Dynamic Responses of Oxygen Uptake at the Onset and End of Moderate and Heavy Exercise in Trained Subjects*

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Catalogue Data

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Abstract/Résumé
Inconsistencies about dynamic asymmetry between the on- and off-transient responses in $\dot{V}O_2$ are found in the literature. Therefore the purpose of this study was to examine $\dot{V}O_2$ on- and off-transients during moderate- and heavy-intensity cycling exercise in trained subjects. Ten men underwent an initial incremental test for the estimation of ventilatory threshold (VT) and, on different days, two bouts of square-wave exercise at moderate ($<VT$) and heavy ($>VT$) intensities. $\dot{V}O_2$ kinetics in exercise and recovery were better described by a single exponential model ($<VT$), or by a double exponential with two time delays ($>VT$). For moderate exercise, we found a symmetry of $\dot{V}O_2$ kinetics between the on- and off-transients (i.e., fundamental component), consistent with a system manifesting linear control dynamics. For heavy exercise, a slow component superimposed on the fundamental phase was expressed in both the exercise and recovery, with similar parameter estimates. But the

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on-transient values of the time constant were appreciably faster than the associated off-transient, and independent of the work rate imposed (<VT and >VT). Our results do not support a dynamically linear system model of $\dot{V}O_2$ during cycling exercise in the heavy-intensity domain.

Des contradictions sur l’asymétrie entre les réponses dynamiques de la cinétique de $\dot{V}O_2$ on- et off- sont présentes dans la littérature. Le but de cette étude était donc d’examiner la cinétique de $\dot{V}O_2$ on- et off- lors d’exercices de pédalage à intensité constante modérée et difficile. Dix hommes ont réalisé dans l’ordre, un test progressif permettant l’évaluation du seuil ventilatoire (VT), puis deux répétitions d’exercice à intensité constante modérée (<VT) et difficile (>VT). Les cinétiques de $\dot{V}O_2$ on- et off- étaient caractérisées par un modèle exponentiel simple (<VT) ou par un modèle à double exponentiel avec des temps de retards indépendants (>VT). Pour l’exercice modéré, nous avons trouvé une symétrie de la cinétique de $\dot{V}O_2$ entre les transitions on- et off- (i.e., composante fondamentale), compatible avec un système dynamique linéaire. Pour l’exercice difficile, une composante lente équivalente en sus de la composante fondamentale était présente lors des périodes d’exercice et de récupération. Cependant les valeurs de la constante de temps de la transition on- étaient sensiblement plus rapides que celles de la transition off-, et étaient indépendantes de la charge de travail imposé (<VT et >VT). Nos résultats ne soutiennent pas un système de contrôle dynamique linéaire pour $\dot{V}O_2$ lors d’exercice de pédalage à intensité difficile.

Introduction

Compared to low or moderate constant-load exercise, the oxygen uptake ($\dot{V}O_2$) response above the lactate threshold is more complex and is characterized by an additional rise in $\dot{V}O_2$ that has been termed the $\dot{V}O_2$ slow component (Barstow and Molé, 1991; Paterson and Whipp, 1991; Whipp, 1994). To date, the underlying mechanism(s) of the $\dot{V}O_2$ slow component remain to be established. $\dot{V}O_2$ kinetics during the recovery period is often neglected by exercise physiologists. Nevertheless, the study of $\dot{V}O_2$ kinetics during recovery could be important for clarifying the etiology of the $\dot{V}O_2$ slow component and examining the possibility that the slow component of exercise $\dot{V}O_2$ should also be reflected in recovery.

Dynamic linearity is largely based on the apparent constancy of the on-transient fundamental time constant ($\tau$) to work rates of different magnitude (Whipp, 1971), and to the on/off symmetry of the fundamental response to exercise bouts of either moderate or heavy intensity (Paterson and Whipp, 1991). Regarding moderate exercise, a symmetrical response between the $\dot{V}O_2$ on- and off-transients (i.e., Phase 2) has often been reported (Engelen et al., 1996; Langsetmo and Poole, 1999; Özyener et al., 2001; Paterson and Whipp, 1991), but not by all (e.g., Rossiter et al., 2002). In the domain of heavy exercise, neither mono-exponentially nor on/off symmetry characterize the overall $\dot{V}O_2$ response (Linnarsson, 1974; Özyener et al., 2001; Paterson and Whipp, 1991); this is because the slow component that is evident in exercise is not very prominent in recovery. Previous findings from various laboratories have led to different conclusions concerning the linearity of $\dot{V}O_2$ during moderate- and heavy-intensity exercise (Brittain et al., 2001; Linnarsson, 1974; Özyener et al, 2001; Paterson and Whipp, 1991; Rossiter et al., 2002).

From these studies, it was suggested that the resultant dynamic nonlinearities operate exclusively at exercise intensities above (e.g., Rossiter et al., 2002) and
sometimes in the domain of moderate exercise (Brittain et al., 2001; Rossiter et al., 2002). For a range of physiologically defined exercise intensities, overall pulmonary O₂ uptake kinetics has been shown to be not symmetrical, except for moderate intensity exercise (Özyener et al., 2001). In apparent contrast to that study are the observations of Scheuermann et al. (2001), who found a perfect symmetrical dynamic response in the fundamental and even the slow components (separated by a time delay of ~130 s) of exercise and recovery periods for repeated cycling exercise above VT. It is likely, however, that some of the current divergence of opinion arose from differences in resolution of the responses and in the parameter estimation strategies.

Also, we cannot rule out the effect of exercise modality and muscle mass in these previous studies. Since symmetry is an essential feature of the dynamics of linear control (e.g., Milsum, 1966), we were interested in reexamining the relationship between the on- and off-transient kinetic responses of VO₂ to moderate- and heavy-intensity exercise in trained subjects. With respect to training level, there is a paucity in the literature that needs to be addressed. The resolution of this issue has important implications for the energy differences that act to modulate the kinetics of on- and off-transients. There is reason to suspect that the exercise and recovery responses might be different for heavy-intensity cycling exercise, with a discernible slow component during recovery and the fundamental off-transient τ being longer than the on-transient.

Methods

SUBJECTS AND PROCEDURES

Ten well-trained male cyclists, who had averaged 12 to 16 hours of training per week for at least 3 years, took part in the study. Their age, height, and body mass (√̅ ± SD) were 20 ± 4 yrs, 176 ± 6 cm, and 66 ± 9 kg, respectively. After receiving complete verbal and written details of the protocol, each subject gave informed written consent on a form approved by the local research ethics committee and in accordance with the guidelines set by the Declaration of Helsinki. Medical examinations and health questionnaires were conducted prior to the experiments, and all subjects were found to be healthy.

Exercise Protocol. The subjects came to the laboratory for three visits, during which all exercise tests were performed on a mechanically braked cycle ergometer (Ergoméca, Etablissement Giordano, Toulon, France). The first visit was used to gather descriptive data (questionnaires, anthropometric characteristics) and to conduct a continuous incremental exercise test so as to determine estimated ventilatory threshold (VT) and maximal oxygen uptake (VO₂max). After a 4-min warm-up period (100 W), the work rate was increased by 30 W every 3 min until voluntary exhaustion, which was defined as the inability to sustain the recommended pedaling rate of 80 rpm despite vigorous encouragement. The estimated VT was determined as the breakpoint in the plot of CO₂ uptake (VCO₂) as a function of VO₂, where the slope becomes >1 (V-slope method according Whipp et al., 1982). VO₂max was taken at the highest VO₂ over a 15-s period during the test. By using individual linear regressions of VO₂ over power output, we calculated the work rates for the subsequent visits for each subject.
A few days after the first visit, subjects undertook on different days two repetitions of both moderate and heavy exercise in a square-wave protocol consisting of 3 min rest, 6 min of exercise at the predetermined level, and 6 min of unloading pedaling (recovery). The work rates corresponded to 80% VT (moderate) and VT + 50%Δ (heavy), where delta (Δ) is defined as the difference between VT and VO2max. The work rate was applied and taken off abruptly, over 1–2 s without warning. Subjects were instructed to maintain a constant pedaling rate of 80 rpm throughout the tests. Visual feedback helped them maintain the cadence. We paid careful attention to this aspect, especially during the 50%Δ tests.

**Pulmonary Gas Exchange Measurement.** During all tests, minute expired ventilation and pulmonary gas exchange were measured continuously on a breath-by-breath basis using a metabolic measurement system (MedGraphics CPXD, Medical Graphics Corp., St Paul, MN). Subjects wore a noseclip and breathed through a mouthpiece. Prior to each exercise test, the O2 and CO2 gas analyzers were calibrated using gases of known concentrations. Volume calibration was performed with a 3L syringe (Hans Rudolph, Kansas City, MO). Ventilation was measured by a low-resistance pneumotachograph.

Heart rate was monitored using a three-lead electrocardiogram (Schiller Cardiovit AT60). The primary physiological data were displayed on-line and also stored on computer for subsequent analysis. A blood sample was taken from the ear lobe to determine blood lactate concentration (Lactate LM5, analyser, Analox instruments) 3 min after the end of the incremental test, at rest, and again every minute from the 3rd minute until the end of both the 80%VT and 50%Δ exercise tests.

**Data Analysis.** The breath-by-breath VO2 for each step transition in work rate were linearly interpolated at 1-s intervals, time-aligned to the onset of exercise, and ensemble-averaged to provide a single on- and off-transient for the 80%VT and 50%Δ exercise tests for each subject. This allowed us to reduce the breath-to-breath noise and enhance the underlying characteristics of the physiological responses (Engelen et al., 1996; Lamarra et al., 1987). The VO2 kinetics at exercise onset was characterized by either single or double exponential models (Barstow and Molé, 1991) as a function of time (t) by using a nonlinear least-squares fitting procedure. The immediate Phase I region (arising from increased lung perfusion) was excluded from the fitting field (i.e., ~20 s at the onset of an increased work rate). The VO2 on-transient was modeled throughout the entire exercise period with a single exponential model (M1; moderate-intensity exercise at 80%VT):

$$\dot{V}O_2(t) = A_1 \cdot [1 - e^{-(t-TD_1)/\tau_1}] \cdot U_1$$

(M1)

or with a double exponential model (M2; heavy-intensity exercise at 50%Δ):

$$\dot{V}O_2(t) = A_1 \cdot [1 - e^{-(t-TD_1)/\tau_1}] \cdot U_1 + A_2 \cdot [1 - e^{-(t-TD_2)/\tau_2}] \cdot U_2$$

(M2)

where $A_1$ is the difference between the last 30-s unloaded cycling baseline and the steady-state VO2 of the fundamental component, $A_2$ represents the asymptotic value for the slow component magnitude, $\tau_1$ and $\tau_2$ are the associated time constants, and $TD_1$ and $TD_2$ are the independent time delays of the fundamental and slow components, respectively. $U_1 = 0$ when $t < TD_1$; $U_1 = 1$ when $t \geq TD_1$; $U_2 = 0$ when $t < TD_2$; and $U_2 = 1$ when $t \geq TD_2$. 


For the off-transient after the 80% VT and 50%Δ exercise tests, the individual responses were fitted by using three models derived from the literature (i.e., Engelen et al., 1996; MacDonald et al., 1997; Paterson and Whipp, 1991). There was a marked drop in $\dot{V}O_2$ in the first few seconds after workload cessation, suggesting also the presence of a “cardiodynamic” phase in recovery. Therefore Phase I was not modeled during the off-transient (omission of the first 20 s). The first model, M3, consisted of a single exponential model for the entire recovery period (Paterson and Whipp, 1991):

$$\dot{V}O_2 (t) = EE\dot{V}O_2 - A_1 \cdot [1 - e^{-(t-TD_1)/\tau_1}] \cdot U_1$$  \hspace{1cm} (M3)

where $EE\dot{V}O_2$ is the end-exercise $\dot{V}O_2$, $A_1$ is the difference between $EE\dot{V}O_2$ and the steady-state $\dot{V}O_2$ (asymptote) for the exponential term, and $\tau_1$ and $TD_1$ are the associated time constant and delay: $U_1 = 0$ when $t < TD_1$, and $U_1 = 1$ when $t \geq TD_1$.

The second model, M4, was a double exponential model with the two terms beginning after independent time delays (Scheuermann et al., 1998; 2001):

$$\dot{V}O_2 (t) = EE\dot{V}O_2 - A_1 \cdot [1 - e^{-(t-TD_1)/\tau_1}] \cdot U_1 - A_2 \cdot [1 - e^{-(t-TD_2)/\tau_2}] \cdot U_2$$  \hspace{1cm} (M4)

Finally, the third model, M5, represented a double exponential model with both the fundamental and slow components constrained to begin simultaneously at the same time delay (Carter et al., 2000; Engelen et al., 1996; Özyener et al., 2001):

$$\dot{V}O_2 (t) = EE\dot{V}O_2 - A_1 \cdot [1 - e^{-(t-TD_1)/\tau_1}] \cdot U_1 - A_2 \cdot [1 - e^{-(t-TD_1)/\tau_2}] \cdot U_1$$  \hspace{1cm} (M5)

Statistical Analysis. The results are presented as mean ± SD for all variables. The $\dot{V}O_2$ kinetic parameter estimates were analyzed using a repeated-measures ANOVA design with 80%VT vs. 50%Δ exercise and on- vs. off-transients as the main effects. A significant $F$-ratio was further analyzed using Scheffé post hoc analysis. An $F$-test ($F = \text{model variance} \cdot \text{residual variance}^{-1}$) was used to decide which model led to a significant reduction in the sum of squared residuals as the criterion measure for each response. The more complex model was accepted if $p < 0.05$. Following the procedures outlined previously (Lamarra et al., 1987), we calculated the 95% confidence intervals to estimate the fundamental increase at the onset and the decrease at the end of exercise, as determined by $\tau_1$. Differences were declared to be significant at $p < 0.05$.

Results

Subject characteristics and the results of the physiological data obtained during the incremental test are listed in Table 1. $\dot{V}O_2\text{max}$ was 59.6 ± 4.0 ml·kg⁻¹·min⁻¹, with VT corresponding to 86.3 ± 5.4% of $\dot{V}O_2\text{max}$. Maximal heart rate was 191 ± 9 b·min⁻¹ and maximal respiratory exchange ratio was above 1.10 for all subjects. These results are consistent with other findings for endurance-trained athletes (Norris and Petersen, 1998; Poole et al., 1990).

Coefficient of determination obtained between actual $\dot{V}O_2$ and modeled responses was significant for all models used to characterize the kinetics of the $\dot{V}O_2$ response at the onset and end of moderate- and heavy-intensity exercise, $p < 0.05$. 
For the off-transient in moderate-exercise intensity, the parameter estimates derived from the double exponential models, M4 and M5, provided a lower residual summed squared error than the simplest model, M3, as confirmed by a higher $F$-test value. However, taking into account the complexity of the models, the residual variance was not significantly reduced for M4 and M5. Thus, as for the $V\text{.}O_2$ on-transient M1, a single monoexponential model was used to assess the $V\text{.}O_2$ off-transient during moderate exercise, M3. For the $V\text{.}O_2$ off-response after heavy-intensity exercise, there was a significantly reduced residual summed squared error for M4, $p < 0.05$, compared to M3 and M5, as confirmed by a higher $F$-test value.

Taking into account the complexity of the model (gain in variance), the residual variance was significantly reduced in 7 of the 10 subjects for M4, the double exponential model with two independent time delays. Moreover, time constants and amplitudes of all components did not differ significantly between M4 and M5. In short, M2 and M4 fit the data better than the other models tested for the heavy workloads, and thus were used to characterize both the $V\text{.}O_2$ on- and off-kinetic responses, respectively. These models incorporated the time course of the fundamental component and allowed a better discrimination of the fundamental and slow components transition.

The group mean $V\text{.}O_2$ on- and off-kinetic responses during the 80% VT and 50%Δ tests are shown in Figure 1. For the fundamental component, TD$_1$ did not change appreciably, regardless of exercise intensity and between the on- and off-transients. Similar $\tau_1$ values were obtained for the on-transient between the 80%VT and 50%Δ tests (Table 2). Lower values of $\tau_1$ were obtained for the off-transient

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**Table 1** Subject Characteristics Determined During Incremental Test

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Age (yrs)</th>
<th>Wt (kg)</th>
<th>Ht (cm)</th>
<th>Pmax (W)</th>
<th>$\text{VO}_2\text{max}$ (ml·kg$^{-1}$·min$^{-1}$)</th>
<th>$\text{VO}_2\text{max}$ (L·min$^{-1}$)</th>
<th>VT (%$\text{VO}_2\text{max}$)</th>
<th>HR max (b·min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>61</td>
<td>166</td>
<td>256</td>
<td>63.6</td>
<td>3.9</td>
<td>83.8</td>
<td>177</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>66</td>
<td>173</td>
<td>310</td>
<td>50.3</td>
<td>3.3</td>
<td>95.6</td>
<td>197</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>72</td>
<td>173</td>
<td>344</td>
<td>57.6</td>
<td>4.1</td>
<td>75.2</td>
<td>171</td>
</tr>
<tr>
<td>4</td>
<td>15</td>
<td>70</td>
<td>185</td>
<td>288</td>
<td>58.0</td>
<td>4.1</td>
<td>85.2</td>
<td>184</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>84</td>
<td>177</td>
<td>328</td>
<td>62.0</td>
<td>5.2</td>
<td>86.1</td>
<td>187</td>
</tr>
<tr>
<td>6</td>
<td>24</td>
<td>63</td>
<td>176</td>
<td>308</td>
<td>60.5</td>
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<td>186</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>59</td>
<td>181</td>
<td>290</td>
<td>56.8</td>
<td>3.4</td>
<td>86.3</td>
<td>201</td>
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<tr>
<td>8</td>
<td>27</td>
<td>58</td>
<td>174</td>
<td>256</td>
<td>62.4</td>
<td>3.6</td>
<td>86.1</td>
<td>202</td>
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<tr>
<td>9</td>
<td>17</td>
<td>54</td>
<td>173</td>
<td>256</td>
<td>61.3</td>
<td>3.3</td>
<td>87.1</td>
<td>196</td>
</tr>
<tr>
<td>10</td>
<td>26</td>
<td>76</td>
<td>182</td>
<td>352</td>
<td>63.0</td>
<td>4.8</td>
<td>85.4</td>
<td>184</td>
</tr>
<tr>
<td>M</td>
<td>20</td>
<td>66</td>
<td>176</td>
<td>298</td>
<td>59.5</td>
<td>3.9</td>
<td>86.3</td>
<td>191</td>
</tr>
<tr>
<td>$SD$</td>
<td>±4</td>
<td>±9</td>
<td>±6</td>
<td>±35</td>
<td>±4.0</td>
<td>±0.6</td>
<td>±5.4</td>
<td>±9</td>
</tr>
</tbody>
</table>

*Note:* VT = ventilatory threshold; $\text{VO}_2\text{max}$ = maximal $O_2$ uptake; HR max = maximal heart rate; Pmax = maximal aerobic power.
during moderate exercise when compared with heavy exercise, but the difference was not statistically significant, \( p = 0.09 \). Note that \( \tau_1 \) was significantly longer (+6.5 s) during the \( \dot{V}O_2 \) off- than the \( \dot{V}O_2 \) on-transient, but only for the heavy-intensity exercise. With two repetitions, the 95% confidence intervals for estimating \( \tau_1 \) were 2.9 and 4.0 s on average for heavy exercise and subsequent recovery, respectively (Lamarra et al., 1987). As expected, the amplitude of the fundamental component (\( A_1 \)) for heavy exercise was significantly higher than for moderate exercise for both the \( \dot{V}O_2 \) on- and off-transients. For heavy-intensity exercise, no significant differences were observed concerning the slow component parameter estimates (i.e., \( A_2 \), \( \tau_2 \), and \( TD_2 \)) between the on- and off-kinetic responses (Table 2).

For both moderate- and heavy- intensity exercise, \( \dot{V}O_2 \) returned to baseline values within 6 min of recovery (baseline \( \dot{V}O_2 \): 22.7 ± 1.8 vs. end-recovery \( \dot{V}O_2 \): 22.0 ± 3.0 ml·kg\(^{-1}\)·min\(^{-1}\) for the 50%Δ test; baseline \( \dot{V}O_2 \): 21.9 ± 4.6 vs. end-recovery \( \dot{V}O_2 \): 20.3 ± 2.8 ml·kg\(^{-1}\)·min\(^{-1}\) for the 80% VT test, \( p > 0.05 \)).
The literature on studies that have examined VO₂ kinetics during the recovery period following exercise of various intensities shows different results concerning dynamic linearity in VO₂ responses. The issue of whether the slow component is retained or is much less pronounced during recovery is still being debated (Cunningham et al., 2000; Engelen et al., 1996; Özyener et al., 2001; Paterson and Whipp, 1991; Scheuermann et al., 1998; 2001). The major findings of the present study performed with trained subjects were twofold. First, there was a dynamic asymmetry of O₂ uptake between the on- and off-transients (i.e., fundamental component) in heavy-intensity exercise, but not in moderate-intensity exercise. Second, the slow component of O₂ uptake observed during heavy-intensity exercise was retained in recovery, with similar parameter estimates.

Discussion

The literature on studies that have examined VO₂ kinetics during the recovery period following exercise of various intensities shows different results concerning dynamic linearity in VO₂ responses. The issue of whether the slow component is retained or is much less pronounced during recovery is still being debated (Cunningham et al., 2000; Engelen et al., 1996; Özyener et al., 2001; Paterson and Whipp, 1991; Scheuermann et al., 1998; 2001). The major findings of the present study performed with trained subjects were twofold. First, there was a dynamic asymmetry of O₂ uptake between the on- and off-transients (i.e., fundamental component) in heavy-intensity exercise, but not in moderate-intensity exercise. Second, the slow component of O₂ uptake observed during heavy-intensity exercise was retained in recovery, with similar parameter estimates.

**VO₂ KINETIC ANALYSIS**

In the present study, VO₂ off-transients for the 80% VT test were well described by a single exponential model (Phase I not modeled), and no discrete slow component for the VO₂ recovery response could be resolved. This is consistent with previous studies which also reported that VO₂ kinetics following exercise of moderate intensity was best fitted by a single (Özyener et al., 2001; Paterson and Whipp, 1991) or by a double (Phase I included, Scheuermann et al., 1998) exponential model. Regarding the VO₂ recovery response for the 50%Δ test, a significantly reduced residual variance was obtained with the double exponential model M4 with two independent time delays, in comparison with the single exponential model M3 and the double exponential model M5 with a similar time delay.

Taking into account the complexity of the model (gain in variance), it appeared that M4 was more appropriate for describing the VO₂ kinetic response than

**Table 2** Summary of Parameter Estimates (M ± SD) for Model Fit of Response in Exercise and During Recovery Period (N = 10)

<table>
<thead>
<tr>
<th></th>
<th>Moderate Intensity (80% VT)</th>
<th>Heavy Intensity (50%Δ)</th>
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<tbody>
<tr>
<td></td>
<td>On-transient</td>
<td>Off-transient</td>
</tr>
<tr>
<td>A₁ (ml·min⁻¹)</td>
<td>1420 ± 397*</td>
<td>1596 ± 296*</td>
</tr>
<tr>
<td>τ₁ (s)</td>
<td>20.9 ± 8.9</td>
<td>22.4 ± 6.6</td>
</tr>
<tr>
<td>TD₁ (s)</td>
<td>19.8 ± 6.9</td>
<td>16.3 ± 7.9</td>
</tr>
<tr>
<td>A₂ (ml·min⁻¹)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>τ₂ (s)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>TD₂ (s)</td>
<td>–</td>
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</table>

*Significantly different from corresponding parameter during 50%Δ exercise, p < 0.05; ††Significantly different compared to off-transient, p < 0.01.
M3 or M5. When considering the recovery period following exercises performed above VT, even if most studies are in agreement with the fact that three phases can be distinguished, there is no consensus in the literature as to which mathematical model should be used. Recently MacDonald et al. (1997), Engelen et al. (1996), and Cunningham et al. (2000) allowed Phases II and III to begin together (i.e., M5). MacDonald et al. justified the selection of this model by the absence of a statistically significant improvement in fit between M4 and M5, whereas Engelen et al. justified their choice because the two main exponential processes observed during heavy exercise begin at different times into exercise (TD1 and TD2), and both are present at the end of exercise and would decay simultaneously during early recovery, but presumably at different rates.

Scheuermann et al. (2001) included independent time delays for each recovery phase in their model, M4, and reported the presence of a slow component with similar patterns during exercise as during recovery, highlighting the same results as in our study regardless of the subjects’ training level. To date there seems to be no clear physiological explanation for the presence of a second time delay during recovery, as well as for the presence of a common time delay (Scheuermann et al., 1998; 2001).

Initially the VO2 off-transient kinetics after a bout of high-intensity exercise was described by the sum of (a) a fast exponential component corresponding to the phosphocreatine and myoglobin replenishment stores by 2–3 min, and (b) a slow component with a slower time course previously considered as the “lactic O2 debt” (Margaria et al., 1933), suggesting a second metabolic compartment. Nowadays, evidence suggests that the fast component, and much of the slow component, may be explained by the changes in phosphocreatine concentration (PCr) during and after exercise (Harris et al., 1976; Rossiter et al., 2002). The slow component is also attributed by Harris et al. (1976) to the changes in muscle pH occurring after exhausting exercise. Since the mitochondrion is the site of O2 consumption in the cell, the explanation of the biphasic time course of VO2 off-transient should be found at the level of this cellular organelle. In this case we are faced with a complex issue, as a variety of additional factors (e.g., catecholamines, calcium ions, muscle temperature) have been shown to exert an influence on mitochondrial respiration, which was proposed to explain the slow component during exercise (Linnarsson, 1974; Whipp, 1994). More work is needed in this area, however, to resolve the issue.

**DYNAMIC RESPONSES OF THE FUNDAMENTAL COMPONENT**

In the present study, no significant differences were observed between the VO2 on- and off-transients with regard to TD1, τ1, and A1 for the 80%VT cycling exercise. This result is in accordance with previous studies which also noted a symmetry between the exercise and recovery kinetics of VO2 for exercise below VT (Engelen et al., 1996; Özyener et al., 2001; Paterson and Whipp, 1991; Scheuermann et al., 1998). This symmetry is consistent with traditional theories about O2 deficit and debt during such exercise intensities, i.e., the postexercise VO2 quantitatively matched the O2 deficit.

Regarding the 50%Δ test, no significant differences were observed between the on- and off-transients for TD1 and A1 (Figure 1), but a significantly longer τ1
was obtained for the off-transient. This result concerning $\tau_1$ is apparently not in agreement with some recent studies from the literature. Engelen et al. (1996), Özyener et al. (2001), and Scheuermann et al. (2001) showed similar $\tau_1$ values for the VO$_2$ on- and off-transients during heavy cycling exercise. In contrast with our study in which we used an identical mathematical model during both exercise and recovery (i.e., M4) with trained subjects, most of these authors used a double exponential model beginning after independent time delays (except for Scheuermann et al., 2001) to describe the fundamental and slow components during exercise, but the latter components were constrained to begin together after a common time delay during recovery. Surprisingly, for exercise of similar intensity (but with untrained subjects) and with a model similar to M4, Scheuermann et al. (1998) reported faster VO$_2$ off- compared to VO$_2$ on-responses during the fundamental phase.

In the present study, however, the fact that the fundamental component during recovery was slower than during the 50%Δ exercise period is consistent with recent studies evaluating O$_2$ uptake and PCr kinetics (e.g., Rossiter et al., 2002). Rossiter et al. (2002) reported a consistent “on/off” asymmetry during moderate and high-intensity knee-extensor exercises for O$_2$ uptake and PCr.

Several studies (Arnold et al., 1984; MacCann et al., 1995; Taylor et al., 1986) have shown that the rate of recovery of PCr is slower following intense as opposed to light exercise. This phenomenon is not clearly identified, however, and possible factors have been put forward and discussed: the effects of increased concentrations of hydrogen ions on the creatine kinase equilibrium reaction, shifting the equilibrium toward PCr hydrolysis, binding or sequestration of inorganic phosphate, or muscle fiber recruitment patterns (MacCann et al., 1995). Taken together, this may help explain the similar time constant during and following the 80%VT test as well as the slower $\tau_1$ during the fundamental phase after the 50%Δ test. Note that in our study, $\tau_1$ did not change with work rate intensity during VO$_2$ on-transient. This result is consistent with the notion that there is an apparent fundamental phase of VO$_2$, the kinetics of which is independent of the energy requirement (i.e., the work rate imposed), as previously suggested (Barstow et al., 1996; Özyener et al., 2001).

DYNAMIC FEATURE OF VO$_2$ SLOW COMPONENT

The second important result of the present study was the presence of a delayed VO$_2$ slow component, with similar amplitude and time-constant estimates, during recovery following heavy-intensity exercise (Table 2 and Figure 1). Recovery VO$_2$ kinetics showed a clear downward trend after the fundamental component was resolved (i.e., TD$_2$ of ~111 s) and returned to preexercise levels at the end of the 6-min recovery period. Finally, the percentage contribution of the slow component to the total VO$_2$ response amplitude in exercise and recovery was similar (11.5 vs. 10.6%, respectively). These data differ from those of Paterson and Whipp (1991) and Özyener et al. (2001), who found, for exercise of comparable intensity, a greater amplitude for the fundamental component and a slow component considerably smaller in recovery compared to exercise.

Our results, however, are in line with other studies (Barstow et al., 1996; Engelen et al., 1996; Scheuermann et al., 1998; 2001) which have also reported a slow component of significant magnitude during the recovery period of heavy-
intensity exercise. But most of these later studies reported either a nonexistent (Barstow et al., 1996; Engelen et al., 1996; Özyener et al., 2001) or a considerably reduced time delay (Scheuermann et al., 1998) of the slow phase in recovery compared to exercise. Only Scheuermann et al. (2001) obtained similar time delay values of 124 ± 39 for exercise and 147 ± 85 for recovery. Thus our results and those of Scheuermann et al. (2001) with trained and untrained subjects, respectively, suggest that the physiological process involved in the VO₂-on slow component could be retained in the VO₂-off slow component.

The symmetry of the slow-component VO₂ kinetics has significant implications for the mechanism itself. Actually, the main physiological process proposed to explain the slow component is an increase in recruited muscle mass and/or an increased reliance on less efficient type II muscle fibers in order to maintain the required power output. However, the presence of a second time delay with the same characteristics for recovery as for exercise proves that alternative explanations must be considered. Support for this is provided by Scheuermann et al. (2001), showing that the slow component of O₂ uptake is not accompanied by changes in muscle EMG during heavy exercise.

The underlying mechanism(s) of the recovery slow component have not been well examined and remain unclear. Several mechanisms that have been suggested as potentially participating in the recovery slow component include blood lactate, temperature, or catecholamines (Gaesser and Brooks, 1984; Langsetmo and Poole, 1999; Scheuermann et al., 1998). In the present study, A2 during the recovery after the 50%Δ test was not significantly correlated with the end-exercise blood lactate concentration (r = 0.27, p > 0.05). From the physiological data of the present study, however, it was impossible to clearly determine the other factors responsible for the recovery slow component, and further studies are warranted.

In conclusion, the results of the present study demonstrated that a dynamic asymmetry of the fundamental component was observed between VO₂ on- and off-transients of heavy cycling exercise in trained subjects, as shown by a longer time constant in recovery, but not for moderate exercise. For heavy exercise intensity (>VT), a slow component of oxygen uptake was present during both exercise and recovery, with similar magnitude and time course. These results demonstrated that the physiological process involved in the VO₂ slow component during exercise could be retained in recovery.

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References


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