The Effects of Interval Training on Oxygen Pulse and Performance in Supra-threshold Runs

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Abstract

The aim of this study was to examine (i) the effects of a severe interval training period on oxygen pulse kinetics (O₂-p, the ratio between VO₂ and heart rate), and (ii) to study the consequences of these effects on the variation of performance (time to exhaustion) during severe runs. Seven athletes were tested before and after an eight-weeks period of a specific intermittent training at v₅₀, i.e., the intermediate velocity between the lactate threshold (vLT) and the velocity associated with VO₂max (vVO₂max). During the test sessions, athletes performed an incremental test and an all-out test at the pretraining v₅₀. After the training period they also completed an additional all-out test at the posttraining v₅₀ (v₅₀bis). Results showed that after training there was i) an increase in the O₂-p maximal value during the incremental test (22.7 ± 1.5 mlO₂.b⁻¹ vs. 20.6 ± 1.5 mlO₂.b⁻¹; p < 0.04), ii) a decrease in the time to reach the O₂-p steady state (TRO₂-p) at the same absolute v₅₀ (33 ± 7 s vs. 60 ± 27 s; p < 0.04) and iii) an increase in the O₂-p steady state duration (TSSO₂-p) at the same absolute v₅₀ (552 ± 201 s vs. 407 ± 106 s; p < 0.04). However, there was no relationship between the improvement of these two O₂-p kinetics parameters (TRO₂-p and TSSO₂-p) and those of the performance. This study found that after an individualised interval-training program conducted at the same absolute velocity, the O₂-p kinetics reached a steady state quicker and for a longer duration than before training. This is however not related with the improvement of performance.

Keywords: Oxygen pulse, oxygen uptake, training, running.

Introduction

Several studies showed that improved performance after a training period is not necessarily accompanied by a significant modification of the maximal oxygen uptake (VO₂max) or the lactate threshold (vLT) (Flynn et al., 1994; Billat et al., 1999). Thus, in addition to the monitoring of heart rate (HR) or VO₂, the analysis of a third cardio-vascular parameter may deepen understanding of the relationship between modification of these parameters and the improvement of the performance. It is well know that the stroke volume (SV) is the major function of VO₂max and then of the performance in exercise lasting more than two minutes (Billat et al., 2001a for marathon; di Prampero et al., 1993, for middle distance). Furthermore, it has been reported that in well endurance-trained subjects, SV increase until VO₂max in an incremental exercise (Billat et al., 2001b; Gledhill et al., 1994; Zhou et al., 2001) and is increased by high intensity training (Billat et al., 2001a; Wilmore et al., 2001). However, the measurement of SV at high intensity cannot be done on the track and field, where athletes are currently tested thanks to advent of the portable gas exchange analyser. According to the Fick equation, VO₂ is the product of the cardiac output (Q) and the arteriovenous O₂ difference ((a-vO₂)D). Thus, the oxygen pulse (O₂-p, the ratio between VO₂ and HR) estimates the product of the stroke volume (SV) and (a-vO₂)D. HR and VO₂ are the two most commonly cardio-pulmonary parameters measured easily and reliably on field and laboratory (Léger et Thivierge, 1988). However, several authors have studied O₂-p and the theoretical relationship with SV and (a-vO₂)D. They suggested (Mahler et al., 1985; Sagiv, 1991; Whipp et al., 1996) or showed (Bhambhani et al., 1994; Bhambhani, 1995) an important correlation between the evolution of SV and O₂-p, and other (Hossack et al., 1980) found a strong relationship between (a-vO₂)D and O₂-p during maximal treadmill exercise. The theoretical relationship between O₂-p, SV and (a-vO₂)D was therefore confirmed and seems to be reliable. Hence, the interest to study the O₂-p kinetics is

Accepted: 29 May, 2003

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to appraise the evolution of the product of SV and (a−vO₂)D. Consequently, using simply the VO₂ and HR measurement, O₂-p allows to get a third information on the cardiopulmonary response in addition to VO₂ and HR. On the other hand, different studies showed that the parameters of VO₂ kinetics may be modified after a training period (Hagberg et al., 1980; Casaburi et al., 1987; Yoshida et al., 1992), and that some of these modifications are correlated to the performance. We hypothesize also that the O₂-p kinetics during a severe intensity exercise is modified after a training session, and that these modifications are correlated with the performance.

Therefore, the aim of this study was (i) to examine the effect of an eight-weeks severe endurance interval training program on the O₂-p kinetics during running, and (ii) to examine the consequences of these modifications on the performance during runs of severe intensity.

Materials and methods

Subjects

Seven endurance-trained male (25.3 ± 4.5 years, 175.0 ± 1.0 cm, 69.0 ± 4.0 kg and 60.6 ± 4.4 ml O₂.kg⁻¹.min⁻¹; mean ± SE) volunteered to participate in this study. They specialised in middle and long-distance running. All gave written voluntary informed consent in accordance with the guidelines of the University of Paris.

Experimental design

The test sessions were completed on a 400-m covered track before and after an eight-weeks period of a specific endurance interval-training. Before training, each test session consisted of an incremental test and an all-out test at vD₅₀, the velocity midway between the velocity associated with the lactate threshold (vLT) and the velocity associated with VO₂max (vV₂max). After training, subjects completed the same incremental and all-out tests, plus an additional all-out test at vD₅₀bis, the velocity calculated from the post-training vLT and the post-training vVO₂max. The maximal oxygen uptake (VO₂max), vVO₂max, vLT, vD₅₀ and the running economy (RE, defined as the rate of oxygen uptake for a given submaximal running velocity, Cavanagh & Williams, 1982) were determined during the incremental test. The all-out tests were carried out to determine the time to exhaustion (Tₑₓ). Velocity was regulated with an audio-visual system and strictly controlled by photoelectric cells (Brower Timing Systems, USA, Utah, Salt Lake City). Each subject performed tests at the same time of day, in a climate-controlled environment.

Procedures

During each test, the respiratory and pulmonary gas exchange variables were measured using a breath-by-breath portable gas analyser (Cosmed K4b², Roma, Italy), which was calibrated before each test according to the manufacturer's instructions (Hausswirth et al., 1996). Breath-by-breath data were later reduced to 5-s stationary averages (Data Management Software, Cosmed, Rome, Italy). Fingertip capillary blood samples were collected into a capillary tube, and were analysed for lactate concentration using a Doctor Lange analyser (GmbH, Berlin, Germany), which was calibrated before the tests. HR was monitored by the breath-by-breath gas analyser (Cosmed K4b², Roma, Italy) from the signal transmitted by a cardiofrequencemeter transmitter (Polar, PE 4000, Finland) adjusted on the chest of the athlete and was breath-by-breath measured. Thus, the O₂-p was calculated from these values.

The incremental test (3-min stages) was set to induce exhaustion within 20 min for each subject. Thus, velocity of the initial stage was set at the average velocity maintained over a 3000-m race (which has been described close to the VO₂max, Billat, 1996a) minus 6 km.h⁻¹. The velocity increment was set at 1 km.h⁻¹. All stages were followed by a 30-s rest period. A fingertip capillary blood sample was collected to measure the lactate concentration during each rest period, and in addition, before the test, immediately and 3 min after the test. VO₂max and HRmax were defined respectively as being

Fig. 1. Experimental design.
the highest 30-s oxygen uptake and beat values reached during the incremental test. VO2max was defined as the minimal velocity at which VO2max was achieved (Billat & Koralisztein, 1996b). In the case of the subject did not reached VO2max during the previously stage and if he stopped to run close to the half stage, VO2max was considered as being the median velocity between the last two stages (Kuipers et al., 1985). vLT was defined as the velocity associated with an increase in blood lactate concentration corresponding to 1 mmol.l⁻¹ which occurred at approximately 3.5 to 5 mmol.l⁻¹ (Aunola & Rusko, 1984). vΔ50 was defined as the velocity midway between vLT and vVO2max (Gaesser & Pool, 1996). Running economy (RE), defined as the rate of VO2 for a given submaximal work rate (Cavanagh & Williams, 1982), was calculated using rate of VO2 averaged between the second and the third minute of the stage run at 13 km.h⁻¹.

The aim of the all-out constant test at vΔ50 was to determine the time until exhaustion at vΔ50 and vΔ50bis, i.e., the time until the subject was unable to maintain the fixed velocity. Before each all-out test, subjects completed a 15-min warm-up period at 60% of VO2max then observed a 5-min rest period. Each subject was verbally encouraged. A fingertip capillary blood sample was collected before the test, immediately and 3 min after the test.

### Training program

Subjects were already well trained in endurance before their participation. They completed a eight-weeks endurance training program, which was composed of two interval-training sessions and three easy continuous running sessions per week. The exercise duration of the interval-training sessions was set at 50% of the time to exhaustion (Tlim) of the all-out pre-test at vΔ50 and the active recovery duration at 25%. Thus, the duration ratio was of 2/1. The intensity of the interval-training sessions was set at vΔ50 for exercise runs and 50% of the pretraining vVO2max for the active recovery. To set the number of repetitions of the interval-training sessions, subjects ran until exhaustion during the first and the eighth session in order to measure the maximal number of repetition (n). Thus, to vary and to avoid an overload of the interval-training sessions the number of repetitions of the other interval-training sessions was set at (n) minus 2 repetitions (n – 2) or 1 repetition (n – 1) during respectively the first or second interval-training session of each week. Intensity was unchanged during the training period, nevertheless, the training volume was fitted according to the improvements of each subject with an adjustment of (n) during the eighth interval-training session. The other sessions (recovery) were an easy run during one hour at 60–70% of the pre-training VO2max.

A professional trainer controlled each training session in order to be sure that these instructions were respected.

#### Oxygen pulse kinetics determination

The oxygen pulse (O2-p) kinetics was analysed with the software Sigmat Plot (SPSS, Chicago, IL, USA). Data were fitted to the mono-exponential equation based on the model of Linnarson (1974). According to Barstow et Mole (1991), the equation is as follows:

\[
y = y_0 + A \times (1 - e^{-t/\tau})
\]

with \(y_0\), the y value at time = 0; A, the amplitude of the mono exponential i.e., the \(y\) maximal value above \(y_0\); \(\tau\), the time constant of O2-p response defined as the time required to attain 63% of the O2-p steady state; \(t\), the time (s).

#### Calculation of the time to reach the O2-p steady state (TRO2-p)

If a 5% error is admitted in the determination of the O2-p value (the same error as for the determination of

### Table 1. Weekly training program.

<table>
<thead>
<tr>
<th></th>
<th>First week</th>
<th>Second week</th>
<th>Third week</th>
<th>Fourth week</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
</tr>
<tr>
<td>Tuesday</td>
<td>IT session: (n) intervals</td>
<td>IT session: (n - 1) intervals</td>
<td>IT session: (n - 2) intervals</td>
<td>IT session: (n - 1) intervals</td>
</tr>
<tr>
<td>Wednesday</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
</tr>
<tr>
<td>Thursday</td>
<td>IT session: (n - 2) intervals</td>
<td>IT session: (n - 2) intervals</td>
<td>IT session: (n - 1) intervals</td>
<td>IT session: (n - 2) intervals</td>
</tr>
<tr>
<td>Friday</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
<td>Easy continuous running</td>
</tr>
<tr>
<td>Saturday</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
</tr>
<tr>
<td>Sunday</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
</tr>
</tbody>
</table>

*Note* These four weeks of training has been repeated two times, with a reappraisal of the number \(n\) at the eighth interval training session (IT). Example of the IT session with a subject having a vVO2max of 18.5 km.h⁻¹, a vLT of 16 km.h⁻¹, a vΔ50 of 17.3 km.h⁻¹ and a \(t_{lim}\) at vΔ50 of 425 s.

During these sessions, the subject would run at a hard intensity during 213 s, whereas the length of the light runs was 107 s. The interval-training was run at 17.3 km.h⁻¹ for the hard runs and at 9.3 km.h⁻¹ for the light runs. Subjects completed \(n - 1\) or \(n - 2\) intervals, where \(n\) is the maximal repetition number that the subject is able to achieve; \(n\) was recorded during the first and the eighth interval-training sessions. The easy continuous running involved running during one hour under 13 km.h⁻¹.
As VO2, it may be accepted that a steady state in O2-p is attained above 95% of the asymptotic value reached during the test.

Thus, to calculate TRO2-p, Equation (1) was changed to the following:

\[ O_2-p(t) = O_2-p(base) + A \times (1 - e^{-\frac{t}{\tau}}) \]  

(2)

with O2-p(t) is the oxygen pulse as a function of time; O2-p(base), the oxygen pulse at rest; A, the amplitude of the mono exponential which corresponded to the maximal O2-p value above rest; \( \tau \), the slope; t, the time.

Given that the plateau value is equal to the maximal O2-p value reached during the test (O2-pmax), TRO2-p is equal to 95% O2-pmax. Moreover,

\[ 95\% O_2-p = 0.95 \left[ A + O_2-p(base) \right] \]

Therefore, by expressing t as a function of the other variables, Equation (2) was changed to the following:

\[ TA95\% O_2-p(max) = TR_{O2-p} = -\tau \times \ln \left[ 1 - 0.95 \times A - 0.05 \times O_2-p(base) \right] A \]

(3)

**Time spent at the O2-p steady state (TSSO2-p)**

This time corresponds to the duration of the plateau of the mono exponential. Thus, TSSO2-p is calculated by the difference between the total time and TRO2-p.

**Heart rate (HR) and VO2 kinetics**

HR and VO2 kinetics parameters were analysed according to the identical model of the O2-p kinetics replacing O2-p by HR and VO2 respectively.

**Statistical analysis**

To compare data before and after training, data were compared using a Student’s t-test for paired values and an analysis of variance with repeated measures. Results are presented as means and standard deviation (SD). The level of significance was established at 0.05.

### Results

**Training effect on velocities and physiological maximal values in incremental and vΔ50 test**

Training improved significantly vΔ50 in mean by 3% (17.6 ± 1 km.h\(^{-1}\) vs. 17.2 ± 1 km.h\(^{-1}\); p = 0.03) and running economy in mean by 4.7% (0.194 ± 0.01 ml.m\(^{-1}.kg^{-1}\) vs. 0.205 ± 0.02 ml.m\(^{-1}.kg^{-1}\); p = 0.04), whereas vVO2max and vLT remained unchanged (respectively 18.5 ± 0.9 km.h\(^{-1}\) and 16.1 ± 1.1 km.h\(^{-1}\)). The other aerobic fitness values were not significantly improved.

Training improved significantly O2-pmax during the incremental test (22.7 ± 1.5 ml.O2.b\(^{-1}\) vs. 20.6 ± 1.5 ml.O2.b\(^{-1}\); p = 0.048) and the amplitude of O2-p during the all-out test at v50 (13.4 ± 1.9 ml.O2.b\(^{-1}\) vs. 11.9 ± 2.7 ml.O2.b\(^{-1}\); p = 0.01). This last observation is the consequence of the decrease of the maximal HR value at vΔ50 (187 ± 6 bpm vs. 193 ± 4 bpm; p = 0.02) whereas the maximal VO2 and [Lmax] values were not significantly different.

**Training effect on the O2-p, VO2 and HR kinetics parameters at vΔ50 test**

Time to reach the O2-p steady state (TR_O2-p) and \( \tau \) were significantly lower after training (respectively 60 + 27 s vs. 33 ± 7 s; p = 0.04; and 24 + 10 s vs. 13 ± 3 s; p = 0.03). Thus, the O2-p steady state was reached earlier and was maintained in mean during 88 ± 4% of the time until exhaustion (T_ea) (between 6 ± 2% and 94 ± 3% of T_ea) vs. 80 ± 5% (between 13 ± 6% and 94 ± 3% of T_ea) before training. Time spent at the O2-p steady state (TSSO2-p) increased significantly after training (552 ± 201 s vs. 407 ± 107 s; p = 0.04). There was no significant difference in O2-p kinetic parameters to compare results of the vΔ50 test before training and the vΔ50bis test.

The HR and VO2 kinetics parameters during each all-out test (before and after training) are presented in Table 5.

### Table 2. Training effects on the aerobic fitness parameters.

<table>
<thead>
<tr>
<th></th>
<th>vVO2max (km.h(^{-1}))</th>
<th>vLT (km.h(^{-1}))</th>
<th>%vVO2max</th>
<th>vΔ50 (km.h(^{-1}))</th>
<th>%vVO2max</th>
<th>RE at 13 km.h(^{-1}) (ml.kg(^{-1}.m^{-1}))</th>
<th>T_ea at vΔ50 (s)</th>
<th>T_ea at vΔ50bis (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before training</td>
<td>18.5 ± 0.9</td>
<td>16.1 ± 1.1</td>
<td>87 ± 2</td>
<td>17.2 ± 1</td>
<td>93 ± 1</td>
<td>0.205 ± 0.02</td>
<td>509 ± 131</td>
<td>–</td>
</tr>
<tr>
<td>After training</td>
<td>18.6 ± 0.9</td>
<td>16.2 ± 1.0</td>
<td>87 ± 3</td>
<td>17.6 ± 1(^{\dagger})</td>
<td>94 ± 3</td>
<td>0.194 ± 0.01(^{\dagger})</td>
<td>617 ± 201</td>
<td>470 ± 78*</td>
</tr>
<tr>
<td>Test t</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>p = 0.03</td>
<td>NS</td>
<td>p = 0.04</td>
<td>NS</td>
<td>p = 0.04</td>
</tr>
</tbody>
</table>

Note: VO2max, maximal oxygen uptake; vVO2max, velocity associated with VO2max; vLT, velocity associated with the lactate threshold (LT); vΔ50, median velocity between vLT and vVO2max; vΔ50bis, the velocity calculated from the post-training vLT and the post-training vVO2max; RE, running economy; T_ea, time to exhaustion. NS: not significantly different. \(^{\dagger}\) Significant difference between before and after training. * Significant difference between the test at the new vΔ50 and the one at old vΔ50 after training.
Results of the training effects were similar to VO₂ and HR kinetics parameters. Thus, during the vΔ50 test, time to reach the steady state (TR) was significantly lower in HR (104.2 ± 41.6 vs. 59.6 ± 19.4; p < 0.05) and VO₂ (109.5 ± 11.2 s vs. 71.8 ± 25.7) after training that before training. The same evolution was observed in τ of HR (42.9 ± 18.5 vs. 25.4 ± 7.7; p < 0.05) and VO₂ kinetics (40.4 ± 5.5 s vs. 26.5 ± 9.9 s; p < 0.05). Identical results were observed when compared results of vΔ50 test before training and result of vΔ50bis test (Table 5).

Relationship between the training effect on cardio respiratory parameters and the performance (Tex at vΔ50)

There was no relationship between the modifications in O₂-p kinetics and the improvement in Tex at vΔ50. There was no correlation between the improvement in RE and the improvement in Tex at vΔ50 (r = −0.459; p = 0.321) and between RE and the other variables measured in this study. There was also no correlation between the O₂-p kinetics parameters and the VO₂ kinetics parameters as well as between the O₂-p kinetics parameters and the HR kinetics parameters.

Discussion

The main results of this study showed that: i) endurance training accelerated the oxygen pulse (O₂-p) kinetics; ii) and there was no relationship between the modification in the O₂-p kinetics and the increase in endurance.

The O₂-p kinetics

O₂-p may represent a third cardiopulmonary information in addition to VO₂ and HR (i.e., the product of the stroke
volume (SV) and the arteriovenous O₂ difference (a-vO₂D) during tests on the track and field. This is true only if changes in O₂-p kinetics after training do not reflect the modifications in VO₂ or HR kinetics. Thus, three cases may occur: the first would be that only the HR kinetics is modified with training, the second would be that only the VO₂ kinetics is modified with training and the third would be that both VO₂ and HR kinetics are modified with training. The two first cases imply that the modification of the O₂-p kinetics correlates and reflects only the modification of HR or VO₂ kinetics. Thus, in this case the interest to study the O₂-p kinetics is poor. On the other hand, the third case supposes both variables (VO₂ and HR) are modified with training. Thus, the O₂-p kinetics modifications do not correlated with any kinetics of the sensible variables. Only in that case O₂-p brings new information compared with the HR or VO₂ kinetics. In this study, none of O₂-p kinetics parameters are correlated with HR or VO₂ kinetics parameters, which confirm the interest to study the O₂-p kinetics. We can therefore conclude that the O₂-p kinetics is no redundant with the study of HR or VO₂ kinetics, and that O₂-p kinetics is the reflect of the product of SV and (a-vO₂D). However, a next study would be carried out to determine the role of these two variables in the O₂-p kinetics while measuring them.

To our knowledge, only one publication (Mille-Hamard et al., 2000) has studied the O₂-p kinetics during a constant and severe intensity exercise but without training period. Mille-Hamard et al. (2000) have studied the O₂-p kinetics during an all-out test at 95% vVO₂max and they showed that the time spent at the O₂-p steady state (TSSO₂-p) was included between 10 and 90% of the time to exhaustion (T_ex). Indeed, they did not study the effects of an endurance-training period on TSSO₂-p and on the modification of the kinetics parameters.

The present study showed that after a eight-weeks period of interval-training and during a test at vΔ50, TSSO₂-p was increased while starting earlier. Thus, we find that after training subjects maintained TSSO₂-p from 6% (± 2) to 94% (± 3) of T_ex (vs. 13 ± 6% and 94 ± 3% of T_ex before training). The time to reach the O₂-p steady state (TRO₂-p) and τ decreased significantly during the vΔ50 test after training, but this two kinetics parameters were very similar and no significantly different during exercise at vΔ50bis. Thus, in accordance with Sietsema et al. (1989), results showed that the endurance training influenced significantly de O₂-p kinetics and that the O₂-p kinetics in response to exercise is dependent on work rate and also influenced by the fitness of the subject (characterized by peak VO₂ or anaerobic threshold values). Finally, in accordance to different longitudinal studies (Kasch et al., 1973; Mahler et al., 1985), we could have observed an increase in O₂-pmax after eight weeks of endurance training during the incremental test.

### Relationship between T_ex and O₂-p kinetics

In the present study, O₂-pmax was increased after training. Thus, the specific interval training allowed to improve specifically vΔ50 and O₂-p kinetics. Other studies may be carried out to evaluate the role of SV and (a-vO₂D) in the modification of the O₂-p kinetics. T_ex at vΔ50 was improved after training (617 ± 201 vs. 509 ± 131 s before training), but not significantly, due to a large SE. This increase of T_ex was 109 s in average, which represents an average increase of 23%. However, the modifications of the O₂-p kinetics parameters were not correlated with the improvement of performance after training at vΔ50. Billat et al. (1994) showed that the measurement of T_ex at the maximal aerobic speed (MAS)
in a laboratory setting was reproducible for a group of runners at 1-week interval and that the average value could be used for studying the effects of training, tapering, and/or different warm-up procedures in a group of runners. Furthermore, they showed that the time spent at the VO₂ steady state was reproducible at 1-week interval. Thus, it cannot be excluded that Tex at vΔ50 could be related to the improvement of the O₂-p kinetics or to other bioenergetics characteristics in a wider sample of runners. The improvement in Tex could be explained also by the improvement in RE. However, regarding the effects of training on RE, some studies reported no difference (Overend et al., 1992; Ramsbottom et al., 1989), whereas others found an improvement in RE (Wilcox & Bulbulian, 1984; Kirwan et al., 1988) as result of training. Recently, a study showed an improvement in RE after six weeks of plyometric training (Turner et al., 2003), whereas other studies showed also an improvement in RE but after an endurance training (Billat et al., 1999). Thus, responsible mechanism of the improvement in RE must still be determined but are out of the scope of this study. Further researches are still necessary to specify the mechanisms of running economy improvements. Thus, given results of the present study, it is not possible to hypothesize objectively a peripheral improvement as result of training.

**Role of SV and (a–vO₂)D in the O₂-p kinetics**

According to the Fick equation, the O₂-p is the product between stroke volume (SV) and arterio-venous oxygen difference (a–vO₂)D. Our results showed that O₂-pmax determined during the incremental test increased significantly after training while HR and VO₂max remained unchanged. The increase in O₂-pmax could be explained by three different phenomena: the first would be that both SV and (a–vO₂)D increased; the second would be that only SV increased; and the last would be that only (a–vO₂)D increased. It is difficult to estimate how SV and (a–vO₂)D evolved during training because one limitation of the present study was that SV and (a–vO₂)D are not measured. Bhambhani et al. (1994) and Hossack et al. (1980) have shown that during cycle or treadmill exercises in trained as well as in untrained subjects, O₂-p was significantly correlated with SV, but not necessarily with (a–vO₂)D. Moreover, Wolfe et al. (1992) have showed that training during 11 weeks increased SV significantly whereas (a–vO₂)D was not increased. Therefore, many authors (Sagiv et al., 1991; Wolfe et al., 1992; Bhambhani et al., 1994; Bhambhani, 1995; Whipp, 1996) suggested that O₂-p may be a good means of appraising indirectly SV during a submaximal exercise in trained and untrained subjects since O₂-p seems poorly influenced by the variability of (a–vO₂)D: according to Whipp et al. (1996), SV may be estimated as 5 times the slope of the VO₂-HR relationship (where 5 is...
approximately 1/\text{CaO}_2). In this context, we hypothesised that the improvement in O_2-p is mainly due to the increase in SV in the present study, but other studies measuring SV and (a-V_O_2)p during severe exercises, should be carried out to confirm the validity of O_2-p in order to predict SV.

In conclusion, the present study showed that after endurance training, O_2-p kinetics was faster and the steady state was sustained for a longer period during an all-out test at vΔ50, and also both for HR and VO_2. Secondly, there was no relationship between modification of the O_2-p kinetics and improvement of the performance during exercise at vΔ50. The hypothesis according to which the improvement of the O_2-p kinetics reflects mainly the improvement of SV may be validated with SV measured during severe exercise before and after a training session. These measures are today possible by the non invasive kinetics (Charloux et al., 2000; Christensen et al., 2000).

Acknowledgements

This study was supported by grants from Caisse Centrale des Activités Sociales d’Electricité et Gaz de France.

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