow-component amplitude with training are linked to reductions in \( \Delta \)blood [La] (e.g., Ref. 30). The influence of changes in pedal rate on the \( \dot{V}O_2 \) response to heavy exercise in subjects with differences in muscle fiber-type distribution is not known, and it is possible that subjects with “extremes” of muscle fiber-type distribution respond differently to changes in pedal rate. However, Barstow et al. (2) could determine no interaction between the muscle fiber type of their subjects and changes in pedal rate between 45 and 90 rpm.

Assuming that higher pedal rates did indeed enhance type II fiber recruitment, our results are consistent with cross-sectional studies that have demonstrated a significant correlation between \%type II muscle fibers and the relative amplitude of the \( \dot{V}O_2 \) slow component (2, 31). Barstow et al. (2) reported that the \%type II muscle fibers in the vastus lateralis were significantly positively correlated with the relative amplitude of the \( \dot{V}O_2 \) slow component during heavy-cycle exercise. More recently, our laboratory has also reported significant correlations between \%type II muscle fibers and the relative amplitude of the \( \dot{V}O_2 \) slow component during both heavy (\( r = 0.74 \)) and severe (\( r = 0.64 \)) exercise (31). Earlier studies (predominantly in rodent muscle) indicated that type II muscle fibers produced more heat and consumed more oxygen for the same rate of tension generation and ATP turnover than type I muscle fibers (12, 41). This presumed lower efficiency of contraction of type II muscle fibers led to the hypothesis that the progressive recruitment of type II motor units during heavy exercise was responsible for the development of the slow component (2, 16, 42). However, in a recent study, He et al. (19) reported that the peak thermodynamic efficiencies of slow- (\( \sim 0.21 \)) and fast-twitch (\( \sim 0.27 \)) fibers from human vastus lateralis muscle were not significantly different, despite almost fourfold differences in ATP hydrolysis rate and maximum mechanical power. Interestingly, peak efficiency in type IIA fibers was reached at a higher shortening velocity and greater relative load compared with type I fibers. This suggests that, although the contribution of type II fibers to the exercise power output is likely to be greater at higher pedal rates during heavy-cycle exercise, the effect on exercise efficiency might not be easily predicted.

An interesting finding in the present study was the reduction in the gain of the \( \dot{V}O_2 \) primary component with higher pedal rates at the same relative exercise intensity, an effect that is also evident in the data of Barstow et al. (2). It has generally been considered that the primary gain term represents the initially anticipated \( O_2 \) cost of exercise that is subsequently modified with time as the \( \dot{V}O_2 \) slow component emerges (3). Recent studies, however, indicate that the primary gain term (typically \( \sim 10 \text{ ml} \cdot \text{min}^{-1} \cdot \text{W}^{-1} \)) should not be considered an immutable feature of the \( \dot{V}O_2 \) response to exercise. Several studies have demonstrated a clear trend for the primary gain term to fall as power output is increased (9, 10, 21, 26), and it has been suggested that this might reflect an increased contribution of the characteristics of \( O_2 \) consumption in type II fibers to the pulmonary \( \dot{V}O_2 \) signal (10, 21). In the present study, the primary gain was significantly lower during heavy exercise compared with moderate exercise at 75 rpm (Fig. 2). The primary gain is negatively correlated with the \%type II fibers during heavy (2, 31) and severe (31) exercise. Also, the optimum velocity of shortening for mechanical efficiency in type II fibers is closer to 115 than to 35 rpm (13, 19, 23, 35), and the greater contribution of type II muscle fibers at high-power outputs and movement frequencies may serve to minimize muscle activation and maximize muscle efficiency (35). Therefore, the fall in the primary gain term with increases in pedal rate and exercise intensity might be explained by a greater proportional contribution of type II fibers at higher pedal rates and greater exercise intensities.

Although the mechanism responsible for the \( \dot{V}O_2 \) slow component is often assumed to be the progressive recruitment of type II fibers with time as heavy exercise proceeds (2, 16, 43), it is also possible that a high
proportion of (fatigue-sensitive) type II fibers are recruited at the onset of heavy exercise. This might be particularly true at high pedal rates (34). If it is accepted that type II fibers have a larger gain and slower kinetics than type I fibers, then our results are consistent with the view that a greater relative contribution of type II fibers to the power output would result in a lower primary gain and a greater slow component (2, 31). The suggestion that type II fibers may be recruited initially during heavy exercise is supported by a number of glycogen depletion studies (1, 14, 39, 40). It has been reported that all type I and IIA fibers are recruited from the start of exercise at 75% maximum \( \dot{V}O_2 \) (\( \dot{V}O_2_{\text{max}} \)) (40) and that all type I, IIA, and IIB fibers are recruited within 4–7 min of exercise at 84–100% \( \dot{V}O_2_{\text{max}} \) (1, 14). Vollestad and Blom (39) demonstrated that virtually all of the muscle fibers in the vastus lateralis were recruited within 10 min of the onset of exercise at 91% \( \dot{V}O_2_{\text{max}} \) and noted that the loss of force in fatigued fibers must be compensated by increased activity in other fibers to maintain the required tension. Therefore, an alternative hypothesis to explain the slow component (and its greater amplitude at higher pedal rates) is that fatigue in the type II fiber pool as exercise proceeds necessitates an increased activity (i.e., increased firing frequency) of type I fibers. Recent evidence indicates that type I fibers are relatively less efficient at higher force requirements and contraction velocities than are type II fibers (19). Increased activation of type I muscle fibers during heavy exercise (especially at higher pedal rates) might, therefore, reduce muscle efficiency. Additionally, fatigued type II fibers might continue to consume oxygen as they recover (i.e., there will be a continued phosphate and \( O_2 \) cost associated with Ca\(^{2+}\) and Na\(^{+}\)-K\(^{-}\) pumping as homeostasis is restored) but without contributing appreciably to force production. This suggestion is consistent with the recent data of Rossiter et al. (33), which indicate that the slow component is associated with a high-phosphate cost of force production, rather than a high-oxygen cost of phosphate production. Furthermore, the scenario of early recruitment and subsequent fatigue of the type II fiber pool followed by increased activation of the type I fibers is consistent with reports of an increased or unchanged integrated electromyogram but no change in the mean power frequency during heavy exercise (8, 28, 36). However, this suggestion remains speculative at present.

Whereas changes in motor unit recruitment patterns appear to provide the most likely explanation for the differences in the physiological responses that we observed, other factors must also be considered. For example, it is possible that higher pedal rates affect the extent or pattern (e.g., timing or duty cycle) of muscle recruitment and/or demand the involvement of other muscle groups. The reported effect of pedal rate on muscle activation is inconsistent, with some studies indicating an increase in electromyography at higher pedal rates (24), and others indicating that electromyography is minimized if pedal rate is increased with increases in power output (23). However, there is some evidence that cycling at high pedal rates requires the recruitment of additional muscles to stabilize the trunk (18) and causes a reduction in the effectiveness of the force applied at the pedals (27). Differences in contraction frequency might also influence blood flow to the exercising muscle. Hoelting et al. (20) recently demonstrated that increasing contraction frequency resulted in a reduction in mean blood flow during knee-extension exercise. A reduced blood flow might result in increased type II fiber recruitment and increased lactate production.

The greater slow-component amplitude at 115 rpm resulted in the EE \( \dot{V}O_2 \) at 6 min being significantly higher at this pedal rate than at 35 and 75 rpm. It is of note that previous studies that examined the influence of pedal rate on exercise efficiency have not considered the influence of the \( \dot{V}O_2 \) slow component on the measurement of exercise efficiency at power outputs above the VT. Our observations, made through careful partitioning of the \( \dot{V}O_2 \) response into its constituent primary and slow components, indicate that inconsistencies in the reported effect of differences in pedal rate on delta efficiency (11, 15, 25, 38) might be related both to the intensity of exercise and the time during exercise when \( \dot{V}O_2 \) was measured.

In conclusion, for exercise at the same relative intensity and after controlling for differences in the \( O_2 \) cost of unloaded pedaling, higher pedal rates were associated with a lower gain of the \( \dot{V}O_2 \) primary component and a greater amplitude of the \( \dot{V}O_2 \) slow component. Assuming that type II fibers possess slower kinetics and lower efficiency than type I fibers, one interpretation of these data is that they are related to the greater contribution of type II muscle fibers (relative to type I muscle fibers) during heavy exercise at higher pedal rates. However, this simple interpretation is confounded by the fact that type II fibers are relatively more efficient at higher contraction velocities. Whereas this latter point might help to explain the lower primary component gain, alternative explanations for the larger slow component at higher pedal rates should be considered.

REFERENCES