Passive versus Active Recovery during High-Intensity Intermittent Exercises

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ABSTRACT

DUPONT, G., W. MOALLA, C. GUINHOUYA, S. AHMAIDI, and S. BERTHOIN. Passive versus Active Recovery during High-Intensity Intermittent Exercises. Med. Sci. Sports Exerc., Vol. 36, No. 2, pp. 302–308, 2004. Purpose: To compare the effects of passive versus active recovery on muscle oxygenation and on the time to exhaustion for high-intensity intermittent exercises. Methods: Twelve male subjects performed a graded test and two intermittent exercises to exhaustion. The intermittent exercises (15 s) were alternated with recovery periods (15 s), which were either passive or active recovery at 40% of VO₂max. Oxyhemoglobin was evaluated by near-infrared spectroscopy during the two intermittent exercises. Results: Time to exhaustion for intermittent exercise alternated with passive recovery (962 ± 314 s) was significantly longer (P < 0.001) than with active recovery (427 ± 118 s). The mean metabolic power during intermittent exercise alternated with passive recovery (48.9 ± 4.9 mL·kg⁻¹·min⁻¹) was significantly lower (P < 0.001) than during intermittent exercise alternated with active recovery (52.6 ± 4.6 mL·kg⁻¹·min⁻¹). The mean rate of decrease in oxyhemoglobin during intermittent exercises alternated with passive recovery (2.9 ± 2.4%·s⁻¹) was significantly slower (P < 0.001) than during intermittent exercises alternated with active recovery (7.8 ± 3.4%·s⁻¹), and both were negatively correlated with the times to exhaustion (r = 0.67, P < 0.05 and r = 0.81, P < 0.05, respectively). Conclusion: The longer time to exhaustion for intermittent exercise alternated with passive recovery could be linked to lower metabolic power. As intermittent exercise alternated with passive recovery was characterized by a slower decline in oxyhemoglobin than during intermittent exercise alternated with active recovery at 40% of VO₂max, it may also allow a higher reoxygenation of myoglobin and a higher phosphocreatine resynthesis, and thus contribute to a longer time to exhaustion. Key Words: NEAR-INFRARED SPECTROSCOPY, TIME TO EXHAUSTION, OXYGEN UPTAKE, INTERVAL TRAINING

Short intermittent exercises are frequently used in training programs in order to improve maximal oxygen uptake (VO₂max: 19,21,35) and/or anaerobic capacity (35). The performance and physiological adaptations associated with these exercises depend on the interaction between different parameters such as exercise intensity, recovery type, exercise and recovery periods, and number of repetitions. The recovery type, which can be either active or passive, represents one of these variables. For short intermittent exercises, it has been recommended to introduce active recovery between exercises rather than passive recovery in order to decrease blood lactate concentration ([La]ₜ; 8.9) and thus to increase time to exhaustion (TTE). This assumption was based on the fact that active recovery enhances blood lactate removal in comparison with passive recovery (11,22,36). However, for short intermittent runs of 15 s at 120% of maximal aerobic speed, alternated with either 15 s of passive recovery or active recovery at 50% of maximal aerobic speed, it has been reported that TTE with passive recovery was longer than with active recovery (18). The latter hypothesized that passive recovery allowed a higher reoxygenation of myoglobin and hemoglobin, and a higher phosphocreatine (PCr) resynthesis than active recovery. For short intermittent exercise, oxyhemoglobin (HbO₂) variations have already been analyzed using near-infrared spectroscopy (NIRS), and it has been demonstrated that such variations depended on exercise duration and intensity. Christmass et al. (15) reported that the decline in muscle HbO₂ during long intermittent exercise (work:recovery ratio of 24s:36s) was significantly greater than for short intermittent exercises (work:recovery ratio of 6s:9s). These results showed that the contrast in HbO₂ between the two intermittent exercises was linked to the duration of the work period, as suggested by Åstrand et al. (1) in 1960. Bae et al. (2) found that the deoxygenation level was dependent on exercise intensity, as the decreased oxygenation level was significantly lower in an intermittent exercise at 100% of the power eliciting VO₂max than during an intermittent exercise.
at 95% of the ventilatory threshold. However, to our knowledge, no study has focused on the effects of recovery type on the muscle HbO2 variations and its possible influence on the TTE, especially for short intermittent exercises.

This study was designed: 1) to compare TTE using a cycle ergometer for short intermittent exercise (15 s) interspersed with passive recovery (IE-PR) and short intermittent exercise (15 s) interspersed with active recovery at 40% of VO2max (IE-AR) and 2) to compare HbO2 variations using NIRS between IE-PR and IE-AR. We hypothesized that TTE would be longer for IE-PR than for IE-AR and that the deoxygenation during IE-PR would be slower than during IE-AR.

METHODS

Subjects

Subjects were 12 male physical education students specialized in soccer, who trained from 3 to 5× wk⁻¹ and performed one match per week. Written voluntary consent to participate was obtained from all subjects after informing them of the purpose of the experiment, the procedure, and the possible risks. This investigation received the approval of the local Consultative Committee for the Protection of Persons in Biomedical Research. Their age, height, and body mass were 24.3 ± 4.1 yr, 175.8 ± 6.7 cm, and 71.1 ± 7.2 kg, respectively. Their percentage body fat, estimated from a calibrated bioelectrical impedance balance (Tanita TBF 543), was 15.8 ± 3.8% (range 7.9–19.5%).

Experimental Protocol

Subjects came to the laboratory for eight visits. During a first visit they received a medical examination and were familiarized with experimental equipment and protocol. During the second and third visits, they performed two then three 10-min constant power tests, respectively. Three pretests were performed during the next three visits: a maximal graded test, a force-velocity test, and a Wingate anaerobic test (WanT). Then, in a random order, the subjects completed two intermittent exercises until exhaustion, one alternated with passive recovery, the other with active recovery at 40% of VO2max. For each test session, the subjects were required to consume their last meal at least 3 h before the test and were asked to refrain from smoking and consuming beverages containing caffeine. All tests were conducted on a cycle ergometer (Monark 814E, Varberg, Sweden) at the same time of day and were separated by a minimum of 2 d. Subjects were instructed to grip the handlebars and not to lift off the saddle while cycling. The subject’s feet were firmly strapped to the pedals, and the saddle height was adjusted to allow a slight flexion of the knee at the lowest level of the pedal cycle. During the constant power tests, the graded test and intermittent exercises, the cranking velocity was set at 60 rev-min⁻¹ (rpm). The constant power tests (60, 70, 80, 90, and 100 W) were performed in a random order and were separated by a 10-min recovery period. These tests were carried out to calculate the individual VO2 versus power relationship and then to estimate the power associated with 40% of VO2max.

A standardized warm-up, consisting of 6-min pedaling at 60 W followed by three bouts of 30s at 80, 100, and 120 W separated by 30 s of rest, preceded the force-velocity test, the WanT, and the two intermittent exercises.

Pretests

The graded test aimed to determine VO2max and power output at 100% of VO2max. During this test, the subjects were asked to maintain a cycling cadence of 60 rpm. Subjects performed a 2-min warm-up at 60 W and then the power output was increased by 30 W every minute until exhaustion. For this test, the subjects were asked to exercise for as long as possible. They were considered as exhausted when they could not maintain the required frequency despite vigorous encouragement. The power at the last completed stage was retained as the power output at 100% of VO2max. For the force-velocity test and the WanT, the cycle ergometer was interfaced with a computer to measure the cranking velocity. Six magnetic interrupters, placed on the cycle ergometer every 60°, were activated by a magnet positioned on the pedal crank. The signal thus generated was transmitted to a computer to calculate cranking velocity. The signal was analyzed with JFB software (JFB International Medical, Bessanay, France). The force at which the WanT was performed was set according to the procedure proposed by Vandewalle et al. (37). For Inbar et al. (25), this procedure seemed to be optimal for braking force settings on an individual basis. The force-velocity test consisted of short maximal sprints (about 6 s) against different braking forces. The peak velocity was recorded and used to calculate the force-velocity relationship. The test began with a braking force equal to 9.81 N (load of 1 kg). After a 5-min recovery, the braking force was increased by 9.81 N (1 kg), then the same exercise was repeated with an increasing braking force until the subjects were unable to reach a peak velocity higher than 100 rpm. The first two bouts (with the two lowest braking forces) served as warm-up and learning exercises; they were performed again at the end of the test. The maximum theoretical isometric force (F0) was the x intercept of the force-velocity relationship. The WanT consisted in pedaling for 30 s as fast as possible against a braking force corresponding to 0.5 F0 (38). This test aimed to determine the mean anaerobic power output (i.e., the mean power output sustained over the 30-s exercise period).

Intermittent Exercises

These consisted of repeating exercises for 15 s at a high intensity alternated with 15 s of passive (IE-PR) or active recovery (IE-AR) until exhaustion. For these exercises, the subjects were asked to exercise for as long as possible. They were considered as exhausted when they could not maintain the required frequency despite vigorous encouragement. As recommended by Barnett et al. (4), to decrease the variance
in TTE in comparison with intensity based solely on VO$_2$max for exercises at high intensities, the target power output was calculated from the mean anaerobic scope (i.e., the difference between the mean power output determined during the WanT and the power output at 100% VO$_2$max determined during the graded test). This involved adding 20% of the mean anaerobic scope to the power output at 100% VO$_2$max.

The braking force applied on the cycle ergometer wheel was adjusted according to the desired power, whereas the cycling frequency was identical (60 rpm) for the two intermittent exercises and for the active recovery. For IE-PR, the experimenter manually adjusted the braking force applied on the wheel, within 1 s, during the transitional periods between exercise and recovery. The active recovery was carried out at 40% VO$_2$max. The active recovery intensity was chosen to be within the range of those recommended to remove blood lactate concentration (7,30). Times to exhaustion were measured for IE-AR and IE-PR. Recovery periods were included in the TTE.

**Measurements**

**Near-infrared spectroscopy.** Changes in tissue HbO$_2$ were estimated using a near-infrared spectroscopy device (NIRS; RunMan Unit, NIM, Philadelphia, PA) during the two intermittent exercises. This unit has been previously validated based on the linearity of the signal output and the relationship to O$_2$ concentration in human blood (5,13). The measurement principle and the technical characteristics of the device have been described previously (5,13). The NIRS consists of an optical detector and a light source using two wavelengths at 760 and 850 nm. The difference in recovered light at these two wavelengths provides a relative measure of changes in HbO$_2$ and deoxyhemoglobin. The major NIRS signal in human tissues originates for about 90% from hemoglobin, whereas the contribution of myoglobin to the signal is considered to be minimal, about 10% (27,33). To record the NIRS signal, the probe was placed over the right vastus lateralis muscle, approximately 14–20 cm from the knee joint along the vertical axis of the thigh. A permanent marker was used to identify the location on the skin where the probe was placed in order to put it on the same area during the second intermittent exercise. The NIRS probe was protected from skin moisture by transparent adhesive tape and a strap was wrapped around the thigh to prevent displacement during exercise. Before beginning the intermittent exercise, subjects rested on the cycle ergometer and the NIRS unit was calibrated according to the manufacturer’s specifications and protocol. The electrical output of the NIRS unit was adjusted by initially setting the balance at 0 ± 10 mV and then by adjusting the gain control between 600 and 1000 mV at 760 nm. The absorbency measurements were then checked at 850 nm to ensure that the values were negative in value and within 10% of those at 760 nm. A settling time of approximately 30 s was allowed at each wavelength to allow for stability of the readings (31). Calibration procedures were repeated until the NIRS signal output stabilized at the baseline level. At the completion of each intermittent exercise, a blood pressure cuff was placed around the upper thigh and inflated to 280 mm Hg during a 10-min maximum to induce arterial occlusion. The saturation level was evaluated with a relative scale where 0% saturation was defined as the nadir reached during the end of occlusion period and 100% as the maximal saturation level recorded after release of the occlusion. The NIRS signal was collected and analyzed across 1-s periods. The decline in HbO$_2$ corresponded to the difference between the baseline values (the HbO$_2$ values at the beginning of exercises) and the lowest HbO$_2$ values (measured at the end of the intermittent exercises). For each 1-s period, the decrease in HbO$_2$ was expressed in percent per second, the mean rate of decrease in HbO$_2$ corresponded to the mean of these calculated values. As TTE was longer for each subject during IE-PR in comparison with IE-AR, the HbO$_2$ variations were also studied for IE-PR with a duration equal to the TTE of IE-AR (IE-PR2).

**Ventilation, respiratory gas exchanges and heart rate.** Respiratory gas exchange values were measured breath-by-breath using a portable system (Cosmed K4b$^2$, Rome, Italy) in order to determine ventilation (VE), oxygen uptake (VO$_2$), and carbon dioxide production (VCO$_2$). This analyzer has previously been validated for measuring these parameters over a wide range of exercise intensities (29). Respiratory gas exchanges and heart rate (HR; Polar Electro, Kempele, Finland) values were averaged either every 15 s for the graded test or every 5 s for intermittent exercises. Before each test, the O$_2$ and CO$_2$ analysis systems were calibrated using ambient air and with a gas mix of known O$_2$ and CO$_2$ concentrations. The calibration of the turbine flowmeter of the K4b$^2$ was performed using a 3-L syringe (Quinton Instruments, Seattle, WA). Maximal values corresponded to values obtained during the graded test, whereas peak values corresponded to the highest mean values attained in three successive 5-s periods during intermittent exercises. The VO$_2$max was defined as the highest VO$_2$ attained in two successive 15-s periods for the graded test. It was judged that subjects had reached their VO$_2$max when three or more of the following criteria were met: 1) a plateau in VO$_2$ despite increasing power, 2) a final respiratory exchange ratio (RER) higher than 1.1, 3) an inability to maintain the required power, 4) a lactate concentration higher than 9 mmol·L$^{-1}$, or 5) a HR above 90% of age-predicted maximum HR.

**Blood lactate concentration.** Three minutes after the graded test and the intermittent exercises, fingertip blood samples (10 μL) were collected in order to measure blood lactate concentrations ([La]$^b$) by a spectrophotometer technique (Dr. Lange, miniphotometer + LP20, type LPG 344), which had previously been validated (26). The blood samples were analyzed within 15 min of the end of the tests. The accuracy of the analyzer was checked by standard solutions in lactate concentration (2, 4, 10, 15, and 30 mmol·L$^{-1}$).
Metabolic Power. Overall metabolic power was calculated for the two intermittent exercises. The energy release per unit of time corresponded to the sum of aerobic and anaerobic lactic energy productions (17) and was calculated as follows:

\[ E/t = (\Sigma V\dot{O}_2/TTE) + \beta([La]_b/TTE) \]

where \( E/t \) was the overall metabolic power (i.e., the oxygen requirement in mL \( O_2 \cdot kg^{-1} \cdot min^{-1} \)), \( \Sigma V\dot{O}_2/TTE \) was the time integral of the \( V\dot{O}_2 \) curves (mL \( O_2 \cdot kg^{-1} \cdot min^{-1} \)), \( \beta \) was the amount of energy released from lactate production (about 3 mL \( O_2 \cdot kg^{-1} \cdot mmol^{-1} \); 17) and \([La]_b/TTE\) the rate of lactate accumulation in the blood (mmol L\(^{-1}\) \cdot min\(^{-1}\)).

Statistical Analysis

Results are expressed as means ± standard deviations. The normality distribution of the data was checked with the Kolmogorov-Smirnov test. The Student’s \( t \)-test was used for paired data to determine differences between the parameters measured or calculated during IE-PR and IE-AR. Regression analysis was used to examine the relationships between TTE and the rate of decline in HbO\(_2\) for the two intermittent exercises. The level for significance was set at \( P < 0.05 \).

RESULTS

Pretest. The \( V\dot{O}_{2\text{max}} \), \( HR_{\text{max}} \), RER, \( VE_{\text{max}} \), and \([La]_b\) obtained for the graded test were 55.0 ± 6.3 mL\( \cdot \)kg\(^{-1}\)\cdot min\(^{-1}\), 183.9 ± 13.8 beats\( \cdot \)min\(^{-1}\), 1.14 ± 0.02, 134.8 ± 31.3 L\( \cdot \)min\(^{-1}\), and 12.3 ± 1.5 mmol L\(^{-1}\), respectively. The power output at 100% of \( V\dot{O}_{2\text{max}} \) corresponded to 287.5 ± 34.9 W. The braking force (\( F_0 \)) eliciting a nil velocity during the subjects’ force-velocity test was 138.3 ± 15.5 N (14.1 ± 1.6 kg.) The braking force set for the WanT was 68.7 ± 7.8 N (7.0 ± 0.8 kg). The mean power output measured during the WanT was 641.5 ± 51.7 W.

Intermittent exercises. For intermittent exercises, the power output was set at 358.3 ± 37.0 W, or 124.9 ± 3.2% of the power output at \( V\dot{O}_{2\text{max}} \). The TTE, \([La]_b\), mean cardiorespiratory parameters, and metabolic power measured for intermittent exercises are presented in Table 1. The \( V\dot{O}_{2\text{peak}} \) elicited during IE-PR (55.5 ± 9.5 mL\( \cdot \)kg\(^{-1}\)\cdot min\(^{-1}\)) and during IE-AR (57.1 ± 7.0 mL\( \cdot \)kg\(^{-1}\)\cdot min\(^{-1}\)) were not significantly different from the \( V\dot{O}_{2\text{max}} \) measured during the graded test.

NIRS data. Figure 1 shows an example of HbO\(_2\) versus time relationship for the IE-PR and for IE-AR. The baseline levels, the lowest values, the declines in muscle HbO\(_2\), and the rates of decrease in HbO\(_2\) for IE-PR, IE-AR, and IE-PR2 are presented in Table 2. Significant relationships were found between TTE and the mean rate of decrease in HbO\(_2\) for IE-PR (\( r = 0.67; P < 0.05 \)) and for IE-AR (\( r = 0.81; P < 0.05 \)).

TABLE 1. Means ± SD for TTE, \([La]_b\), peak and average \( V\dot{O}_2\) values, and peak and average HR values for intermittent exercises alternated with active recovery and with passive recovery.

<table>
<thead>
<tr>
<th>Intermittent Exercises</th>
<th>Active Recovery</th>
<th>Passive Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTE (s)</td>
<td>426.8 ± 118.4</td>
<td>961.8 ± 314.3***</td>
</tr>
<tr>
<td>([La]_b) (mmol L(^{-1}))</td>
<td>12.6 ± 1.7</td>
<td>13.1 ± 2.7**</td>
</tr>
<tr>
<td>Peak ( V\dot{O}_2) (mL( \cdot )kg(^{-1})\cdot min(^{-1}))</td>
<td>57.1 ± 7.0</td>
<td>55.5 ± 9.9**</td>
</tr>
<tr>
<td>(% ( V\dot{O}_{2\text{max}}))</td>
<td>103.9 ± 8.3</td>
<td>100.7 ± 10.7</td>
</tr>
<tr>
<td>Average ( V\dot{O}_2) (mL( \cdot )kg(^{-1})\cdot min(^{-1}))</td>
<td>46.9 ± 5.8</td>
<td>46.1 ± 5.0**</td>
</tr>
<tr>
<td>(% ( V\dot{O}_{2\text{max}}))</td>
<td>85.3 ± 6.6</td>
<td>84.1 ± 7.4</td>
</tr>
<tr>
<td>Peak HR (bmp)</td>
<td>183.3 ± 13.9</td>
<td>182.5 ± 15.0**</td>
</tr>
<tr>
<td>(% HR(_{\text{max}}))</td>
<td>99.7 ± 3.3</td>
<td>99.2 ± 3.6</td>
</tr>
<tr>
<td>Average HR (bmp)</td>
<td>166.6 ± 11.9</td>
<td>165.6 ± 14.6**</td>
</tr>
<tr>
<td>(% HR(_{\text{max}}))</td>
<td>90.7 ± 4.5</td>
<td>90.1 ± 5.4</td>
</tr>
<tr>
<td>Metabolic power (mL( \cdot )kg(^{-1})\cdot min(^{-1}))</td>
<td>52.6 ± 4.8***</td>
<td>48.9 ± 4.9</td>
</tr>
</tbody>
</table>

*** Significantly different from IE-AR with \( P < 0.001 \).
** No significant difference.

FIGURE 1—An example of oxhemoglobin (HbO\(_2\)) variations versus time relationship during IE-PR and IE-AR for an individual subject.

DISCUSSION

The purpose of this study was to compare TTE and the HbO\(_2\) variations between IE-PR and IE-AR. It was hypothesized that TTE would be longer for IE-PR than for IE-AR and that the deoxygenation during IE-PR would be slower than during IE-AR. Results obtained confirm these hypotheses as TTE were significantly longer (\( P < 0.001 \)) for IE-PR (962 ± 314 s) than for IE-AR (427 ± 118 s) and the mean rate of decrease in HbO\(_2\) during IE-PR (2.9 ± 2.4% \( \cdot \)s\(^{-1}\)) was significantly lower (\( P < 0.001 \)) than during IE-AR (7.8 ± 3.4% \( \cdot \)s\(^{-1}\)). Consequently, as TTE was longer for IE-PR than IE-AR, a faster deoxygenation occurred with active recovery than with passive recovery. The results of the present study may appear to differ from the literature data, which have shown the interest of active recovery on performance (10,34). However, in these studies focusing on the comparison between active and passive recoveries, the exercise forms and the performance criterion were different from those performed in the present study (mean power output and peak power vs TTE). Signorile et al. (34) compared performance (peak power, fatigue rate, and total work) during a series of eight sprints of 6 s alternating with 30 s of active or passive recovery periods. Bogdanis et al.
(10) studied the effects of recovery type on power output through two 30-s sprints separated by 4 min. In the present study, the effects of recovery type were analyzed on TTE measured during intermittent exercises characterized by shorter recovery periods (15 s). In a previous study, Dupont et al. (18) found results similar to those in the present study. For short intermittent runs of 15 s alternated with either 15 s of passive recovery or 15 s of active recovery, TTE was significantly ($P < 0.001$) longer when passive recovery was performed ($745 \pm 171$ s $445 \pm 79$ s). To explain the longer time to exhaustion for intermittent runs alternated with passive recovery, the authors indicated that mean metabolic power was significantly lower ($P < 0.001$) for intermittent runs alternated with passive recovery than with active recovery. In addition, they hypothesized that for short intermittent exercises alternated with short recovery periods (15 s), the deoxygenation over the whole intermittent exercise would be slower during intermittent exercises alternated with passive recovery in comparison with active recovery. Results of the present study confirm this hypothesis by the use of NIRS signal.

The NIRS signal penetrates the skin, subcutaneous fat, and underlying muscle and is either absorbed or scattered within the tissue in order to measure muscle oxygenation (32). Several studies (12,28) have reported that the adipose tissue thickness has an effect on light propagation in muscles. In the present study, subjects were active soccer players and their estimated percentage body fat was 15.8% for untrained subjects (height 183 cm; body weight 74.4 kg; body mass index 22.3). Several studies (12,28) have reported that the adipose tissue thickness of the vastus lateralis did not alter the NIRS signal. Consequently, as the depth of penetration of the NIRS signal is about 2–3 cm, it could be assumed that the adipose tissue thickness of trained subjects ($V_{O2\text{max}} = 55.0 \pm 6.3$ mL·kg$^{-1}$·min$^{-1}$ in the present study) was sufficiently small so that the NIRS signal also reflected the oxygenation changes occurring mainly in the muscle tissue. In the present study, the baseline values, the lowest values, and the decline in $HbO_2$ were not significantly different between IE-PR and IE-AR. These results indicate that, at exhaustion, similar levels of deoxygenation were observed for the two intermittent exercises. Nevertheless, the decline in $HbO_2$ measured during IE-PR2 (28.6 ± 12.6%) was significantly lower than for IE-AR (37.9 ± 4.6%). The mean rate of decrease in $HbO_2$ calculated during IE-PR2 (5.3 ± 3.8%·s$^{-1}$) was significantly faster ($P < 0.01$) than during IE-PR (2.9 ± 2.4%·s$^{-1}$). This difference could come from deoxygenation kinetics according to exercise phases. Christmass et al. (14) showed that, for intermittent exercises made up 12 s of runs at 120% of VO$_{2\text{peak}}$ alternated with 18 s of rest, HbO$_2$ response was characterized by a fast decline from the start until 5 min of exercise and then by a steady state. According to these authors, the fast decline at the beginning of exercise would be caused by the exercise-induced hyperemia, whereas the steady state phase was characterized by oxygenation and deoxygenation according to exercise periods and recovery periods (14,15). As a consequence, the influence of the decline in $HbO_2$ during the initial phase would be smaller for an exercise with a longer duration, as for IE-PR, than for an exercise with a shorter duration, as for IE-PR2.

For the two intermittent exercises forms, only the recovery type was different, whereas TTE was significantly longer for IE-PR than for IE-AR. This result suggests that the passive recovery intervals allow subjects to recover faster than during active recovery intervals. During short intermittent exercise at high intensities, adenosine triphosphate (ATP) is resynthesized by anaerobic and aerobic pathways. During a single short-duration bout of high-intensity exercise, the greater part of the ATP needed to fuel contractile activity is supplied by anaerobic metabolism from phosphocreatine (PCr) and glycolysis (20), whereas the contribution of aerobic metabolism to ATP resynthesis increases when the type of exercise is repeated with short recovery intervals (3,20). Our results suggest that the deoxygenation was lower when exercises were alternated with passive recovery rather than with active recovery at 40% of VO$_{2\text{max}}$, the mean rate of decrease in $HbO_2$ being significantly lower for IE-PR than for IE-AR and the decline in $HbO_2$ measured during IE-PR2 being significantly lower than during IE-AR. Already, in 1960, Åstrand et al. (1) suggested that, for short intermittent exercise, oxygen stored on myoglobin has the time to be reloaded before the following work period begins. This implies that the myoglobin oxygen replenishments should be also higher during IE-PR than during IE-AR. As IE-PR is characterized by a lower decline in $HbO_2$ than during IE-AR, it should allow a higher reoxygenation of the myoglobin. Likewise, as PCr resynthesis depends on oxygen availability (23,24), a greater quantity of PCr should be resynthesized during IE-PR rather than during IE-AR. This suggestion is confirmed by Yoshida et al. (39), who found that the mean time constant during active recovery appeared slower (30.2 ± 3.0 s) than during passive recovery (25.3 ± 2.6 s).

The longer TTE obtained for IE-PR in comparison with IE-AR may also come from the lower energy requirement and by faster oxygen replenishment during passive recovery intervals than during active recovery intervals. Metabolic power was lower during IE-PR ($48.9 \pm 4.9$ mL·kg$^{-1}$·min$^{-1}$) than during IE-AR ($52.6 \pm 4.6$ mL·kg$^{-1}$·min$^{-1}$), which may partly explain why TTE was longer for IE-PR than for IE-AR. As
it has been found that HbO₂ decreases in proportion to the increase in the work rate during continuous exercise (6,16) and that a decreased oxygenation level is significantly lower for intermittent exercises performed at the highest workload (2), it may indicate a faster deoxygenation during IE-AR than during IE-PR.

In conclusion, the main findings of the present study were that TTE was significantly longer for IE-PR than for IE-AR and that the deoxygenation was slower during IE-PR than during IE-AR. The longer TTE for IE-PR could be explained by higher metabolic power for IE-AR than for IE-PR. Moreover, as IE-PR was characterized by a lower decline in HbO₂ than during IE-AR, it should allow a higher reoxygenation of myoglobin and a higher PCR resynthesis. This would explain why TTE were significantly correlated with the mean rate of decrease in HbO₂ during IE-PR and IE-AR. In perspective, it would be interesting to characterize the PCR kinetics for short intermittent exercises and to analyze the possible influence of other recovery intensities or recovery times on TTE.

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