

# $\dot{V}O_2$ and heart rate kinetics in cycling: transitions from an elevated baseline

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Received 16 May 2000; accepted in final form 16 January 2001

**Bearden, S. E., and R. J. Moffatt.**  $\dot{V}O_2$  and heart rate kinetics in cycling: transitions from an elevated baseline. *J Appl Physiol* 90: 2081–2087, 2001.— The purpose of this study was to examine oxygen consumption ( $\dot{V}O_2$ ) and heart rate kinetics during moderate and repeated bouts of heavy square-wave cycling from an exercising baseline. Eight healthy, male volunteers performed square-wave bouts of leg ergometry above and below the gas exchange threshold separated by recovery cycling at 35%  $\dot{V}O_{2\text{ peak}}$ .  $\dot{V}O_2$  and heart rate kinetics were modeled, after removal of phase I data by use of a biphasic on-kinetics and monoexponential off-kinetics model. Fingertip capillary blood was sampled 45 s before each transition for base excess,  $\text{HCO}_3^-$  and lactate concentration, and pH. Base excess and  $\text{HCO}_3^-$  concentration were significantly lower, whereas lactate concentration and pH were not different before the second bout. The results confirm earlier reports of a smaller mean response time in the second heavy bout. This was the result of a significantly greater fast-component amplitude and smaller slow-component amplitude with invariant fast-component time constant. A role for local oxygen delivery limitation in heavy exercise transitions with unloaded but not moderate baselines is presented.

oxygen uptake kinetics; cycling

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AT THE ONSET OF AN ABRUPT INCREASE in work rate (square-wave exercise), ATP consumption rises immediately, whereas oxygen consumption ( $\dot{V}O_2$ ) increases more slowly. What keeps  $\dot{V}O_2$  from rising immediately to its steady-state level? Are the kinetics a function of metabolic inertia, or is delivery of oxygen to blame? Below the gas-exchange threshold (GET; moderate exercise), metabolic inertia is believed the limiting factor (17, 19). Controversy remains for transitions above the GET (heavy exercise), where  $\dot{V}O_2$  kinetics are more complex.

In heavy exercise, the kinetics incorporate at least two components [beyond the initial phase I component (32)] that manifest in series (4). An intrinsic property of a system where components manifest in series is that the mean response time may be altered by changes to the component time constants, amplitudes, and/or time delays. For example, the mean response time may decrease by decreasing the component time constants without changing component amplitudes, by

changing the ratio of the amplitudes with no change in the time constants, or by an earlier slow-component onset despite invariant time constants and amplitudes. Therefore, the conclusions of previous studies that were focused on the mean response time (7, 15, 23) are not easily applied to understanding the underlying physiology of such a complex system. Without a detailed analysis of the  $\dot{V}O_2$  kinetic components, the issue of metabolic inertia or oxygen delivery for heavy exercise remains unresolved.

This study was driven by the hypothesis that the rate of increase in  $\dot{V}O_2$  in heavy square-wave exercise is set by metabolic state (inertia and demand) and is not limited by the ability of the cardiovascular system to deliver oxygen in healthy adults. Therefore, it was postulated that 1) there would be no difference in the initial (fast component) time constant among moderate and repeated heavy transitions; 2) systemic acidosis would not be a necessary condition for the speeded mean response time in the second heavy bout (acidosis was previously implicated in improving perfusion and we hypothesized perfusion is not the limiting factor); and 3) cardiac output kinetics could be dissociated from the  $\dot{V}O_2$  response.

To this end, an elevated baseline ( $\sim 35\%$   $\dot{V}O_{2\text{ peak}}$ ,  $\sim 60\%$  GET) was used, which differs from the typically employed unloaded cycling baseline. The elevated baseline was used, in part, to enhance recovery (addressing *hypothesis 2*); an active recovery of this moderate intensity is optimal for speeding systemic metabolic recovery (13). Moreover, an elevated baseline may slow heart rate kinetics (21) and was used in this study for its potential to dissociate  $\dot{V}O_2$  and heart rate during the nonsteady state (addressing *hypothesis 3*). The elevated baseline was expected to raise baseline cardiac output, setting stroke volume closer to its plateau (25) and to allow more reliable inferences to cardiac output kinetics directly from the heart rate response (also addressing *hypothesis 3*). This work rate ( $\sim 35\%$   $\dot{V}O_{2\text{ peak}}$ ,  $\sim 60\%$  GET) has been shown to maximize parasympathetic withdrawal, leaving the slower, sympathetic nervous system to mediate increases in heart rate and cardiac output (33).

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## METHODS

**Subjects.** Eight healthy male volunteers experienced with cycling on a stationary ergometer gave written informed consent to participate in this study. The procedures were approved by the Florida State University Human Subjects Review Board. Subjects were  $27 \pm 3$  yr old,  $177 \pm 4$  cm tall, and  $72 \pm 8$  kg in body mass.

**Preparation.** Subjects were prohibited from alcohol and strenuous activity for 24 h and from caffeine for 15 h before arrival in the laboratory. No one reported taking dietary supplements or ergogenic aids aside from vitamin/mineral supplements. Subjects consumed a light carbohydrate meal 2–3 h before arrival in the laboratory and repeated this meal before all test days. Testing was at the same time of day,  $\pm 2$  h, for each subject. Subjects were not permitted to cycle to the laboratory and remained sedentary in the testing area for at least 30 min before each test.

**Testing.** Subjects cycled at 90 rpm on an electrically braked leg ergometer (Lode, Groningen, Netherlands) on 4 separate testing days. Ninety revolutions per minute was the approximate mean preferred cadence among the subjects and the one most often chosen on a blinded familiarization day. Cadence differences appear to alter the kinetics response only to a small degree within the typically employed range (3).

Gas exchange was measured every breath with a Parvo-medics MMS-2400 system (Consentius Technologies, Salt Lake City, UT). Total dead space of the system (mouthpiece, valve, collection tube, pneumotach, mixing chamber, and sampling tube) was 4.98 liters. A seven-point flowmeter calibration was made before each test with a 3-liter syringe (Hans Rudolph, Kansas City, MO) at rates that spanned the expected measurements. Gas calibration was made immediately before each test, with gases spanning the range of  $O_2$  and  $CO_2$  expected during data collection.

The first day was a test for  $\dot{V}O_{2\text{ peak}}$  (1 W/5 s) and continued until the cadence could not be maintained despite verbal encouragement. The GET was defined as the break in slope of the  $CO_2$  production ( $\dot{V}CO_2$ )- $\dot{V}O_2$  relationship by use of Levenberg-Marquadt estimation to identify the intersection of two lines that minimized the sum of the squared residuals. Starting values in the iteration procedures were 1.0 for the initial slope, threshold estimated visually (30), and 1.3 for the upper slope. The computer-identified GET was, in all cases, in close agreement with the visually identified  $\dot{V}CO_2$ - $\dot{V}O_2$  break.

The last three sessions were divided into one moderate (Mod) and two heavy (Hvy) exercise days, which were completed in random order. On each day, after a 5-min baseline warm-up at a  $\dot{V}O_2$  of 35% peak, subjects completed two 10-min cycling periods at an elevated work rate, separated by 10 min of baseline recovery cycling. The elevated work rate was either a  $\dot{V}O_2$  of 90% GET (Mod) or a  $\dot{V}O_2$  of 30% of the difference between GET and  $\dot{V}O_{2\text{ peak}}$  (30% $\Delta$ ; Hvy). Test sessions for a given subject were separated by  $\geq 2$  days.

The  $\dot{V}O_2$  responses for the 2 Hvy transition days were time aligned. To remove nonphysiological datum points resulting from coughing, sneezing, etc., any breaths more than four standard deviations away from the mean of the surrounding six breaths (3 before and 3 after) were deleted. The decision to remove these points was confirmed visually to ensure that only clearly nonphysiological outliers were deleted; these amounted to about six to eight breaths over the 10-min period for each test. The superimposed data set was then smoothed (rolling five-breath average).

Because the elevated baseline is different from the unloaded cycling baseline in previous studies (15, 23), we pre-

liminarily analyzed the two Mod bouts separately. There was no difference (paired *t*-test) between the two Mod bouts for any on- or off-kinetic parameter ( $P > 0.05$ ). Therefore, the two Mod bouts, completed on the same day, were time aligned and averaged to produce a single response, which enhances confidence in model fitting (22).

Before modeling, each test was examined for a steady state. Accurate and complete kinetic modeling is not assured without establishing, or rigorously estimating, a steady state for the parameter(s) to be modeled. Linear regression was applied to the final 3 min of data for each average response. The 95% confidence interval for each regression slope was examined; in all cases, the 95% confidence interval included zero. This was the basis for deciding that a steady state had been reached. To further confirm the steady state, the 9- to 10-min average  $\dot{V}O_2$  was compared with the modeled asymptote (paired *t*-test) and was not different ( $P > 0.05$ ).

As described by Whipp and colleagues (32), the phase I component (9) was removed before modeling by visually analyzing the  $\dot{V}O_2$  and respiratory exchange ratio (RER) responses for each transition. Initially,  $\dot{V}O_2$  kinetics were modeled using a monoexponential formula (Eq. 1) to compare these results with the faster mean response time (MRT) reported previously (7, 15, 23). The MRT was calculated as  $TD_1 + \tau_1$ . After a significant speeding of the overall kinetics (smaller MRT in the second bout) was found, gas exchange data were modeled using a biphasic formula with independent time delays (Eq. 2)

$$\dot{V}O_2(t) = B + A_1(1 - e^{-(t - TD_1)/\tau_1}) \quad (1)$$

$$\dot{V}O_2(t) = B + A_1(1 - e^{-(t - TD_1)/\tau_1}) + A_2(1 - e^{-(t - TD_2)/\tau_2}) \quad (2)$$

where  $\dot{V}O_2(t)$  is whole body  $\dot{V}O_2$  at time *t*, B is baseline (warm-up)  $\dot{V}O_2$  calculated as the average  $\dot{V}O_2$  over the last 2 min of warm-up,  $A_1$  and  $A_2$  are the fast and slow-component amplitudes, respectively,  $TD_1$  and  $TD_2$  are their respective time delays, and  $\tau_1$  and  $\tau_2$  are their respective time constants. Invariably, the slow component ( $A_2$ ) regressed toward zero in the Mod transitions; therefore, the Mod bouts were reanalyzed using a monoexponential formula (Eq. 1).

Each amplitude component was allowed to begin only after its time delay. Confidence in the time constant ( $\tau_1$ ) was  $2.23 \pm 0.41$  s in Hvy<sub>1</sub>,  $2.34 \pm 0.45$  s in Hvy<sub>2</sub>, and  $4.53 \pm 0.89$  s in Mod, based on formulas reported by Lamarra and colleagues (22).

Off-kinetics were initially modeled using a biphasic formula; however, the model consistently regressed to a monoexponential function of time (the time constants for the two components were not different). Therefore, the off-kinetics were modeled using a monoexponential equation

$$\dot{V}O_2(t) = EE\dot{V}O_2 - 'A_1(1 - e^{-(t - 'TD_1)/'\tau_1}) \quad (3)$$

where  $EE\dot{V}O_2$  is end-exercise  $\dot{V}O_2$  with the other parameters as described above; the prime mark (') designates these as off-kinetics parameters.

Heart rate (HR) was monitored telemetrically (Polar Electro, Woodbury, NY) and recorded online at the end of every breath (signaled from the pneumotach). The data were then superimposed, averaged, filtered, and modeled in the identical manner as for the  $\dot{V}O_2$  data. Therefore, the heavy transitions were modeled much like the method used by Engelen and colleagues (14). The justification for choosing the biphasic model is that visual analysis clearly revealed a fast phase approaching a plateau with a subsequent delayed rise. These response characteristics were particularly clear after averaging of the two responses.

Table 1. On-transition  $\dot{V}O_2$  and HR responses to Hvy and to Mod exercise transition

	B, l/min	A <sub>1</sub> , l/min	TD <sub>1</sub> , s	$\tau_1$ , s	A <sub>2</sub> , l/min	TD <sub>2</sub> , s	$\tau_2$ , s	G <sub>1</sub> , ml/W	G <sub>T</sub> , ml/W
$\dot{V}O_2$									
Mod	1.53 ± 0.22	0.71 ± 0.11†	22.9 ± 6.5	21.0 ± 5.6					9.72 ± 0.90
Hvy <sub>1</sub>	1.54 ± 0.25	1.14 ± 0.09	18.7 ± 5.3	19.5 ± 7.0	0.29 ± 0.12	118.6 ± 34.5	211.6 ± 133.3	9.17 ± 0.51	11.48 ± 1.05 <sup>b</sup>
Hvy <sub>2</sub>	1.61 ± 0.30 <sup>a,b</sup>	1.23 ± 0.13 <sup>a</sup>	20.2 ± 5.1	18.5 ± 4.1	0.16 ± 0.04 <sup>a</sup>	258.0 ± 97.6 <sup>a</sup>	82.6 ± 53.7 <sup>a</sup>	9.84 ± 0.77 <sup>a</sup>	10.94 ± 0.74 <sup>b</sup>
	B, bpm	A <sub>1</sub> , bpm	TD <sub>1</sub> , s	$\tau_1$ , s	A <sub>2</sub> , bpm	TD <sub>2</sub> , s	$\tau_2$ , s		
$HR$									
Mod	107 ± 12	24 ± 7†	13.6 ± 9.3	35.1 ± 10.4*					
Hvy <sub>1</sub>	104 ± 11	38 ± 8	10.1 ± 3.8*	32.2 ± 7.8*	21 ± 6	134.0 ± 34.1	506.7 ± 229.1		
Hvy <sub>2</sub>	112 ± 12	35 ± 7	12.9 ± 5.0*	35.0 ± 10.7*	9 ± 5 <sup>a</sup>	216.3 ± 64.8 <sup>a</sup>	233.3 ± 171.7 <sup>a</sup>		

Values are means ± SD. B, baseline; bpm, beats/min;  $\dot{V}O_2$ , oxygen uptake; HR, heart rate; G<sub>1</sub>, fast component gain for the repeated heavy (Hvy) bouts (A<sub>1</sub>/W); and G<sub>T</sub>, steady state or total gain [(A<sub>1</sub> + A<sub>2</sub>)/W in Hvy and A<sub>1</sub>/W in a single moderate (Mod) exercise transition]. Second bout  $\dot{V}O_2$  kinetics became monoexponential in *subject 6*; therefore, A<sub>2</sub>, TD<sub>2</sub>, and  $\tau_2$  are for *n* = 7 in Hvy<sub>2</sub>. HR responses were monoexponential for *subject 7*; therefore, HR A<sub>2</sub>, TD<sub>2</sub>, and  $\tau_2$  are for *n* = 7. All other data are *n* = 8. <sup>a</sup>Significantly different from Hvy<sub>1</sub>; <sup>b</sup>significantly different from Mod (*P* < 0.05). \*Significantly different from concurrent  $\dot{V}O_2$  parameter; †Mod was not compared with either Hvy bout, because the work rates were different between the Mod and Hvy bouts.

As described by Stringer and colleagues (29), cardiac output ( $\dot{Q}$ ) was estimated as  $\dot{Q} = \dot{V}O_2/[5.721 + (0.1047 \times \% \dot{V}O_{2\text{ peak}})]$ . Stroke volume (SV) was estimated as  $SV = \dot{Q}/HR$ .

Capillary blood was taken from a fingertip 45 s before each of the two abrupt increases in work rate. Blood lactate (Accusport, Indianapolis, IN), pH, base excess, and [HCO<sub>3</sub><sup>-</sup>] (AVL, Roswell, GA) were measured immediately. Blood analysis instruments were calibrated immediately before every test by use of standards that spanned the expected range of test measurements (≥1 point above and 1 point below the expected measurement).

Gains for the responses were calculated as the fast-component gain, G<sub>1</sub> = A<sub>1</sub>/ΔW, and total gain, G<sub>T</sub> = (A<sub>1</sub>+A<sub>2</sub>)/ΔW.

**Statistics.** Paired *t*-tests were used to compare the initial MRTs between the two Hvy transitions. Paired *t*-tests were used to compare A<sub>1</sub>, A<sub>2</sub>, TD<sub>2</sub>,  $\tau_2$ , 'A<sub>1</sub>, G<sub>1</sub>, base excess, [HCO<sub>3</sub><sup>-</sup>], blood lactate, and pH between the two Hvy transitions. ANOVA (randomized block design) was used to compare baseline  $\dot{V}O_2$ , baseline HR, TD<sub>1</sub>,  $\tau_1$ , G<sub>T</sub>, 'TD<sub>1</sub>, and ' $\tau_1$  among the Mod and two Hvy transitions; subjects served as blocks, the category of comparison as the fixed factor, and the measurement as the dependent variable. Tukey's post hoc comparisons were used whenever overall significance was found to identify the differing pairs. Paired *t*-tests were used to compare time delays and time constants between the  $\dot{V}O_2$  and HR responses. Pearson moment correlations were computed for each bout (Mod, Hvy<sub>1</sub>, Hvy<sub>2</sub>) between  $\dot{V}O_{2\tau_1}$  and HR  $\tau_1$ . Alpha was set at *P* = 0.05.

**RESULTS**

$\dot{V}O_{2\text{ peak}}$  and GET were 60 ± 3 ml O<sub>2</sub>·kg<sup>-1</sup>·min<sup>-1</sup> and 58 ± 7% peak, respectively. Blood lactate and pH returned to baseline before the onset of the second bout ([La<sup>-</sup>]: 1st = 1.78 ± 0.34 mM, 2nd = 2.24 ± 0.64 mM; pH: 1st = 7.38 ± 0.01, 2nd = 7.38 ± 0.02; mean ± SD). In contrast, base excess and [HCO<sub>3</sub><sup>-</sup>] were significantly lower before the onset of the second bout (base excess: 1st = 0.70 ± 1.37 mM, 2nd = -0.96 ± 1.11 mM; [HCO<sub>3</sub><sup>-</sup>]: 1st = 25.90 ± 1.28 mM, 2nd = 24.13 ± 0.89 mM; mean ± SD).

Actual work rates were baseline 35.6 ± 5.2%  $\dot{V}O_{2\text{ peak}}$  or 60.5 ± 10.2% GET, Mod asymptote 52.2 ± 5.9%  $\dot{V}O_{2\text{ peak}}$  or 89.9 ± 7.2% GET, and Hvy asymptote 69.4 ± 7.1%  $\dot{V}O_{2\text{ peak}}$  or 27.2 ± 13.2% Δ.

$\dot{V}O_2$ . Initial analysis of mean response times revealed a significant speeding of the overall kinetics in Hvy<sub>2</sub> (MRT: 1st = 55.5 ± 10.1 s, 2nd = 44.3 ± 7.7 s). The on-transition parameters are given in Table 1. *Subject 6*, for whom the first Hvy bout generated a slow-component amplitude of 92 ml O<sub>2</sub>/min, demonstrated monoexponential kinetics in the second bout (A<sub>2</sub> regressed to zero). Therefore, comparisons between the two bouts for  $\dot{V}O_2$  slow-component parameters (A<sub>2</sub>, TD<sub>2</sub>,  $\tau_2$ ) were made with seven subjects. The amplitude and gain of the fast component (A<sub>1</sub>, A<sub>1</sub>/W) were significantly larger than in the first bout, as was the overall projected asymptote for the fast component (B + A<sub>1</sub>). The time constant of the fast component was not different among Mod, Hvy<sub>1</sub>, and Hvy<sub>2</sub>. The amplitude of the slow component (A<sub>2</sub>) was significantly smaller in Hvy<sub>2</sub> than in Hvy<sub>1</sub>. The onset of the slow component (TD<sub>2</sub>) was significantly later in Hvy<sub>2</sub> and its time constant ( $\tau_2$ ) was significantly smaller than in Hvy<sub>1</sub>. The  $\dot{V}O_2$  asymptote and overall gain (G<sub>T</sub>) was not different between Hvy<sub>1</sub> and Hvy<sub>2</sub>. The two Hvy bouts for *subject 5* are shown superimposed in Fig. 1.

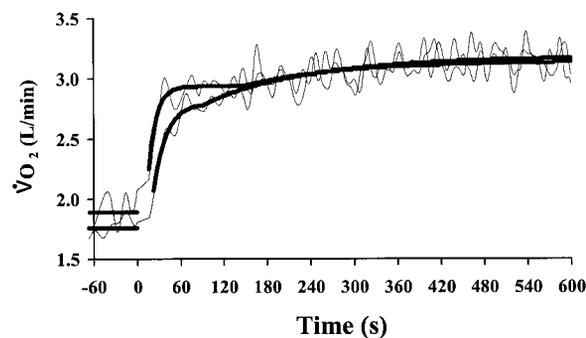


Fig. 1. Representative response to repeated heavy cycling transitions (*subject 5*). First and second transitions from 60% gas-exchange threshold (GET) to 30% of the difference between the GET and peak oxygen uptake ( $\dot{V}O_{2\text{ peak}}$ ) are superimposed. Note the greater fast-component amplitude (A<sub>1</sub>). There was no difference in the fast-component time constant ( $\tau_1$ ) or overall response asymptote between the two bouts.

Off-transition kinetic parameters are given in Table 2. There were no differences in the off-transition parameters among the Mod and Hvy bouts.

**HR.** The on-transition kinetics of the heart rate responses are shown in Table 1. For *subject 7*, the kinetics were monoexponential in both Hvy bouts. The time delay ( $TD_2$ ) for the slow component of the HR kinetics was significantly longer in Hvy<sub>2</sub> than in Hvy<sub>1</sub> and was not significantly different from  $TD_2$  for  $\dot{V}O_2$  in either bout. The initial rise in HR ( $A_1$ ) for the two Hvy bouts was not different, and its time constant was not different among the Mod and the two Hvy bouts. However, in each case, the time constant ( $\tau_1$ ) was significantly longer than for  $\dot{V}O_2$ . Furthermore, the HR and  $\dot{V}O_2$  fast-component time constants ( $\tau_1$ ) were not significantly correlated (Fig. 2). The slow component of HR kinetics had a smaller amplitude and time constant in Hvy<sub>2</sub> than in Hvy<sub>1</sub>; the time constant was not significantly different from the  $\dot{V}O_2\tau_2$  for the same bout. There was a significantly lower HR asymptote in Hvy<sub>2</sub> than in Hvy<sub>1</sub>.

Baseline HRs were not different among the Mod and two Hvy bouts, meaning that HR had recovered in the 10-min recovery period and that the starting values for the three transitions were not statistically different. Off-transition kinetic parameters for HR are given in Table 2. The amplitude of recovery in Hvy<sub>2</sub> was significantly smaller than in Hvy<sub>1</sub>, reflecting a return to the same HR from a significantly lower asymptote. The time constant for recovery in Hvy<sub>2</sub> was significantly longer than for the Mod bout, although not different from Hvy<sub>1</sub>. The Mod and Hvy<sub>2</sub> recovery time constants were significantly longer than for  $\dot{V}O_2$  in the same bouts.

Estimated  $\dot{Q}$  and SV comparisons are given in Fig. 3. Estimated stroke volume was not different across conditions; the elevated baseline appeared to serve its purpose in raising stroke volume to a plateau. Therefore, it is assumed that HR was responsible for any changes in  $\dot{Q}$  and that HR kinetics reflect  $\dot{Q}$  kinetics.

Table 2. Off-transition  $\dot{V}O_2$  and HR responses to Hvy and to Mod exercise transition

	' $TD_1$ , s	' $A_1$ , l/min	' $\tau_1$ , s
	$\dot{V}O_2$		
Mod	19.7 ± 6.5	0.71 ± 0.08 <sup>†</sup>	25.7 ± 5.9
Hvy <sub>1</sub>	14.4 ± 4.2	1.35 ± 0.10	35.6 ± 9.7
Hvy <sub>2</sub>	17.3 ± 5.2	1.33 ± 0.11	31.0 ± 6.0
	HR		
	' $TD_1$ , s	' $A_1$ , bpm	' $\tau_1$ , s
Mod	13.3 ± 5.8	19 ± 5 <sup>†</sup>	38.5 ± 9.6*
Hvy <sub>1</sub>	9.3 ± 7.4	44 ± 11	43.2 ± 12.9
Hvy <sub>2</sub>	11.6 ± 6.9	39 ± 9 <sup>a</sup>	49.1 ± 13.3 <sup>b,*</sup>

Values are means ± SD. <sup>a</sup>Significantly different from Hvy<sub>1</sub>; <sup>b</sup>significantly different from Mod ( $P < 0.05$ ). <sup>\*</sup>Significantly different from corresponding  $\dot{V}O_2$  parameter; <sup>†</sup>Mod was not compared with either Hvy bout, because the work rates were different between the Mod and Hvy bouts.

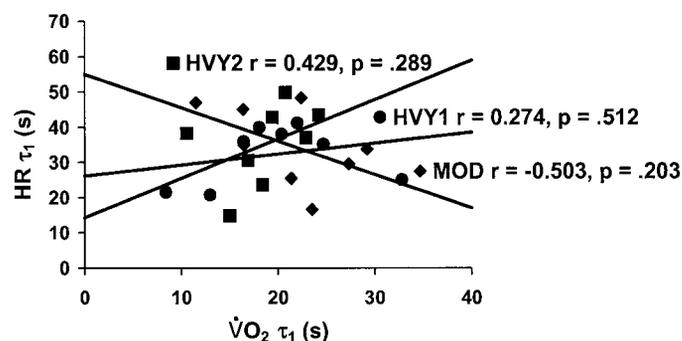


Fig. 2. Correlation plots for heart rate (HR) and  $\dot{V}O_2 \tau_1$  in moderate (Mod) and heavy (Hvy<sub>1</sub> and Hvy<sub>2</sub>) exercise transitions. Dissociation of cardiac and  $\dot{V}O_2$  kinetics emphasizes the importance of local controllers of blood flow. See text for further discussion.

## DISCUSSION

The primary finding in the present study was that a faster MRT in repeated heavy transitions is not due to a smaller fast-component time constant but to a relative shift in kinetic amplitudes (larger  $A_1$  and smaller  $A_2$ ) with the same final asymptote. It was further demonstrated that HR (and presumably  $\dot{Q}$ ) kinetics can be dissociated from  $\dot{V}O_2$  kinetics with a baseline of ~35%  $\dot{V}O_{2\text{ peak}}$ .

Burnley and colleagues (8) modeled repeated heavy transitions from a 20-W baseline and demonstrated a greater fast-component asymptote with invariant time constant (~25 s). Thus the faster MRT in repeated heavy transitions does not appear to be the result of faster initial kinetics regardless of baseline work rate. This leads to the conclusion that factors limiting  $\tau_1$  are not different in repeated bouts.

It has been demonstrated that previous leg (7, 15, 23) and previous arm (7) exercise results in a faster MRT in a subsequent bout of heavy leg ergometry. If a shift in the relative amplitudes is the cause of the faster MRT, then what causes these changes even when the previous exercise is in a different muscle group?

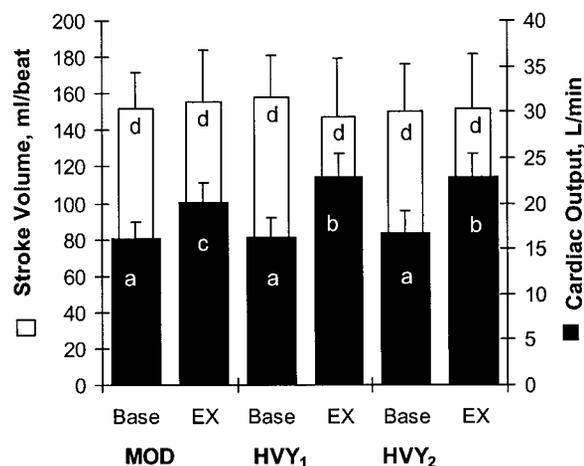


Fig. 3. Estimated stroke volume and cardiac output. Bars with the same letter are not significantly different; bars with different letters are significantly different. Base, baseline exercise value; Ex, steady-state exercise value.

*A potential mechanism for shifts in kinetic component amplitudes.* Bangsbo and colleagues (1) reported that previous heavy arm exercise caused a greater potassium loss from subsequently exercising legs than in the control (no previous arm exercise) condition and concluded that elevated potassium concentration in the leg interstitium was an important mediator of fatigue. We hypothesize that previous heavy exercise (arm or leg) disrupts the sarcolemmal electrochemical gradient through elevated extracellular potassium concentration in the leg interstitium, as reported by Bangsbo and colleagues. The resulting fatigue would demand the recruitment of additional, potentially less economic fibers to begin the subsequent bout and mediate a greater fast-component asymptote, as modeled in the present study and recently by others (8).

Oxygen demand over the pre-TD<sub>2</sub> period is no greater than the observed A<sub>1</sub> asymptote (5). Consistent with this, Grassi and colleagues (16) demonstrated that elevating pretransition O<sub>2</sub> delivery along with adenosine infusion (to induce vasodilation) in maximally stimulated mammalian muscle speeds  $\tau_1$  without altering A<sub>1</sub>. These demonstrations support the conclusion that the greater A<sub>1</sub> in Hvy<sub>2</sub> was the result of a greater oxygen demand (potentially from additionally recruited fibers), not the removal of a delivery limitation. Dispersing the same work rate over a larger motor unit pool at the onset of Hvy<sub>2</sub> would lead to less fatigue of individual myocytes, demanding a smaller additional recruitment in the slow-component period to maintain force output. This is supported by the present data showing 1) a later slow-component onset (consistent with a better ability to sustain the initial change in work rate without fatigue), 2) a smaller slow-component time constant (consistent with a reduced need to serially recruit new myocytes with developing fatigue), and 3) a smaller slow-component amplitude (consistent with a smaller net increase in motor unit recruitment). These considerations are consistent with the prevailing theory that the slow component is the result of increased motor unit recruitment (3, 6, 28, 31).

In contrast to the present study, Burnley and colleagues (8) found the overall asymptote to be lower in the second bout. This is most likely a methodological difference; Burnley and colleagues (8) used 6-min bouts resulting in termination of the exercise at a time when only ~50% of the apparent slow component had developed (before one time constant had elapsed). The 10-min bouts in the present study included >85% of the slow-component data (i.e., >2 $\tau_2$ ), and the  $\dot{V}O_2$  time slope was not different from zero over the last minutes of each bout. Short bouts may not allow for a full adjustment to the work rate and may lead to inaccurate modeling. However, it is possible that differences in the two studies exist that facilitated a truly smaller asymptote in their study (8), but these must await further investigation. Because the final asymptote was not different across bouts in the current study, it is assumed that the same final motor unit pool was recruited (or its

metabolic equivalent) and that the primary difference between the two bouts was in the partitioning of recruitment order.

An elevated baseline may speed  $\dot{V}O_2$  kinetics (11, 12), although this is not a universal conclusion (21). An intriguing finding in the present study was the similar  $\tau_1$  for moderate and heavy transitions (Table 1). In contrast,  $\tau_1$  is usually significantly slower in heavy compared with moderate transitions; note that we used a baseline of moderate exercise, whereas previous studies have used a light or unloaded baseline (8, 14, 24). Grassi and colleagues (16) showed that elevated pretransition oxygen availability could significantly speed  $\tau_1$  from ~25 to ~18 s. These values are remarkably similar to the  $\tau_1$  of ~19 s in the present study with an elevated baseline and the ~25-s  $\tau_1$  generally reported using a light or unloaded baseline (2–4, 8, 14, 24). It is possible that our elevated baseline facilitated a pretransition increase in oxygen availability for newly recruited motor units. A mechanism for this is illustrated by the elegant work of Segal and colleagues (for reviews, see Refs. 26 and 27), which has demonstrated local vasoactive metabolites may activate upstream vasodilation. Due to the structure of the capillary bed, this vasodilation increases oxygen availability to both active and inactive fibers. Should the inactive fibers be required for the subsequent increase in work rate, they would then have sufficient oxygen to rapidly accelerate oxidative metabolism, potentially speeding  $\tau_1$ . However, there is variability among laboratories and subjects larger than the ~18- to ~25-s differences suggested here. Thus it is emphasized that these ideas are speculation only and deserve further research.

*HR kinetics.* The present results are consistent with the ability to slow HR kinetics by using an elevated baseline work rate (20, 21). The elevated baseline effectively removes the more rapid influence of the parasympathetic system (33).

The complexity of the HR response to heavy square-wave transitions has previously been observed (14, 18) and modeled into fast and slow components as in the present study (14). Engelen and colleagues (14) showed that HR $\tau_1$  was slower than  $\dot{V}O_2\tau_1$  by ~15 s, although no statistical comparison was given. Significant dissociation of HR $\tau_1$  (and presumably cardiac output  $\tau_1$ ) and  $\dot{V}O_2\tau_1$  in the present study emphasizes the importance of local blood flow control in matching oxygen demand and delivery during the nonsteady state.

*Off-kinetics.* Because the off-kinetics reduced to a monoexponential function, the two phases of the on-kinetics (the fast and slow phases) appear to have equivalent and coincident recovery profiles, at least to the extent that mathematical modeling could detect a difference in these subjects. The off-kinetics time delays and time constants were not different among the Mod and Hvy bouts.

A recent study with the use of unloaded pedaling as a baseline and designed to investigate this issue directly has also concluded that the off-kinetics of  $\dot{V}O_2$

are “independent of the magnitude of the contribution to the slow phase from the on-transient kinetics” (10). These investigators found the off-kinetics well fit by including a slow phase, beginning with the fast phase, that had a small amplitude and large time constant. We did not find a slow phase component to the off-kinetics. The difference may be due to either the different baselines or the longer recovery time in the study of Cunningham and colleagues (10) (15 min vs. our 10 min). Because our elevated baseline is known to speed recovery (13), it is more likely that baseline work rate was the culprit. Thus the slow phase of recovery may be representative of metabolic processes still demanded at our moderate intensity and therefore not a part of the recovery profile in this study. Nevertheless, the two studies are in agreement that whatever causes the slow component appears to begin recovery immediately upon cessation of the exercise, recovers more rapidly than it developed and does so in parallel with the fast component of recovery.

In conclusion, repeated bouts of heavy exercise in this study resulted in a smaller MRT, mediated by an increase in the fast-component amplitude, similar fast-component time constant, and similar final steady state; systemic acidosis was not necessary for the faster MRT. HR (and presumably  $\dot{Q}$ ) kinetics may be slowed and dissociated from  $\dot{V}O_2$  kinetics with a baseline of mild exercise. Most important, these data, in conjunction with previous research, are consistent with a role for local oxygen delivery limitation at the onset of heavy exercise transitions, which is not lifted by repeated bouts but may be reduced using a preparatory baseline of moderate exercise. A role for membrane potential, motor unit recruitment patterns, and pretransition vasodilation was proposed to underlie the  $\dot{V}O_2$  kinetics.

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