Fatigue Responses in Exercise under Control of \(\dot{V}O_2\)

**Abstract**

To examine the fatigue response during an exhaustive heavy exercise performed under control of oxygen uptake (SS@\(\dot{V}O_2\Delta50\)) or power output (SS@pa50), eleven trained male subjects performed an incremental test to determine the peak of the oxygen uptake value (\(\dot{V}O_2\text{peak}\)) and lactate threshold and two exhaustive steady-state cycling exercises at the intermediate value between the lactate threshold and \(\dot{V}O_2\text{peak}\) (SS@\(\dot{V}O_2\Delta50\) and SS@pa50). The control of \(\dot{V}O_2\) induced an oscillation of the power output, which lowered the average power output (276 ± 47 vs. 315 ± 40 W, \(p = 0.004\)) and cancelled the slow component of oxygen kinetics. However, all subjects reached maximal cardiac output (CO) and heart rate (HR) values which were sustained almost two times longer in SS@\(\dot{V}O_2\Delta50\) compared to SS@pa50 (979 ± 854 vs. 475 ± 236 s, \(p = 0.046\) for CO and 1050 ± 890 vs. 513 ± 288 s, \(p = 0.037\) for HR). Furthermore, SS@pa50 elicited \(\dot{V}O_2\text{peak}\) but not SS@\(\dot{V}O_2\Delta50\) (4963 ± 434 vs. 4723 ± 460 mL·min\(^{-1}\), \(p = 0.026\)). Finally, the time spent at the maximal CO and HR values is correlated with time to exhaustion at \(\dot{V}O_2\Delta50\). In conclusion, the cause of fatigue does not seem to have the same origin during exhaustive supra-lactate threshold exercise under control of \(\dot{V}O_2\) (\(\dot{V}O_2\Delta50\)) compared to constant power output (pa50), while both elicit the maximal HR and CO values.

**Introduction**

Exhaustive supra lactate threshold exercises lasting from 10 to 45 min induce a progressive increase in the oxygen uptake (\(\dot{V}O_2\)) and heart rate (HR) until their maximal values in the incremental test are achieved [6,14,26]. However, the causes of fatigue are still unknown during these supra lactate threshold exercise bouts. The \(\dot{V}O_2\) increase has been attributed to the progressive muscular fiber recruitment to support the constant power output, and/or respiratory muscle fatigue during severe exercise [8]. The amplitude of the \(\dot{V}O_2\) slow component and kinetics are not strongly correlated with the time of fatigue [5]. Recently, we observed that severe exhaustive exercise performed at intensities between the lactate threshold and the power associated with maximal stroke volume (pSV\(_{\text{max}}\)), induced maximal \(\dot{V}O_2\) values with a lower cardiac output (CO) compared to incremental tests [22]. This was associated with a slow decrease in oxygen arterial venous difference (a-V\(O_2\)diff) which was correlated with the arterial oxygen desaturation that appeared during constant exercise [22]. Therefore, maximal exercise would terminate before the development of a plateau in either coronary flow, CO, \(\dot{V}O_2\), or the onset of skeletal muscle anaerobiosis. Thus, cardiovascular function “limits” maximum exercise capacity, probably due to myocardial oxygen delivery limitations, as a result of increased sympathetic nerve control during acute exercise [11]. Accordingly, the control of the velocity or power by physiological variables, such as the oxygen uptake, could be an experimental model for appreciating the physiological control of power output during exhaustive exercise and the sustainability of a given exercise intensity.

No study has focused on the effect of the power output versus \(\dot{V}O_2\) steady-state during exhaustive exercise sustained above lactate threshold on the cardiac response and time to exhaustion. Therefore, the purpose of this study was to compare the CO response during exhaustive exercise performed under the control of mechanical power to maintain a steady-state of oxygen uptake (SS@\(\dot{V}O_2\Delta50\)) and a constant mechanical...
power output (SS@p50) at the intermediate value (A50) between the lactate threshold and V\textsubscript{O2}\textsubscript{peak}. We hypothesized that 1) the control of mechanical power to maintain a VO\textsubscript{2} steady-state could induce a decrease in the power which cancelled the slow component of VO\textsubscript{2}, and 2) a longer time spent at the maximal CO and HR for in SS@VO\textsubscript{2}A50 compared to SS@pA50.

**Methods**

**Subjects**

Eleven well-trained male volunteers (9 triathletes and 2 cyclists) participated in this study. These subjects were regional short-distance triathletes and regional level cyclists at the start of the training season. All subjects were free from cardiac and pulmonary disease. Their physical characteristics are reported in Table 1. Each subject was familiarized with the experimental procedures prior to the study. To minimize training effects, subjects were asked to train below 70% of their V\textsubscript{O2}\textsubscript{peak}, whatever their heights were set for each subject and kept constant for all the procedures prior to the study. To minimize training effects, subjects were free from cardiac and pulmonary disease. Their physical characteristics are reported in Table 1. Each subject was familiarized with the experimental procedures prior to the study. To minimize training effects, subjects were asked to train below 70% of their V\textsubscript{O2}\textsubscript{peak}, whatever their physical activities, at least three weeks before and during the study. Before participation, they were informed of the risks and stresses associated with the protocol, and gave a written voluntary informed consent in accordance with the guidelines of the University of Evry – Val d’Essonne.

**Protocol**

All tests were performed with the subjects in the upright position on an electronically braked cycle ergometer (ERGOLINE 900, Hellige, Markett, Bitz, Germany), two to three hours after a light breakfast, in a air-conditioned room (22.0 ± 0.5 °C), without any fluid and substrate replacements. Seat and handlebar heights were set for each subject and kept constant for all the tests, as was the pedalling frequency, which was selected by each subject in a range of 70 and 110 revolutions·min\textsuperscript{-1}.

**Incremental test**

After a threeminute warm-up at 80 Watts, each subject performed an incremental exercise test to exhaustion with a 40 W work increment every three minutes for a total exercise duration not exceeding 20 min. V\textsubscript{O2}\textsubscript{peak} was defined as the highest 30-s oxygen uptake achieved during exercise with a respiratory exchange ratio greater than 1.1, blood lactate greater than 8 mM and a peak heart-rate at least equal to 90% of the age-predicted maximum [4,20]. A plateau of VO\textsubscript{2} was identified if the VO\textsubscript{2} of the last stage was not greater than the previous one by 1.75 mL·kg\textsuperscript{-1}·min\textsuperscript{-1} [4]. The pV\textsubscript{O2}\textsubscript{peak} was determined as the lowest power eliciting V\textsubscript{O2}\textsubscript{peak} [4]. If during the last stage a subject achieved V\textsubscript{O2}\textsubscript{peak} without completing the 3-min stage, pV\textsubscript{O2}\textsubscript{peak} was calculated as following:

\[ pV\textsubscript{O2}\textsubscript{peak} = pF + (\{t/180\}×40) \]

(\text{eq. 1})

Where pF was the power of the last complete stage (W), t as the time the last workload was maintained (s), and 40 W, the power output increment between the last of two stages [20].

**Constant work rate and constant oxygen uptake test**

48 hours after the incremental test, each subject completed two high intensity constant workload exercises until exhaustion separated by at least 48 hours. Time limit (tlim) was the time at which the subject was no longer able to pedal at the given power output. After a 15-min warm-up at 30% of pV\textsubscript{O2}\textsubscript{peak} and 5 min at rest, the work rate was increased within 30 s to pA50 and sustained constant during 3 min. Thus, each subject performed: 1) an all-out constant-load exercise at a work rate requiring pA50 (SS@pA50, derived from the incremental exercise); and 2) an all-out steady-state V\textsubscript{O2} exercise at a V\textsubscript{O2} requiring an oxygen uptake associated with pA50 during the incremental test (SS@VO\textsubscript{2}A50). The power pA50 was defined as the oxygen uptake value midway between the power associated at the lactate threshold (pLT) and the work requiring 100% of pV\textsubscript{O2}\textsubscript{peak}. VO\textsubscript{2}A50 was defined as the oxygen uptake value midway between the VO\textsubscript{2} values associated at pLT and the V\textsubscript{O2}\textsubscript{peak} during the incremental test. The pLT, pA50 and VO\textsubscript{2}A50 values were determined by two independent researchers. All subjects were given verbal encouragement throughout each trial.

**Steady state oxygen uptake test**

The cycle ergometer was connected to one serial port of the PC with the second serial port connected to the K4b\textsuperscript{2} gas exchanges analyzer (COSMED, Roma, Italy) [23]. Physio-Trainer software (COSMED systems, Roma, Italy) contained the handshake protocols to communicate with Ergoline 900 ERG, and to allow a manual or automatic ergometer control. After the increase within 30 sec and 3 min bout at constant pA50 intensity, the investigators were able to use the control function. The Physio-Trainer software dynamically controlled the ergometer workload in order to maintain the desired VO\textsubscript{2} value, according to a feedback algorithm based on the target VO\textsubscript{2}, and the dynamics of its change. The adaptation kinetic of work rate increments depends on 1) the referring parameter response (VO\textsubscript{2} increase), 2) the updating rate of every new power output, 3) the initial, maximal and minimal workload and maximal increment elicited by the ergometer, and 4) the gain (K1) and the stability factor (K2, as a brake to the eventual oscillations) of the feedback system. K1 and K2 parameters determined the degree of the sensibility ergonomic adaptation to the stimulus variability. The maintenance of constant pA50 workload during the first 3 min of exercise allowed us avoid large variations in power during the early moments of exercise before VO\textsubscript{2} had stabilized.

**Data collection procedures**

Gas and cardiac parameters measurements

V\textsubscript{O2}, V\textsubscript{CO2} and V\textsubscript{E} were measured throughout each test using the COSMED K4b\textsuperscript{2} telemetric system [23]. Expired gases were measured, breath-by-breath, and averaged every 5 seconds. In addition, we used a bio-impedance method to determine stroke volume (SV), heart rate (HR) and cardiac output (CO) (Physio-Flow, Manatec Type PFS01L, Strasbourg, France). The theoretical basis for this technique and its application and validity for exercise testing have been previously described [9] and used in pre-

**Table 1** Physical characteristics of the subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age (years)</th>
<th>Mass (kg)</th>
<th>Height (cm)</th>
<th>Body fat (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 11)</td>
<td></td>
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</tr>
<tr>
<td>Mean ± SD</td>
<td>35 ± 6</td>
<td>69 ± 5</td>
<td>176 ± 8</td>
<td>11.3 ± 2.8</td>
</tr>
</tbody>
</table>

where \( u_1 = 0 \) when \( TD_1 = 0 \) s, and \( u_1 = 1 \) when \( TD_1 > 0 \) s, \( u_2 = 0 \) when \( TD_2 = 0 \) s, and \( u_2 = 1 \) when \( TD_2 > 0 \) s. \( V\dot{O}_2(t) \) represents oxygen uptake (mL·min\(^{-1} \)) at time \( t \) in seconds, \( V\dot{O}_2b \) is the baseline of the value at the end of the warm-up, \( A_1 \) is the asymptotic amplitudes of the exponential term, \( \tau_1 \) the time constant of the exponential, \( TD_1 \) as the time delay, \( \tau_2 \) the time delay for the exponential regression, \( A_2 \) is the slope of the linear regression, \( TD_2 \) is the time delay for the linear regression.

Each curve fitting used an iterative least-squares approach. The rationale for selection of the appropriate model was based on analysis of the residuals around the line of best fit. The first 15–20 s, representing the phase I response, were eliminated for these analyses. Similar analysis was also carried out for the stroke volume, heart rate, cardiac output and arterial-venous oxygen difference kinetics in order to explain the CO – \( V\dot{O}_2 \) relationship from fast (phase II) and slow component (phase III) of oxygen uptake response during constant intensity severe exercise.

At last, we applied a fourth model to describe the \( a\text{-}v\dot{O}_2\text{diff} \) kinetics: the mono-exponential plus drop, with the best descriptions for phase II consistently being given by the latter:

\[
a\text{-}v\dot{O}_2\text{diff}(t) = a\text{-}v\dot{O}_2\text{diff}(b) + A_1 \times \left[ 1 - e^{- (t-TD_1)/\tau_1} \right] \times u_1 + A_2 \times \left[ (t-TD_2)/\tau_2 \right] \times u_2 \tag{5.5}
\]

where \( u_1 = 0 \) when \( TD_1 = 0 \) s, and \( u_1 = 1 \) when \( TD_1 > 0 \) s, \( u_2 = 0 \) when \( TD_2 = 0 \) s, and \( u_2 = 1 \) when \( TD_2 > 0 \) s. \( V\dot{O}_2(t) \) represents oxygen uptake (mL·min\(^{-1} \)) at time \( t \) in seconds, \( V\dot{O}_2b \) is the baseline of the value at the end of the warm-up, \( A_1 \) is the asymptotic amplitudes of the exponential term, \( \tau_1 \) the time constant of the exponential, \( TD_1 \) as the time delay, \( A_2 \) is the slope of the linear regression, \( TD_2 \) as the time delay for the exponential regression.

Data analysis

Anthropometry

Height and mass were measured before and after each test. Five skinfold measurements were made (triceps, biceps, suprailiac and subscapular) and \% of body fat calculated using the Durnin and Womersley's formula [13].

Data modelling kinetics

For each exercise bout within all subjects, the repetitions were time aligned to the start of exercise and averaged to reduce the breath-to-breath noise and enhance the underlying physiological response pattern. These averaged responses for each subject were then used to evaluate the effect of exercise on \( V\dot{O}_2 \), HR, SV, CO, and \( a\text{-}v\dot{O}_2\text{diff} \) kinetics. The time courses of measured values were described in terms of exponential functions fitted to the data by nonlinear regression techniques. The calculation of the best-fit parameters was chosen by the program so as to minimize the sum of the squared differences between the fitted function and the observed response (SigmaPlot2000, Jandel, Chicago, IL, USA).

Two exponential models were used to describe \( V\dot{O}_2 \) for each work rate. According to the both following equations, oxygen uptake kinetics fit by either mono (eq. 3) and double-exponential (eq. 4) function [2]:

\[
V\dot{O}_2(t) = V\dot{O}_2b + [A_1 \times e^{- (t-TD_1)/\tau_1}] \times u_1 \tag{3}
\]

\[
V\dot{O}_2(t) = V\dot{O}_2b + [A_1 \times e^{- (t-TD_1)/\tau_1}] \times u_1 + \left[ A_2 \times e^{- (t-TD_2)/\tau_2} \right] \times u_2 \tag{4}
\]

where \( u_1 = 0 \) when \( TD_1 = 0 \) s, and \( u_1 = 1 \) when \( TD_1 > 0 \) s, \( u_2 = 0 \) when \( TD_2 = 0 \) s, and \( u_2 = 1 \) when \( TD_2 > 0 \) s. \( V\dot{O}_2(t) \) represents oxygen uptake (mL·min\(^{-1} \)) at time \( t \) in seconds, \( V\dot{O}_2b \) is the baseline of the value at the end of the warm-up, \( A_1 \) and \( A_2 \) are the asymptotic amplitudes for the second and third exponential of oxygen uptake kinetics, \( \tau_1 \) and \( \tau_2 \) are the time constants, \( TD_1 \) and \( TD_2 \) as the time delays of each exponential.

The mono-exponential plus linear component model was also applied, with the best descriptions for phase II consistently being by the latter:

\[
V\dot{O}_2(t) = V\dot{O}_2b + [A_1 \times e^{- (t-TD_1)/\tau_1}] \times u_1 + A_2 \times (t-TD_2)/\tau_2 \tag{5}
\]

where \( u_1 = 0 \) when \( TD_1 = 0 \) s, and \( u_1 = 1 \) when \( TD_1 > 0 \) s, \( u_2 = 0 \) when \( TD_2 = 0 \) s, and \( u_2 = 1 \) when \( TD_2 > 0 \) s. \( V\dot{O}_2(t) \) represents oxygen uptake (mL·min\(^{-1} \)) at time \( t \) in seconds, \( V\dot{O}_2b \) is the baseline of the value at the end of the warm-up, \( A_1 \) is the asymptotic amplitudes of the exponential term, \( \tau_1 \) the time constant of the exponential, \( TD_1 \) as the time delay, \( A_2 \) is the slope of the linear regression, \( TD_2 \) the time constant of the second function and \( TD_2 \) is the time delay for the linear regression.

Calculation of the time to achieve the maximal values (\( TA \)) and the time to spent at the maximal values (\( T5 \))

The steady-state at \( V\dot{O}_2\text{peak} \) was considered to be achieved where from 95% of the incremental \( V\dot{O}_2\text{peak} \) was achieved. The time to achieve 95% of maximal value of \( V\dot{O}_2 \) during the test (\( TA_{V\dot{O}_2} \)) was calculated according to following equation used for the mono-(Eq. 3) or double-exponential (Eq. 4).

1. For the mono-exponential function starting from Eq. 3, solving time \( (t) \):

\[
t = -\tau \times \ln \left[ 1 - (V\dot{O}_2(t) - V\dot{O}_2b)/A \right] \tag{7}
\]

Specifically, when \( V\dot{O}_2(t) \) has achieved 95% \( V\dot{O}_2\text{peak} \)

\[
TA = -\tau \times \ln \left[ 1 - (0.95 \times V\dot{O}_2\text{peak} - V\dot{O}_2b)/A \right] \tag{8}
\]

2. For the double-exponential function (Eq. 4), a similar approach is used:

\[
TA = TD_2 \times \tau_2 \times \ln \left[ 1 - (0.95 \times V\dot{O}_2\text{peak} - A_1 - V\dot{O}_2b)/A_2 \right] \tag{9}
\]

Similar analysis was also carried out for the stroke volume (\( TA_{SV} \)), heart rate (\( TA_{HR} \)) and cardiac output (\( TA_{CO} \)). Therefore, when associated with the time to exhaustion, the time spent at the maximal value was calculated as the difference between
The maximal values measured during all exercise tests are given in Table 2. During constant exercise, the control of the power output with $\dot{V}O_2$ induced an oscillation of the power output throughout the SS@p50 test (Fig. 1). Though the mean power was lower in SS@p50 compared to the SS@p50 (276 ± 47 vs. 315 ± 40 W, p = 0.004), the maximal HR and SV values were reached in both SS@p50 and SS@V˙O250 tests. Therefore CO reached its maximal value in all exercises. From third minute to exhaustion, a $\dot{V}O_2$ slow component ($\Delta$V˙O2 = 498 ± 147 mL min⁻¹) was superimposed in SS@p50, whereas the power output oscillation cancelled the slow component in $\dot{V}O_2$

kinetics in SS@V˙O250 (Table 3). Therefore, SS@p50 elicited the maximal values of $\dot{V}O_2$ (4963 ± 434 vs. 4950 ± 439 mL min⁻¹ in the incremental test, p = ns), while SS@V˙O250 did not (4723 ± 460 mL min⁻¹, p < 0.05 and p = 0.026, for the incremental and SS@p50 tests, respectively).

Furthermore, the greater the decrease in power, the larger was the increase in the time to fatigue ($r$ = 0.75, p = 0.006) (Fig. 2). Hence, the time to exhaustion was longer in SS@V˙O250 compared to the SS@p50 (1186 ± 897 vs. 616 ± 287 s, p = 0.026). The maximal CO and HR were also sustained during a longer time in SS@V˙O250 compared to SS@p50 (979 ± 854 vs. 475 ± 236 s, p = 0.046 for CO and 1050 ± 890 vs. 513 ± 288 s, p = 0.037 for HR). Finally, the time spent at maximal HR and CO values were highly correlated with the time to fatigue in SS@p50 ($r$ = 0.98, p < 0.001 for HR and r = 0.79, p = 0.034 for CO, n = 11) and in SS@V˙O250 ($r$ = 0.99, p < 0.001 for HR and r = 0.98, p < 0.0001 for CO, n = 11) (Figs. 3 and 4).

### Discussion

The main findings of this study are the following: 1) the control of $\dot{V}O_2$ induced a decrease in power output which cancelled (eliminated) the slow component of $\dot{V}O_2$ kinetics, and b) a longer time to fatigue in the SS@V˙O250 compared to SS@p50; and 2) all subjects reached and sustained their maximal CO and HR for a longer period of time in SS@V˙O250 compared to SS@p50. The time spent at the maximal CO values was also highly correlated with the time to fatigue in both conditions. It has been demonstrated that supra-lactate exercise workloads between 85 and 100% of the power associated with $\dot{V}O_2$peak in an incremental test will elicit $\dot{V}O_2$peak provided exercise is performed for a sufficient time duration (5 – 15 min according to the exercise intensity) [4, 5, 19]. In the present study, a slow rise of $\dot{V}O_2$ until $\dot{V}O_2$peak was observed in the constant power output exercise at p50. However, a decrease in the power output from 87.5 to 76.6% of $\dot{V}O_2$peak allowed a $\dot{V}O_2$ steady-state throughout the SS@V˙O250 test. Therefore, the oscillation of the power output was responsible for the $\dot{V}O_2$ steady-state and a longer exercise time in the SS@V˙O250 test compared to the SS@p50 test for which $\dot{V}O_2$ reached its maximal value. On the other hand, CO and HR reached their maximal values prior to exhaustion in the constant power output and $\dot{V}O_2$ steady-state exercises. Indeed, the CO does not increase immediately to the steady-state value following the onset of exercise. So, the available evidence suggested that the rate of $O_2$ kinetics at the onset of exercise reflected the capacity of central $O_2$ delivery and muscle $O_2$ utilization [27]. Hence, the alveolar $\dot{V}O_2$ could increase exponentially and at similar rates as leg blood flow in fast component kinetics. Indeed, the SV adjustment could be the main factor of the $\dot{V}O_2$ kinetics changes during a constant load exercise at 90% of $\dot{V}O_2$peak [7]. At the onset of dynamic exercise, the mechanical effects of the muscle pump increased the arterial-venous pressure gradient [12]. Therefore, the pumping action of contracting skeletal muscle appeared to form an important regulator of increasing blood flow. So, the rate of increase in blood flow during exercise was closely coupled to motor unit recruitment with dilation beginning at the first contraction. Therefore, the quicker the recruitment of a large muscle mass, the higher the SV and CO response at the onset of exercise. Grassi et al. [17] demonstrated that, in exercising humans during transitions from unloaded pedalling to load pedalling below the ventilatory thresh-

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Table 2 Maximal individual physiological responses and power values determined in incremental and constant exercise tests

<table>
<thead>
<tr>
<th>Subjects (n = 11)</th>
<th>Incr.</th>
<th>SS@p50</th>
<th>SS@V˙O250</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to fatigue (seconds)</td>
<td>1296 ± 140</td>
<td>616 ± 278b</td>
<td>1186 ± 897b</td>
</tr>
<tr>
<td>Power (Watts)</td>
<td>360 ± 40</td>
<td>315 ± 40b</td>
<td>276 ± 47b (mean power)</td>
</tr>
<tr>
<td>Power (% pV˙O2peak)</td>
<td>100</td>
<td>87.5 ± 11.0b (mean power)</td>
<td>76.6 ± 10.3b</td>
</tr>
<tr>
<td>V˙O2 (L min⁻¹)</td>
<td>180 ± 18</td>
<td>175 ± 21</td>
<td>166 ± 29b</td>
</tr>
<tr>
<td>V˙O2 (mL min⁻¹ kg⁻¹)</td>
<td>71.8 ± 4.3</td>
<td>72.0 ± 6.2</td>
<td>68.5 ± 5.4b</td>
</tr>
<tr>
<td>HR (beat min⁻¹)</td>
<td>186 ± 11</td>
<td>183 ± 11</td>
<td>181 ± 10</td>
</tr>
<tr>
<td>SV (mL beat⁻¹)</td>
<td>157 ± 31</td>
<td>158 ± 38</td>
<td>150 ± 48</td>
</tr>
<tr>
<td>CO (L min⁻¹)</td>
<td>28.0 ± 5.4</td>
<td>27.9 ± 5.4</td>
<td>26.9 ± 9.7</td>
</tr>
<tr>
<td>a-vO2diff (mL 100 mL⁻¹)</td>
<td>12.6 ± 3.0</td>
<td>12.9 ± 2.1</td>
<td>13.9 ± 2.2</td>
</tr>
<tr>
<td>[La] (mmol mL⁻¹)</td>
<td>7.1 ± 1.6</td>
<td>12.7 ± 2.0a</td>
<td>11.1 ± 1.9a</td>
</tr>
</tbody>
</table>

HR: maximal heart rate; pV˙O2peak: power at maximal oxygen consumption. Incr.: incremental exercise; SS@p50 and SS@V˙O250: time to exhaustion at constant power and V˙O2 steady-state; *p < 0.05: significant difference between incremental and constant workload or V˙O2 steady-state exercises; p < 0.05: significant difference between constant workload and V˙O2 steady-state exercises.
old, alveolar V\(_{\text{O}_2}\) on-kinetics can be taken as a rather close approximation of skeletal muscle V\(_{\text{O}_2}\) on-kinetics. Experiments conducted on the isolated in situ dog gastrocnemius preparation have shown that, during transitions from rest to approximately 70% of the muscle peak V\(_{\text{O}_2}\), convective oxygen delivery to muscle, intramuscular blood flow and peripheral oxygen diffusion are not limiting factors for skeletal muscle V\(_{\text{O}_2}\) on-kinetics. It is suggested that the limiting factors for skeletal muscle V\(_{\text{O}_2}\) on-kinetics may vary according to the intensity of muscular contractions or of exercise [17]. In the present study, the power sustained during the third minute of both constant exercises (87.4 ± 3.0% of pV\(_{\text{O}_2}\)peak) was slightly higher compared to pSV\(_{\text{max}}\)
The maximal CO and SV values were reached in SS@pD50 and SS@V̇O2D50 tests. Above the lactate threshold, the increase in blood flow and a greater muscle oxygen extraction allowed the maintenance of a constant muscular O2 supply. Therefore, the HR increase would increase the blood flow in order to maintain arterial O2 pressure and muscle oxygenation during prolonged exercise. Conversely, Gonzalez-Alonso et al. [15] reported that prolonged submaximal exercises induced dehydration which appeared largely related to the reduction in blood volume in endurance-trained cyclists. Therefore, the interaction of dehydration with heart rate acceleration would be reduced ventricular filling leading to a SV decrease [10].

Nassis et al. [24] have also shown a moderate correlation between the one-leg quadriceps muscle mass and SV decline in cycling, implying that individuals with a large thigh muscle mass would present a smaller SV decline in prolonged cycling exercise. Nonetheless, our results demonstrate that the SV maintains its maximal value in SS@pD50 and SS@V̇O2D50 tests. Previously, Goodman et al. [16] showed a significant HR drift from 141 ± 2 to 154 ± 3 beats·min⁻¹ associated with a small decline (8%) in end-diastolic volume after 150-min pedalling exercise at 60% of V̇O2peak. They concluded that prolonged exercise at moderate intensity may not induce cardiac fatigue. Jensen-Urstad et al. [18] also reported a parallel increase in SV and left ventricular end-diastolic volume in elite runners with increasing workloads. However, the great systolic function in endurance-trained reflected a myocardial contractility increase contributing to the maintenance of a large SV during exercise [18]. Therefore, our results speculated that the increase in heart rate would be compensated in part by a decline of ventricular end-diastolic volume in order to maintain constant the stroke volume value. Finally, the time spent at the maximal CO and HR values is correlated with time to exhaustion at V̇O2D50. To our knowledge, no study has yet shown a significant relationship between time spent at the maximal CO value and time to exhaustion. Previously, many studies focused on the relationship between time spent at V̇O2peak and the time to fatigue [4]. The endurance time at V̇O2peak can be realistically modelled by a power/time curve, which allows estimation of the velocity for which endurance at V̇O2peak is the longest [5]. Though the time spent at V̇O2peak depends on a balance between the time to reach V̇O2peak and the time to exhaustion, no relationship was found between the time spent at V̇O2peak and the time to fatigue [4]. In the present study, the longer endurance time in SSVO2D50, in the presence of comparable values for cardiac output during both exercise conditions.

\[ r = 0.75, p = 0.006 \]  
\[ n = 11 \]

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**Fig. 2** Time to fatigue increase (Δ time) – mean power decrease (Δ mean power) relationship between constant power (pD50) and VO2 steady-state (VO2D50) exercise tests.

**Fig. 3** Relationship between the time spent at the maximal heart rate value and the time to exhaustion in constant power (pD50) and VO2 steady-state (VO2D50) tests.

**Fig. 4** Relationship between the time spent at the maximal cardiac output value and the time to exhaustion in constant power (pD50) and VO2 steady-state (VO2D50) tests.

ditions, argues that fatigue is a peripheral effect, perhaps designed to protect the muscle from developing an unfavorable interior milieu. In fact, our data demonstrated a prolonged maintenance of maximal cardiac output despite reduction in muscular power output. This, in turn, suggests thatafferent feedback to regulate exercise is more likely to come from receptors in the muscle than from centrally located receptors designed to protect the myocardium.

Conclusion

The present study showed that the control of the power output by \( \dot{V}O_2 \) induced a longer time spent at maximal CO and HR values without reaching \( \dot{V}O_2 \)peak. Therefore, the decrease in power to sustain \( \dot{V}O_2 \) steady-state could reflect the effect of time on the decrease in muscular efficiency. Alternatively, the time spent at maximal HR and CO did not seem to be the limiting factor between two exercises of different power output. However, it could be at a given mechanic or metabolic power output. Thus, the cause of fatigue does not seem to have the same origin during exhaustive supra-lactate threshold exercise under control of the myocardium.

References

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