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$\dot{V}O_2$ kinetics and the O_2 deficit in heavy exercise

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Bearden, S. E., and R. J. Moffatt. $\dot{V}O_2$ kinetics and the O_2 deficit in heavy exercise. *J Appl Physiol* 88: 1407–1412, 2000.—The purpose of this study was to examine a new method for calculating the O_2 deficit that considered the O_2 uptake ($\dot{V}O_2$) kinetics during exercise as two separate phases in light of previous research in which it was shown that the traditional O_2 deficit calculation overestimated the recovery O_2 consumption (ROC). Eight subjects completed exercise transitions between unloaded cycling and 25% (heavy, H) or 50% (very heavy, VH) of the difference between the lactic acid threshold (LAT) and peak $\dot{V}O_2$ for 8 min. The O_2 deficit, calculated in the traditional manner, was significantly greater than the measured ROC for both above-LAT exercises: 4.03 ± 1.01 vs. 2.63 ± 0.80 (SD) liters for VH and 2.36 ± 0.91 vs. 1.74 ± 0.63 liters for H for the O_2 deficit vs. ROC ($P < 0.05$). When the kinetics were viewed as two separate components with independent onsets, the calculated O_2 deficit (2.89 ± 0.79 and 1.71 ± 0.70 liters for VH and H, respectively) was not different from the measured ROC ($P < 0.05$). Subjects also performed the same work rate for only 3 min. These data, from bouts terminated before the slow component could contribute appreciably to the overall $\dot{V}O_2$ response, show that the O_2 requirement during the transition is less than the final steady state for the work rate, as evidenced by symmetry between the O_2 deficit and ROC. This new method of calculating the O_2 deficit more closely reflects the expected O_2 deficit-ROC relationship (i.e., ROC ≥ O_2 deficit). Therefore, estimation of the O_2 deficit during heavy exercise transitions should consider the slow component of $\dot{V}O_2$ as an additional deficit component with delayed onset.

recovery oxygen consumption; lactic acid threshold; square wave; steady state

OXYGEN UPTAKE ($\dot{V}O_2$) rises monoexponentially to its new steady state with an amplitude of 9–10 ml $O_2 \cdot W^{-1} \cdot min^{-1}$ for work rate increments below the lactic acid threshold (LAT). For work rate transitions above LAT, an additional component (referred to as the slow component) is superimposed on the initial monoexponential function, which raises the final $\dot{V}O_2$ cost above 10 ml $O_2 \cdot W^{-1} \cdot min^{-1}$ (Fig. 1). Mathematical modeling has shown that the slow component begins 90–150 s after the onset of the transition (1, 11). However, not all researchers agree with the concept of a delayed onset (9); there is still debate over whether the two phases are physiologically best described with common or independent time delays. If the slow component is a delayed O_2 demand, then it has implications for calculation of the O_2 deficit.

For moderate work rates (i.e., below LAT), the O_2 deficit is equal to or less than the recovery O_2 consumption (ROC) (7, 11, 13). However, asymmetry between the on- and off-transition phases has led to the observation that the O_2 deficit overpredicts the ROC (11, 13) for heavy exercise (i.e., above LAT). Therefore, heavy exercise appears quantitatively and qualitatively more complex.

Accurate estimation of the O_2 deficit requires determination of baseline $\dot{V}O_2$ and of the O_2 demand for the exercise. Traditionally, the end-exercise steady-state $\dot{V}O_2$ was assumed to be the O_2 demand throughout the exercise. This is based on the belief that the energy demand for completing a task, including motor unit recruitment, does not vary during the transition to the new steady state. The determination of a second, slow component to the O_2 kinetics demands a reevaluation of these assumptions and raises questions about the relationship between the O_2 deficit and ROC.

The physiological bases of the O_2 deficit-ROC relationship are still unclear. The ROC is the excess O_2 consumed above baseline during the recovery period and is related to the O_2 deficit primarily by a restoration of tissue O_2 saturation (myoglobin and venous P_{O_2}) and resynthesis of ATP and phosphocreatine (PCr). However, the ROC is not a direct reflection of the O_2 deficit, because it also includes factors such as lactate metabolism and the metabolic demand of elevated cardiac output, ventilation, catecholamines, and temperature (6).

The purpose of this study was twofold: *1*) to test a new model for the calculation of the O_2 deficit above LAT that includes separate deficit phases corresponding to the biphasic $\dot{V}O_2$ kinetics and *2*) to test an implication of the traditional O_2 deficit model, namely that the ROC for 3 min of heavy exercise should be equivalent to the deficit calculated using the steady-state $\dot{V}O_2$ for a long bout of the same intensity (final exercise steady state). This means that the ROC would be larger than the deficit calculated using the observed kinetics projection for the 3-min bout. Our model predicts that the O_2 demand for the fast phase of the transition is its projected asymptote and not the final steady-state $\dot{V}O_2$. We tested this discrepancy in model predictions using 3- and 8-min cycling bouts at the same intensity.

METHODS

Subjects

Eight active, nonsmoking volunteers [7 men and 1 woman (*subject 2*)] took part in the study after giving informed

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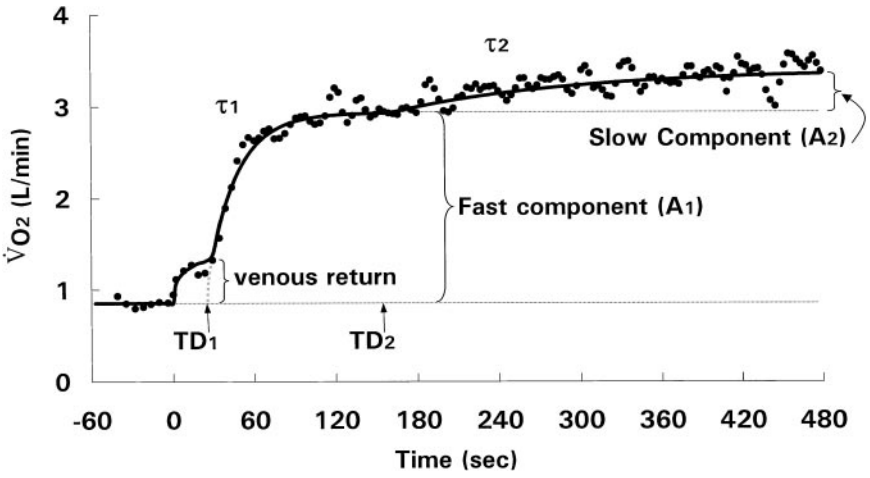

Fig. 1. Three-breath rolling average O_2 uptake ($\dot{V}O_2$) and exponential *model 3* fit for a transition from unloaded cycling to above lactic acid threshold (LAT) during an 8-min bout of heavy exercise (H8). A_1 and A_2, amplitudes of fast and slow components, respectively; TD_1 and TD_2, time delays for onset of fast and slow components, respectively; τ_1 and τ_2, time constants for fast and slow components after their time delays, respectively.

consent and completing a medical history questionnaire. The University Human Subjects Review Board approved the procedures. Subjects were tested on 3 separate days. On all testing days, subjects arrived at the laboratory 6 h after their last meal and had been instructed not to consume alcohol or caffeine for 12 h before arrival and not to engage in strenuous exercise for 24 h before arrival. Compliance with these guidelines was checked by questionnaire on arrival at the laboratory each day. There was 100% compliance.

Exercise Testing

On the 1st day, subjects completed an incremental exercise test (20 W/min, 80 rpm) on a cycle ergometer (Monark 868) until they could no longer maintain the pedal cadence for 15 s, despite verbal encouragement. Gas exchange variables were measured every breath with a Parvomedics MMS-2400 system. Total dead space of the system (mouthpiece, valve, collection tube, pneumotach, mixing chamber, and sampling tube) was 4.98 liters. Because mixing chamber systems do not allow examination of the fine details of gas exchange kinetics during the rapid early adjustment phase, the venous return component was not modeled in this study.

The system was calibrated with known gases spanning the expected range of O_2 and CO_2 in the expirate immediately before every test. A 15-point flowmeter calibration took place before every pair of tests with use of a 3-liter syringe (Hans Rudolph). LAT was estimated from gas exchange with use of the ventilatory equivalent and modified V-slope methods (2, 14, 15). The threshold determined by these two methods was not significantly different. Peak $\dot{V}O_2$ ($\dot{V}O_{2\,peak}$) was taken as the highest 15-s average achieved during the test.

Testing *sessions 2* and *3* began ≥48 h after the incremental exercise test. Each session consisted of two randomized cycling bouts, one short (3 min) and one long (8 min), on a basket-loaded ergometer (Monark 824E), which allows instantaneous application of the resistance. The 8-min bout length was chosen, because previous reports of square-wave transitions to these intensities have suggested that 6 min may not be long enough to attain a steady state. The 3-min bout length was chosen, because 3 min should be sufficient to develop the fast component without considerable contribution of the slow component if the slow component is of delayed onset. Furthermore, the 3-min bout length would allow use of the subsequent ROC as a marker of the O_2 demand during the early phase of the transition.

The short bouts (3-min: heavy, H3 and very heavy, VH3) began with 4 min of unloaded cycling (80 rpm) followed by an immediate transition to the work rate for 3 min and return to unloaded cycling for another 8 min. The long bouts (8-min: H8 and VH8) also began with 4 min of unloaded cycling (80 rpm) but were followed by 8 min at the work rate and return to unloaded cycling for 15 min. The recovery periods were sufficient for all subjects to return to baseline $\dot{V}O_2$. Subjects were unaware of which bout they would be completing and did not know when the work rate transitions were coming.

The work rates were chosen to elicit 25% (H) and 50% (VH) of the difference between LAT and $\dot{V}O_{2\,peak}$. Work rates were assigned randomly each day, but on a given day, both tests were at the same work rate. Bouts were separated by ≥1 h.

Data Modeling

Data were modeled for each work rate transition for each subject by nonlinear regression with minimization of the sum of squared residuals as the primary goal (SPSS 8.0 Professional Statistics package). The first 25 s were always removed from the analysis to ensure that the early venous return component (3, 17) did not influence the results. Iterations continued until successive repetitions reduced the sum of squared residuals by $<10^{-8}$.

On transition. The long bouts (H8 and VH8) were fit with three models: *model 1*, a single monoexponential function with time delay

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

model 2, a double monoexponential function with common time delay

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

where $TD_1 = TD_2$, and *model 3*, a double monoexponential function with independent time delays

$$\dot{V}O_2(t) = B\dot{V}O_2 + A_1[1 - e^{-(t-TD_1)/\tau_1}] + A_2[1 - e^{-(t-TD_2)/\tau_2}] \quad (3)$$

where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at any *time t*, $B\dot{V}O_2$ is baseline $\dot{V}O_2$ (the average $\dot{V}O_2$ for the last 30 s of the 4-min unloaded warm-up), A_1 and A_2 are $\dot{V}O_2$ amplitudes for the fast and slow components, respectively, TD_1 and TD_2 are time delays for the fast and slow components, respectively, and τ_1 and τ_2 are time constants for the fast and slow components after their time delays, respectively. For *Eq. 3*, the statistical model was constrained with a conditional term that forced the slow component $\{A_2[1 - e^{-(t-TD_2)/\tau_2}]\}$ to be included only when $t \geq TD_2$. This conditional statement is important, because without it the model allows the slow component to exert influence

on the predicted $\dot{V}O_2$ at earlier time points, i.e., before the component actually begins for this model.

The short bouts (H3 and VH3) were fit with a single monoexponential function (*Eq. 1*).

Off transition. Two models were applied to the off transitions: *model* 1_{off}, a single monoexponential function with time delay

$$\dot{V}O_2(t) = EE\dot{V}O_2 - A_1[1 - e^{-(t - TD_1)/\tau_1}] \quad (4)$$

and *model* 2_{off}, a double monoexponential function with common time delay

$$\dot{V}O_2(t) = EE\dot{V}O_2 - \{A_1[1 - e^{-(t - TD_1)/\tau_1}] + A_2[1 - e^{-(t - TD_2)/\tau_2}]\} \quad (5)$$

where $TD_1 = TD_2$ and $EE\dot{V}O_2$ is exercise $\dot{V}O_2$ and is equal to the model $\dot{V}O_2$ at the end of exercise minus $B\dot{V}O_2$. Different time delays were not explored in the recovery response, because the slow and fast components are present at the end of exercise and there is no reason to think that their recoveries would not begin immediately.

O_2 deficit. Model 2 fit the data significantly better than *model 1* ($P < 0.001$). Additionally, *model 3* fit the data significantly better than *model 2* ($P = 0.017$), which is in agreement with previous research (1, 11). *Model 2* constrains the second time delay (TD_2), whereas *model 3* is free to fit the data without this constraint. This means that *model 3* could result in equal time delays if this was the optimal solution as defined by the nonlinear regression goal of minimizing the sum of the squared residuals. *Model 3*, even when the starting values in the iterative estimation algorithm for the time delays were the same, did not, for any subject on any test, return a solution where the time delays were <66 s apart. Therefore, the constraints of equal time delays forced *model 2* to find a locally optimal solution that was not the globally optimal solution. Accordingly, O_2 deficit calculations were based on *model 3*.

The O_2 deficit is traditionally calculated (O_2def_{Trad}) as the difference between the O_2 that would have been consumed if a steady state had been attained immediately at the onset of exercise (Fig. 2*D*) and that consumed during the exercise period (definite integral of *Eq. 3*)

$$O_2def_{Trad} = 8(B\dot{V}O_2 + A_1 + A_2) - \int_0^8 Eq.\ 3\ dt \quad (6)$$

Our new model (O_2def_{New}) is similar to the sum of two calculated deficits for work rate increases across LAT. There is a separate deficit for the fast and slow components of *model 3* (Fig. 2*B*). This calculation was made mathematically by subtracting a volume equal to $TD_2 \times A_2$ (gray area in Fig. 2*B*) from the traditional calculation

$$O_2def_{New} = O_2def_{Trad} - (A_2 \times TD_2) \quad (7)$$

Care was taken to combine the calculated time delay and time constant for each component so as to take the model through the origin. If the definite integral of *Eq. 3* is calculated without this consideration, then the time period before TD_1 will appear as a negative $\dot{V}O_2$ and erroneously reduce the calculated amount of O_2 consumed, overestimating the O_2 deficit. Likewise, for *Eqs. 6* and *7*, the slow component $\{A_2[1 - e^{-(t - TD_2)/\tau_2}]\}$ was not included in the integration procedures until its time delay had been reached. For the short bouts, the same considerations were made, and the integral of *Eq. 1* was used.

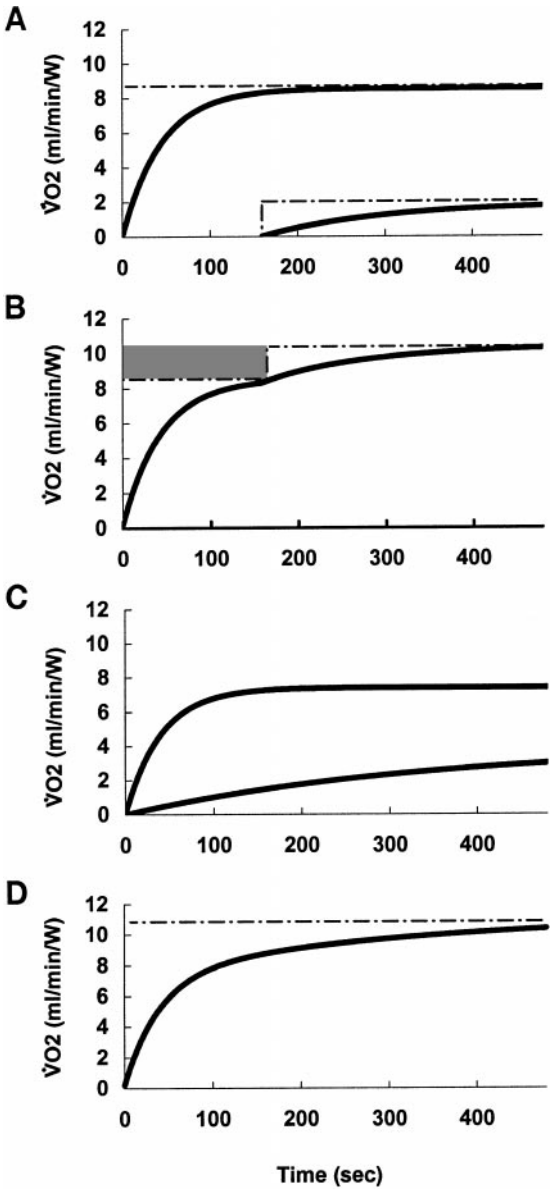

Fig. 2. *A*: 2-compartment model with delayed-onset slow component and respective O_2 deficits for transitions to work rates above LAT. *B*: superimposition of *A* and new model for O_2 deficit; gray area, hypothesized overpredicted amount from traditional calculation. *C*: 2-component model with common time delay. *D*: superimposition of *C* and traditional concept of O_2 deficit, which led to overestimation of recovery O_2 consumption.

ROC. For the 8-min bouts, no significant difference was observed between *models* 1_{off} and 2_{off} ($P = 0.96$). For the 3-min bouts, A_1 and A_2 became interchangeable, so that the regression solution would become any suggested value for either amplitude so long as the sum was equal to the overall amplitude. The result was a sum of squared residuals identical to that for the monoexponential fit. This means that the off transition for these data was monoexponential. Therefore, the simpler model was used, and the ROC was calculated by integration of *Eq. 4* with considerations for the time delay and time constant similar to those for calculation of the deficit

$$ROC = \int_{EE}^{ER} EE\dot{V}O_2 - A_1[1 - e^{-t/(TD_1 + \tau_1)}]\ dt \quad (8)$$

Table 1. *Model parameters for on transition*

	$B\dot{V}O_2$, liters	A_1, liters	TD_1, s	τ_1, s	A_2, liters	TD_2, s	τ_2, s
				Model 1			
VH8	0.81 ± 0.11	1.83 ± 0.42	7.60 ± 9.76	58.81 ± 23.35			
H8	0.82 ± 0.12	1.53 ± 0.43	13.44 ± 7.11	41.37 ± 11.64			
				Model 2			
VH8	0.81 ± 0.11	1.36 ± 0.34	22.68 ± 3.68	20.11 ± 6.28	1.05 ± 0.43	22.68 ± 3.68	808.85 ± 760.85
H8	0.82 ± 0.12	1.20 ± 0.29	22.81 ± 2.93	19.35 ± 3.23	0.62 ± 0.25	22.81 ± 2.93	598.32 ± 791.94
				Model 3			
VH8	0.81 ± 0.11	1.53 ± 0.34	22.73 ± 3.59	22.11 ± 3.29	0.54 ± 0.18	135.76 ± 43.91	296.30 ± 122.49
H8	0.82 ± 0.12	1.37 ± 0.38	23.16 ± 4.41	21.08 ± 5.14	0.25 ± 0.11	163.06 ± 41.17	120.03 ± 42.36
				3-min Bout			
VH3	0.81 ± 0.15	1.66 ± 0.37	21.83 ± 7.00	25.68 ± 6.66			
H3	0.81 ± 0.10	1.45 ± 0.41	18.36 ± 5.92	29.83 ± 6.07			

Values are means ± SD. VH8, 8-min bout of very-heavy-intensity exercise; H8, 8-min bout of heavy-intensity exercise; $B\dot{V}O_2$, baseline O_2 uptake ($\dot{V}O_2$); A_1 and A_2, $\dot{V}O_2$ amplitudes for fast and slow components, respectively; TD_1 and TD_2, time delays for fast and slow components, respectively; τ_1 and τ_2, time constants for fast and slow components, respectively. $TD_1 = TD_2$ for *model 2* by definition of a common time delay in model (see *Eq. 2*).

where *t* is time, EE is end exercise, ER is end recovery, $EE\dot{V}O_2$ is end-exercise $\dot{V}O_2$, and A_1 is the amplitude of recovery $\dot{V}O_2$ with time constant (τ_1) after a time delay (TD_1).

Eight subjects are included in the data for the VH bouts, and seven are included for the H bouts because of complications in gas collection during the H8 bout for *subject 4*.

Statistical Analysis

Within-subjects ANOVA was used for all group comparisons, with a randomized block design, on commercially available computer software (SPSS version 8.0). Tukey's honestly significant difference test was used whenever overall significance was found to determine the location of those differences. Models were compared by *F* test by using the sum of squared residuals as the criterion measure. The α was set equal to 0.05 for all analyses before data collection.

RESULTS

Subjects' age, height, mass, $\dot{V}O_{2peak}$, and LAT (means ± SD) were 27.1 ± 5.3 yr, 177.7 ± 7.0 cm, 79.4 ± 12.7 kg, 49.2 ± 6.5 ml·kg^{-1}·min^{-1}, and 47.8 ± 6.2% $\dot{V}O_{2peak}$, respectively. Model parameters for the on and off transitions can be found in Tables 1 and 2, respectively. Asymptotic $\dot{V}O_2$ projections for each work rate were 23.4 ± 3.6 and 53.6 ± 17.0 (SD) %Δ for the H8 and VH8 bouts, respectively. The O_2 deficit calculated by the traditional method for the 8-min bouts (H8 and VH8) resulted in a significant overestimation of the subsequent ROC ($P = 0.006$ for H8 and $P < 0.001$ for VH8; Table 3). Consideration of the O_2 kinetics as two separate components, each with an independent starting time, asymptotic projection, and intrinsic O_2 deficit, eliminated this overestimation; i.e., the O_2 deficit and ROC were not different when the kinetics were considered as two separate components with separate time delays (Table 3, Fig. 2*B*).

The O_2 deficit and ROC were not different in the H3 work rate, as our new model predicts. In contrast, the ROC was significantly larger than the O_2 deficit for the VH3 work rate when the 3-min projected asymptote was used as the O_2 demand. However, using the higher steady-state O_2 requirement from the VH8 bout as the initial O_2 requirement for this short bout resulted in overprediction of the observed ROC. Taking into account the small amount of slow component that had developed by the 3rd min (as determined from modeling the 8-min bout of the same intensity) restored the relationship predicted by our model (Fig. 3, Table 3).

Table 2. *Model parameters for off transition*

	$EE\dot{V}O_2$, liters	A_1, liters	TD_1, s	τ_1, s	A_2, liters	TD_2, s	τ_2, s
				Model 1$_{off}$			
VH8	1.90 ± 0.43	1.84 ± 0.42	16.15 ± 4.43	35.62 ± 5.32			
H8	1.59 ± 0.45	1.57 ± 0.44	13.33 ± 7.47	38.77 ± 6.09			
				Model 2$_{off}$			
VH8	1.90 ± 0.43	1.61 ± 0.61	19.29 ± 3.93	28.29 ± 4.73	0.33 ± 0.42	19.29 ± 3.93	501.36 ± 818.12
H8	1.59 ± 0.42	1.39 ± 0.47	13.84 ± 7.05	36.64 ± 11.12	0.23 ± 0.08	13.84 ± 7.05	404.95 ± 435.13
				3-min Bout			
VH3	1.65 ± 0.37	1.63 ± 0.36	14.77 ± 6.82	34.62 ± 7.08			
H3	1.47 ± 0.51	1.43 ± 0.38	15.68 ± 7.21	34.67 ± 8.18			

Values are means ± SD. $EE\dot{V}O_2$, exercise $\dot{V}O_2$ (i.e., $\dot{V}O_2$ at end of exercise − baseline $\dot{V}O_2$). $TD_1 = TD_2$ in *model 2$_{off}$* by definition of model (see *Eq. 5*).

Table 3. *O_2 deficit and ROC measurements*

	O_2 Deficit, liters		
	Traditional method	New model	ROC, liters
VH8	4.03 ± 1.01*	2.89 ± 0.79	2.63 ± 0.80
H8	2.36 ± 0.91*	1.71 ± 0.70	1.74 ± 0.63

	O_2 Deficit, liters			
	8-min Asymptote	3-min Asymptote	New model	ROC, liters
VH3	1.85 ± 0.54*	1.22 ± 0.30*	1.51 ± 0.51	1.55 ± 0.34
H3	2.85 ± 0.55*	1.11 ± 0.23	1.09 ± 0.24	1.25 ± 0.60

Values are means ± SD. See Fig. 3 for 8-min asymptote (all areas), 3-min asymptote (solid area), and new model (solid + hatched areas). *Significantly different from recovery O_2 consumption (ROC), $P < 0.05$.

A steady state was reached for all subjects in the lower of the two intensities. Steady state was defined as reaching a $\dot{V}O_2 \leq 1$ ml $O_2 \cdot$kg body mass$^{-1} \cdot$min^{-1} of the model asymptote, because this value is within the error of the measurement system. Thus we have confidence that the model was a good estimation of the final O_2 demand. Only *subjects 1*, *7*, and *8* reached a steady state by 8 min at the higher intensity. However, the asymptotic projection for all subjects was below $\dot{V}O_{2peak}$. During the recovery phase, all subjects returned to baseline $\dot{V}O_2$ within the test time.

DISCUSSION

Our data suggest that calculation of the O_2 deficit in the traditional manner (i.e., the difference between O_2 consumed and that consumed if the final projected steady-state $\dot{V}O_2$ had been reached immediately) is not valid for above-LAT exercise. This finding is accurate under the assumption that the O_2 deficit should not be larger than the ROC. If this assumption is true, then the more accurate calculation of the O_2 deficit above the LAT should consider two distinct components of $\dot{V}O_2$, each with its own deficit (Fig. 2*B*). These data also support the contention that $\dot{V}O_2$ above LAT is composed of two phases, including one that does not begin until ~2–3 min after the onset of the work rate transition. The slow component amplitude (A_2) contributed 15% on average to the overall $\dot{V}O_2$ for a given work rate (~10% at small %Δ and 20% at the higher %Δ).

The 3-min bouts gave us an opportunity to use the ROC as an upper-limit indicator of the expected deficit. If the O_2 demand during the first few minutes of the transition is the final steady-state $\dot{V}O_2$, then the sum of the areas in Fig. 3 should be equal to or less than the ROC. For both intensities, this deficit was significantly greater than the ROC ($P = 0.0005$ for H3 and $P = 0.001$ for VH3; Table 3). If it is assumed that the deficit is always equal to or less than the ROC, this suggests that the O_2 demand during the first minutes of the transition is actually less than the final steady-state $\dot{V}O_2$. With only the monoexponential projection of the 3-min data (solid area in Fig. 3), there was no difference between the deficit and ROC at 25%Δ; however, the deficit at 50%Δ was significantly less than the following ROC ($P = 0.005$; Table 3 and Fig. 3). With use of our new model, which included the small portion of the deficit that had developed during the end of the 3-min bouts (solid and hatched areas of Fig. 3), the O_2 deficit and ROC measurements were not different at either intensity.

Symmetry was observed between the O_2 deficit and ROC for the H3 bout without correction for any partially developed slow component. This is most likely due to a combination of a slightly larger TD_2 (i.e., later onset of the slow component) and a significantly smaller A_2 during these bouts. This would not have contributed enough to the O_2 demand before the end of exercise to enlarge the ROC, as it did at the heavier work rate.

Calculation of the O_2 deficit requires accurate determination of resting (or baseline) $\dot{V}O_2$ and a reliable measurement or estimation of the O_2 demand for the exercise. Traditionally, the final steady-state $\dot{V}O_2$ has been interpreted as the O_2 requirement for the exercise, and it was assumed that this demand was constant

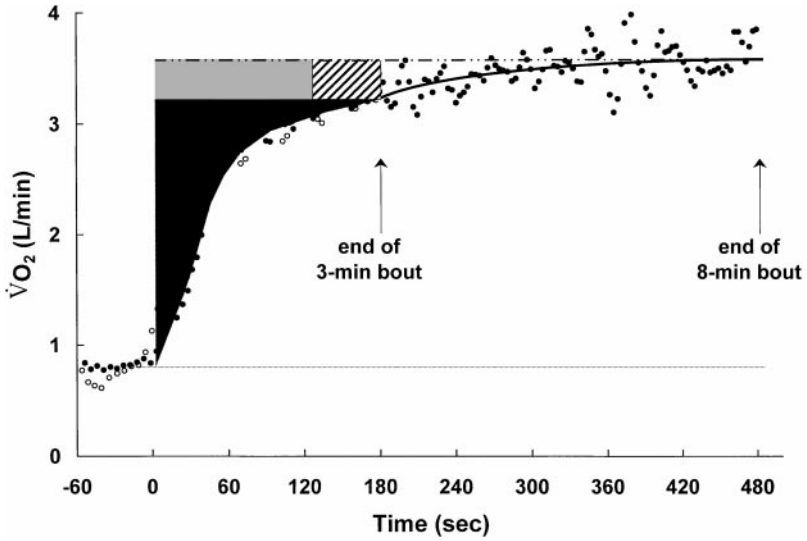

Fig. 3. Response for 8-min bout of very-heavy-intensity exercise (VH8,●) and 3-min bout of very-heavy-intensity exercise (VH3, ○) in same subject. Calculating O_2 deficit for 3-min bout from its projection (solid area) underpredicted recovery O_2 consumption in VH3 bouts. New, 2-compartment model (hatched area) accounted for this difference and resulted in an O_2 deficit equivalent to recovery O_2 consumption. All subjects recovered to baseline $\dot{V}O_2$ within test time on all tests. See Table 3 and DISCUSSION for more details.

throughout the exercise. The latter assumption is based on the idea that the energy demand for completing a task does not vary during the transition to the new steady state. Our data, in concert with others (11, 13), question the validity of this assumption for work rate transitions above the LAT.

Two possibilities exist for the delayed onset of the slow component: *1*) it is an O_2 requirement at the onset of the transition and is late to develop, or *2*) it is an O_2 demand that does not begin until later in the exercise transition. Our data suggest that the slow component is a delayed-onset O_2 requirement. This means that one might consider the transition to heavy exercise as two separate transitions: one immediate and one delayed (Fig. 2*A*). Although this is almost certainly an oversimplified view of the complex kinetics, it should provide a basis for understanding the cause of the slow component and its implications for exercise testing and the O_2 deficit-ROC relationship.

The mechanism(s) involved in developing the slow component is not well understood. However, Poole et al. (12) demonstrated that the exercising skeletal muscle is its likely origin, with 86% of the slow component attributed to the cycling legs in their study. In light of this discovery, the present findings are supported by recent ^{31}P-NMR research, which points to a delayed high-energy phosphate demand that correlates with a drop in intracellular pH ~2–3 min after the heavy work rate transition (10, 16). Whipp et al. (16) recently described a method of simultaneous quadriceps ^{31}P-NMR and pulmonary $\dot{V}O_2$ measurements during heavy knee extension exercise. Examination of Fig. 5 presented in their study suggests that PCr concentration may have slow component characteristics that coincide temporally with the $\dot{V}O_2$ kinetics (16). In support of this, one intriguing finding reported in the literature is the time course of intracellular pH and PCr concentration changes during forearm exercise in humans (10). At ~150 s there appeared to be an additional drop in PCr concentration to a new, lower steady-state level during heavy exercise. Calculations of intracellular H^+ concentration during the same exercise showed no change from resting values until the same time point, ~150 s. Using ^{31}P-NMR, Hogan et al. (8) recently reported a tight coupling between H^+ concentration and fatigue in human ankle plantar flexors. It is likely that, for work rates above LAT, a drop in intracellular and/or local extracellular pH causes a reduction in power output, demanding the recruitment of an additional pool of less economical motor units (4, 5). The recruitment of an additional motor unit pool would raise the O_2 demand for the work rate, resulting in the biphasic O_2 demand and two-compartment O_2 deficit supported by the present study.

Conclusion

Our data support the hypothesis that an additional O_2 demand begins some time after the onset of the work rate transition for work rates above LAT. Thus the O_2 demand for the exercise transition does not appear to be constant over the transition period but seems to be biphasic in nature. The previously described disparity in the O_2 deficit-ROC relationship during exercise above LAT may be rectified by using a model that considers $\dot{V}O_2$ kinetics above LAT as two separate components with corresponding O_2 deficits.

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