Prolonged expiration down to residual volume leads to severe arterial hypoxemia in athletes during submaximal exercise

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Abstract

The goal of this study was to assess the effects of a prolonged expiration (PE) carried out down to the residual volume (RV) during a submaximal exercise and consider whether it would be worth including this respiratory technique in a training programme to evaluate its effects on performance. Ten male triathletes performed a 5-min exercise at 70% of maximal oxygen consumption in normal breathing (NB70) and in PE (PE70) down to RV. Cardiorespiratory parameters were measured continuously and an arterialized blood sampling at the earlobe was performed in the last 15 s of exercise. Oxygen consumption, cardiac frequency, end-tidal and arterial carbon dioxide pressure, alveolar–arterial difference for O₂ (P A O₂ − P a O₂) and P 50 were significantly higher, and arterial oxygen saturation (87.4 ± 3.4% versus 95.0 ± 0.9%, p < 0.001), alveolar (P A O₂) or arterial oxygen pressure, pH and ventilatory equivalent were significantly lower in PE70 than NB70. There was no difference in blood lactate between exercise modalities. These results demonstrate that during submaximal exercise, a prolonged expiration down to RV can lead to a severe hypoxemia caused by a P A O₂ decrement (r = 0.56; p < 0.05), a widened P A O₂ − P a O₂ (r = −0.85; p < 0.001) and a right shift of the oxygen dissociation curve (r = −0.73; p < 0.001).

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1. Introduction

These last few years some new ways of hypoxic training have appeared. This new approach, called intermittent hypoxic training (IHT), tries to reproduce some of the key features of altitude acclimatization in order to improve sea-level performance. Generally, IHT, based on a discontinuous use of normobaric or hypobaric hypoxia, can be divided into two different strategies: (1) providing hypoxia at rest and training in normoxic conditions. This is the concept “live high-train low” (Levine and Stray-Gundersen, 1997) which has been shown to improve sea-level performance (Levine and Stray-Gundersen, 1997; Stray-Gundersen et al., 2001; Brugniaux et al., 2006) and (2) providing hypoxia only during exercise in order to enhance the training stimulus [“live low-train high” (LLTH)].

This method, even though controversial, has also been reported to improve aerobic (Melissa et al., 1997; Meeuwsen et al., 2001; Dufour et al., 2006) and anaerobic performance (Meeuwsen et al., 2001). Unfortunately, for logistical and financial reasons, IHT is difficult to use by most athletes. The access to high altitude training facilities is not easy in all countries and the devices which create a hypoxic environment are quite expensive.

Alternative means of obtaining hypoxemia during exercise without being placed in a hypoxic environment can be imagined. Voluntary reduction of breathing frequency can produce hypoxemia. This idea has already been investigated in the eighties and the early nineties (Craig, 1980; Dicker et al., 1980; Holmer and Gullstrand, 1980; Yamamoto et al., 1987; Stager et al., 1989; Lee et al., 1990). Nevertheless, some studies assumed that the alveolar–arterial difference for O₂ (P A O₂ − P a O₂) was not wide enough when using this respiratory technique and, according to the estimated alveolar oxygen pressure (P A O₂), concluded that the reduction in arterial oxygen saturation (S a O₂) was not marked enough to induce physiological reactions to hypoxemia (Craig,
1980; Dicker et al., 1980; Holmer and Gullstrand, 1980). Basically, the reduced frequency breathing (RFB) technique would have to be carried out at mild altitude to create a real hypoxemia (Stager et al., 1989; Lee et al., 1990), which is still not very practical. Actually, several studies reported more hypercapnic than hypoxic effects (Craig, 1979; Dicker et al., 1980; Holmer and Gullstrand, 1980; Hsieh and Hermiston, 1983). Two reasons could explain a lack of hypoxemia \( [S_{aO_2} \text{ below } 95\% \text{ (Dempsey and Wagner, 1999)}] \) during RFB: (1) when it is carried out at a high pulmonary volume level, a breath holding at total lung capacity (TLC) could facilitate \( O_2 \) diffusion (Yamamoto et al., 1987), enhance alveolar \( O_2 \) stores, and finally avoid arterial desaturation (2) when it is performed in a lying position and therefore especially in swimmers, ventilation–perfusion ratio \( (V/Q) \) is probably more homogeneous which diminishes \( P_{AaO_2} - P_{aO_2} \) and consequently leads to a higher \( S_{aO_2} \).

Nevertheless, RFB could lead to a real hypoxemia if carried out at low pulmonary volume levels and in a standing position. Yamamoto et al. (1987) reported a drop of \( S_{aO_2} \) as low as 88% when this respiratory technique was performed at functional residual capacity (FRC) during an exercise on a cycle ergometer. In those conditions, \( P_{AaO_2} - P_{aO_2} \) was significantly wider than in normal breathing and led to a large arterial desaturation. Previously, it had already been reported that a breath holding at low pulmonary volume levels could lead to severe hypoxemia (Findley et al., 1983). Surprisingly, no study has ever investigated the effects of a RFB training on performance. Indeed, such a HFT technique may have some similar effects than the LLTH method.

The main objective of the present experiment was to assess the \( S_{aO_2} \) level and determine the factors leading to arterial desaturation when RFB is carried out at low pulmonary volume, thanks to a prolonged expiration down to the residual volume (RV). We hypothesized that in these conditions, one could show a greater hypoxemia than the one reported by Yamamoto et al. (1987), that is to say a \( S_{aO_2} \) below 88%. If so, it could therefore be interesting to include the RFB technique in a training programme and evaluate its effects on performance.

2. Methods

2.1. Subjects

Ten male triathletes, with a training volume of 10 h a week on average volunteered for this study. They presented no sign neither had a history of cardiovascular or respiratory disease. They were informed about the nature, the conditions and the risks of the experiment and gave their written informed consent. All the procedures were approved by the ethical committee of Necker Hospital, Paris, France. The characteristics of the subjects are presented in Table 1.

2.2. General experimental design

The subjects came twice to the laboratory. The two visits were separated by 7 days.

Table 1

<table>
<thead>
<tr>
<th>Subjects (( n = 10 ))</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>27.1</td>
<td>8.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.9</td>
<td>6.0</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.8</td>
<td>3.2</td>
</tr>
<tr>
<td>( V_{O_2 \text{ max}} ) (mL min(^{-1}) kg(^{-1}))</td>
<td>61.9</td>
<td>4.9</td>
</tr>
<tr>
<td>( S_{O_2} ) (%)</td>
<td>94.0</td>
<td>1.8</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>5.4</td>
<td>0.63</td>
</tr>
<tr>
<td>MVV (L min(^{-1}))</td>
<td>191.1</td>
<td>8.9</td>
</tr>
<tr>
<td>FEV(_1.0) (L)</td>
<td>4.6</td>
<td>0.56</td>
</tr>
<tr>
<td>FEV(_{1.0})/FVC (%)</td>
<td>88.9</td>
<td>0.58</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>6.8</td>
<td>0.68</td>
</tr>
</tbody>
</table>

Values are mean ± S.D. \( V_{O_2 \text{ max}} \), maximal oxygen consumption; \( S_{O_2} \), pulse oximetry \( O_2 \) saturation in \( V_{O_2 \text{ max}} \); FVC, forced vital capacity; MVV, maximal voluntary ventilation; FEV\(_{1.0}\), forced expiratory volume in 1 s; TLC, total lung capacity.

2.2.1. First visit

2.2.1.1. Rest examinations. Clinical interviews and examinations were conducted: height, weight, blood pressure, lung spirometry (Table 1) and ECG tracing were recorded.

2.2.1.2. Incremental exercise. The subjects then performed a maximal exercise test on an electrically braked cycle ergometer (Jaeger ER 900, Wuerzburg, Germany) to assess maximal oxygen consumption \( (V_{O_2 \text{ max}}) \), maximal power output \( (W_{\text{max}}) \), expired ventilation \( (V_E) \), cardiac frequency \((f_H)\), arterial oxygen saturation by pulse oximetry \( (S_{P_02}) \). After remaining at rest for 5 min, they warmed up for 3 min at an exercise intensity of 60 W. The workload was then started at 90 W and increased by 20 W every minute until exhaustion. The subjects had to maintain a pedaling frequency of 75 rpm and were verbally encouraged to continue the exercise as long as possible.

2.2.1.3. Prolonged expiration (PE). About 1 h after the incremental test, we asked the subjects to perform a low intensity exercise \( (50\% V_{O_2 \text{ max}}) \) in PE. This was done to familiarize all the subjects with the respiratory technique (described below) they had to apply at the second visit.

2.2.2. Second visit

2.2.2.1. Exercise protocol. The subjects were asked to perform three 5-min exercises on the same cycle ergometer they used at the first visit and at the same pedaling frequency (75 rpm):

1. 70% of the power output (PO) corresponding to \( V_{O_2 \text{ max}} \) in normal breathing (NB\(_{70}\));
2. 70% of PO corresponding to \( V_{O_2 \text{ max}} \) with prolonged expiration (PE\(_{70}\));
3. 65% of PO corresponding to \( V_{O_2 \text{ max}} \) with prolonged expiration (PE\(_{65}\)).

We chose 70 and 65% of \( V_{O_2 \text{ max}} \) because these intensities could be adequate to perform RFB training. Furthermore, during
submaximal exercise in RFB, as what happens in hypoxia (Peltonen et al., 1999; Woorons et al., 2006), a slight difference of exercise intensity may have a significant impact on $S_{\text{O}_2}$. The three exercises were randomized and separated by 45 min of rest. Before each exercise, the subjects remained seated at rest on the cycle ergometer for 5 min. They then warmed up for 3 min at an exercise intensity corresponding to 50% of $V_{\text{O}_2}\text{max}$. During the third minute of the warming up and only before an exercise in PE, the subjects began to reduce the frequency of breathing in order to prepare their organism and adjust their breathing to the pedaling frequency. After being warmed and without stopping pedaling, the subjects performed one of the three 5-min periods mentioned above. When the exercise was done in PE, the 5 min were divided into five periods of 1 min with 15 s in normal breathing followed by 45 s in PE (Fig. 1). We did not make the subjects continuously hypoventilate to avoid headaches due to hypercapnia.

2.2.2.2. PE characteristics. To perform PE, the subjects had to continuously, progressively and completely expire for 4 s down to RV and then briefly inspire for a few 1/10e s. To precisely control this time of inspiration/expiration, we used the pedaling frequency which was established at 75 rpm and permanently monitored. Thus, the expiration was made on 5 rev, and the inspiration on 1/2 rev. The subjects were coached during the entire test in order to carry out the exercise as adequately as possible.

2.2.2.3. Measurements.

2.2.2.3.1. Cardiorespiratory parameters. The ventilatory and gas exchanges variables were continuously measured using a breath-by-breath automated exercise metabolic system. The subjects breathed through a rigid mouthpiece connected to a “Y” system fixation with a double valve which ensures anti return (Jaeger, Germany). Expired gases were collected into a metabolicograph (Oxycon, Jeager, Wuerzburg, Germany) to measure: $V_{\text{E}}$, tidal volume ($V_t$), respiratory exchange ratio ($R$), $\text{O}_2$ consumption ($\dot{V}_{\text{O}_2}$), end-tidal $\text{O}_2$ pressure ($P_{\text{ETO}_2}$) and end-tidal carbon dioxide pressure ($P_{\text{ETCO}_2}$). The ventilatory equivalent ($V_{\text{E}}/\dot{V}_{\text{O}_2}$) was calculated. A 12-lead electrocardiogram as well as $f_h$ were recorded continuously.

$S_{\text{O}_2}$ was measured by an ear pulse oximeter (Ohmeda Biox 3740, Louisville, Colorado, USA) reported to be resistant to the effects of subjects motion (Barker, 2002) and whom the accuracy has been proved (Trivedi et al., 1997). Before each use of the oximeter and the attachment of the ear clip, the earlobe was massaged vigorously and prewarmed with a vasodilating capsaicin cream to increase perfusion (Woorons et al., 2005). The breath-by-breath measurements, $f_h$ and $S_{\text{O}_2}$ were averaged over 15-s intervals and data analysed at the end of each minute of exercise. Fig. 1 describes the exercise protocol and provides an example of $S_{\text{O}_2}$ and $P_{\text{ETCO}_2}$ variations during a 5-min period.

2.2.2.3.2. Arterialized blood gases. Two arterialized blood sampling were drawn from a prewarmed earlobe with a vasodilating capsaicin at rest and in the last 15 s of exercise. Blood gases were immediately analysed for arterial oxygen pressure ($P_{\text{aO}_2}$), arterial carbon dioxide pressure ($P_{\text{aCO}_2}$), $S_{\text{aO}_2}$, pH, $P_{\text{S}}$, lactate [La], bicarbonate (HCO$_3^-$) and haemoglobin (Hb) concentration (Radiometer ABL 700, Copenhagen). These measurements were made at an estimated temperature of 37°C at rest and 38°C at exercise. Some studies have demonstrated that $P_{\text{O}_2}$ values were slightly lower when measured by arterialized earlobe blood samples compared with radial artery ones (Sauty et al., 1996; Fajac et al., 1998). However, the accuracy of arterialized capillary blood sampling is also very much operator dependent. The technicians are especially trained in our laboratory to perform the arterialized measurements. These measurements have already been used under pathological conditions and were not different compared with arterial values (Lamberto et al., 2004). $P_{\text{A}O_2}$ and $P_{\text{A}O_2} - P_{\text{aO}_2}$ were calculated from the ideal alveolar gas equation (Cumming et al., 1969) assuming that $P_{\text{aCO}_2}$ was equal to mean alveolar carbon dioxide pressure ($P_{\text{ACO}_2}$)

$$P_{\text{A}O_2} = P_{\text{O}_2} - \left( \frac{P_{\text{ACO}_2}}{R} \right) [1 + F_{\text{O}_2}(1 - R)]$$
where $P_{O_2}$ is partial pressure of oxygen in inspired air and $F_{dO_2}$ is fractional concentration of oxygen in inspired air.

2.2.2.3.3. **Blood pressure and the rating of perceived exertion (RPE).** Blood pressure was measured at rest and during the last minute of exercise. Just after the end of exercise, we asked the subjects to assess the RPE using the Borg scale.

2.2.3. **Data analysis**

To compare NB$_{70}$ and PE$_{70}$ at the end of each minute of exercise, we performed a two-way analysis of variance (ANOVA). When a significant main effect was found, a Tukey post hoc test was carried out for contrasting both exercise modalities at each minute of exercise. We used a Student $t$-test to compare the blood gas values. Concerning PE$_{65}$, we only dealt with the data of $S_{aO_2}$ and $S_{pO_2}$ to assess the level of arterial desaturation. Pearson product moment correlations allowed us to determine the relationships between the data. All the data are expressed as the mean ± S.D. The level of significance was established at $p < 0.05$ for all statistics.

3. Results

3.1. **Cardiorespiratory parameters**

At the end of each minute of exercise, $S_{aO_2}$ fell lower in PE$_{70}$ than both in PE$_{65}$ and NB$_{70}$, and fell lower in PE$_{65}$ than NB$_{70}$ (Fig. 2).

$V_{O_2}, f_{st}$, $P_{ETCO_2}$ and $V_T$ were higher during PE$_{70}$ than NB$_{70}$ from min 1 to min 5. $V_{E}/V_{O_2}$, $P_{ETO_2}$ and $R$ were lower during PE$_{70}$ than NB$_{70}$ from min 1 to min 5. The results of ANOVA did not show any significant exercise modality effect for $V_E$ (Table 2).

**Table 2**

Cardiorespiratory variables at the end of each minute of exercise

<table>
<thead>
<tr>
<th></th>
<th>Min 1</th>
<th>Min 2</th>
<th>Min 3</th>
<th>Min 4</th>
<th>Min 5</th>
</tr>
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<tbody>
<tr>
<td>$V_{O_2}$ (L min$^{-1}$)</td>
<td>NB$_{70}$ 2.65 ± 0.24</td>
<td>PE$_{70}$ 2.90 ± 0.17*</td>
<td>NB$_{70}$ 2.90 ± 0.25</td>
<td>PE$_{70}$ 3.26 ± 0.26*</td>
<td>PE$_{70}$ 3.39 ± 0.23*</td>
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<tr>
<td></td>
<td>NB$_{70}$ 143 ± 15.8</td>
<td>PE$_{70}$ 148 ± 14.7*</td>
<td>NB$_{70}$ 148 ± 15.2</td>
<td>PE$_{70}$ 151 ± 15.3</td>
<td>PE$_{70}$ 158 ± 12.4*</td>
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<tr>
<td></td>
<td>NB$_{70}$ 63.0 ± 5.9</td>
<td>PE$_{70}$ 61.2 ± 8.5</td>
<td>NB$_{70}$ 70.8 ± 7.8</td>
<td>PE$_{70}$ 74.3 ± 7.8</td>
<td>PE$_{70}$ 75.3 ± 7.6</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>NB$_{70}$ 23.8 ± 1.8</td>
<td>PE$_{70}$ 21.2 ± 2.8*</td>
<td>NB$_{70}$ 24.4 ± 1.9</td>
<td>PE$_{70}$ 24.9 ± 1.6</td>
<td>PE$_{70}$ 24.7 ± 1.6</td>
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<tr>
<td></td>
<td>NB$_{70}$ 0.97 ± 0.07</td>
<td>PE$_{70}$ 0.88 ± 0.10*</td>
<td>NB$_{70}$ 1.03 ± 0.08</td>
<td>PE$_{70}$ 1.05 ± 0.07</td>
<td>PE$_{70}$ 1.02 ± 0.07*</td>
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</tr>
<tr>
<td></td>
<td>NB$_{70}$ 99.9 ± 2.2</td>
<td>PE$_{70}$ 84.0 ± 7.5*</td>
<td>NB$_{70}$ 101.0 ± 3.5</td>
<td>PE$_{70}$ 102.0 ± 3.4</td>
<td>PE$_{70}$ 102.6 ± 2.5</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>NB$_{70}$ 47.8 ± 3.2</td>
<td>PE$_{70}$ 55.3 ± 5.3*</td>
<td>NB$_{70}$ 48.8 ± 3.3</td>
<td>PE$_{70}$ 48.0 ± 3.4</td>
<td>PE$_{70}$ 46.9 ± 3.2</td>
</tr>
</tbody>
</table>

Values are mean ± S.D. NB$_{70}$, exercise in normal breathing at 70% of $V_{O_2}$ max; PE$_{70}$, exercise with prolonged expiration at 70% of $V_{O_2}$ max; $P_{ETCO_2}$, oxygen consumption; $f_{st}$, cardiac frequency; $V_{E}$, expired minute volume of gas at body temperature and pressure saturated; $V_{E}/V_{O_2}$, ventilatory equivalent; $V_T$, tidal volume; $R$, respiratory exchange ratio; $P_{ETO_2}$, end-tidal $O_2$ pressure; $P_{ETCO_2}$, end-tidal carbon dioxide pressure; * significant difference from NB$_{70}$ ($p < 0.05$).

3.2. **Arterialized blood gases**

The results are presented in Table 3 and Fig. 3. At rest, there was no significant difference in any parameter between exercise
modalities. At the end of exercise, $P_{A\text{O}_2}$, $P_{a\text{O}_2}$ and pH were lower during PE70 than NB70. There was no difference in [La] and Hb between exercise modalities. $P_{A\text{O}_2}$ - $P_{a\text{O}_2}$, $P_{\text{HCO}_3^-}$ and $P_{50}$ were higher during PE70 than NB70. $S_{a\text{O}_2}$ was lower in PE70 than both PE65 and NB70. Likewise, $S_{a\text{O}_2}$ was lower during PE65 than NB70.

3.3. Arterial pressure and RPE

There was no difference in systolic, diastolic and mean arterial pressure between NB70 and PE70 both at rest and at exercise (Table 4). The RPE was significantly higher during PE70 than NB70 (15.6 ± 0.7 versus 12.1 ± 0.9, $p < 0.05$).

3.4. Relationships between parameters

When we pooled the data of PE70 and PE65, $S_{a\text{O}_2}$ was correlated to $P_{A\text{O}_2}$ ($r = 0.56; p < 0.05$), $P_{A\text{O}_2}$ - $P_{a\text{O}_2}$ ($r = -0.85; p < 0.001$) and $P_{50}$ ($r = -0.73; p < 0.001$) (Fig. 4). During PE70, there was a correlation between $P_{\text{HCO}_3^-}$ and pH ($r = -0.67; p < 0.05$) and $P_{50}$ was correlated with both $P_{\text{HCO}_3^-}$ ($r = 0.91; p < 0.001$) and pH ($r = -0.64; p < 0.05$). On the other hand, we did not find any correlation between pH and [La] ($r = -0.58; p = 0.08$). The difference between PE70 and NB70 in pH (ΔpH) was correlated to Δ$P_{\text{HCO}_3^-}$ ($r = -0.63; p < 0.05$) but not to Δ[La] ($r = -0.31; p = 0.39$).

Table 4

<table>
<thead>
<tr>
<th>Systemic blood pressure</th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NB70</td>
<td>PE70</td>
</tr>
<tr>
<td>Systolic</td>
<td>123.9 ± 16.7</td>
<td>127.8 ± 18.0</td>
</tr>
<tr>
<td>Diastolic</td>
<td>79.4 ± 16.7</td>
<td>81.7 ± 15.4</td>
</tr>
<tr>
<td>Mean</td>
<td>94.1 ± 15.0</td>
<td>96.9 ± 15.6</td>
</tr>
</tbody>
</table>

Values are mean ± S.D. NB70, exercise in normal breathing at 70% of $V_{O_2\text{max}}$; PE70, exercise with prolonged expiration at 70% of $V_{O_2\text{max}}$. $S_{a\text{O}_2}$, arterial oxygen saturation; $P_{a\text{O}_2}$, arterial oxygen pressure; $P_{A\text{O}_2}$, arterial oxygen pressure; $S_{a\text{O}_2}$, alveolar–arterial difference for O2; $P_{\text{HCO}_3^-}$, arterial carbon dioxide pressure; [Hb], haemoglobin concentration; [La], blood lactate concentration; [HCO$_3^-$], blood bicarbonate concentration; $P_{50}$, $P_O$ for $S_{a\text{O}_2} = 50\%$. * significant difference from NB70 ($p < 0.05$).
4. Discussion

This study showed that during a submaximal exercise, a prolonged expiration down to RV could lead to a severe hypoxemia ($S_{O_2} <$ 88%). The subjects were asked to alternate some 4-s expirations with brief inspirations, inducing a drop of $S_{O_2}$ as low as 87%. To our knowledge, this is the lowest $S_{O_2}$ reported in similar exercise conditions, even though $P_{O_2}$ and $S_{O_2}$ values may be slightly lower in arterialized blood samples compared with arterial ones (Sautyt et al., 1996; Fajac et al., 1998). The majority of studies investigating the effects of RFB were carried out in the eighties and failed to find a significant hypoxemia (Craig, 1980; Dicker et al., 1980; Holmer and Gullstrand, 1980). Yamamoto et al. (1987) however reported a $S_{O_2}$ of about 88% when RFB was performed at FRC. In our study, while $S_{O_2}$ fell down to 87% during PE70, it was about 4% higher during PE65 with the same time of inspiration/expiration as PE70. This means that the intensity of exercise is an important factor to take into account for inducing a severe hypoxemia. Actually, the challenge is to adequately combine the exercise intensity with the duration of breath holding. Thus, during PE65, $S_{O_2}$ would probably have been lower with an expiration time beyond 4 s.

As expected, hypoxemia was also accompanied by a marked hypercapnia. This phenomenon is the consequence of hypoventilation and had already been reported by several studies investigating the effects of RFB (Craig, 1979; Dicker et al., 1980; Holmer and Gullstrand, 1980; Hsieh and Hermiston, 1983). Nevertheless, neither during nor after the test in PE, our subjects complained from headache or any other problem related to hypoxemia. On the other hand, hypercapnia was probably the main reason which made them feel less comfortable during PE, as attested by the RPE. The marked hypercapnic acidosis found during PE70 was probably responsible for a right shift of the oxygen dissociation curve (ODC), as shown by the dramatic increase in $P_{SO_2}$.

$V_{E}/V_{O_2}$ and $P_{ETO_2}$ were expectedly lower during PE70 than NB70. However, we did not find any difference in $V_{E}$ between exercise modalities. This could be explained by the higher $V_{O_2}$ during PE70, which consequently required a higher ventilation level. The higher $V_{O_2}$ at a given power output during PE70 may be due to a higher ventilatory cost. Indeed, the subjects had to force their expiration to reach RV and, regarding $V_{T}$ during exercise, they reached high pulmonary volume during a short inspiration with very high inspiratory flow. This consequently led to a greater demand of the respiratory muscles than with normal breathing. The unusual pattern of breathing in PE might have interfered with the measurement of pulmonary gas exchange and slightly overestimated $V_{O_2}$ values. However, this hypothesis is improbable since the linearity of the sensor was rigorously checked before each measurement for a large scale of flows (0–12 L s$^{-1}$). The increase in $f_{I}$ was certainly related to the higher $V_{O_2}$, as well as greater hypercapnia and acidosis, through a higher activation of the adrenergic system (Ehrsam et al., 1982).

The present study reported a lower pH during PE70 than NB70 but similar values of [La] in the two exercise modalities. Thus, one could argue that the lower pH during PE70 was probably more related to respiratory acidosis, resulting from hypercapnia, than to metabolic acidosis due to the release of lactate in the blood. Besides, the correlations we found between $P_{ETCO_2}$ and pH on one hand, and $\Delta P_{ETCO_2}$ and $\Delta pH$ on the other hand, argue in favour of the respiratory acidosis hypothesis. However, we cannot completely exclude that $PE_{70}$ led to a greater muscle lactate production than NB70. It is well known that blood lactate underestimates muscle lactate. Several studies have reported that hypercapnia could lead to a lower lactate release in blood (Graham et al., 1980; Ehrsam et al., 1982; Graham et al., 1986) and respiratory acidosis resulting from hypercapnia may shift the linear lactate to blood pH relationship during exercise below that in normocapnia (Kato et al., 2005). Yamamoto et al. (1988) reported a greater increase in blood lactate during recovery from exercise with RFB compared with exercise in normal breathing.

This delayed appearance of [La] is consistent with the inhibition of the movement of accumulating [La] out of the working muscles during exercise in PE. Therefore, we could hypothesize that a prolonged expiration down to RV may alter the glycolytic status and induce a higher lactate concentration in the working muscles without a concomitant appearance in the blood.

One of the most interesting points of this study was to determine the causes of the severe arterial desaturation during PE70. Physiologically, three factors could have been involved in this phenomenon: $P_{A0_2}$, $P_{A0_2} - P_{a0_2}$ and a right shift of ODC. Hypoventilation generally leads to a drop in $P_{A0_2}$. Consequently, this parameter ought to have been responsible for the hypoxemia for a great part. Nevertheless, according to our results, while $P_{A0_2}$ had a significant effect on $S_{O_2}$ during exercise, it did not play a more important role in the severe hypoxemia than both $P_{A0_2} - P_{a0_2}$ and the right shift of ODC, namely the Bohr effect. Actually, these last two factors contributed for a part at least as important as $P_{A0_2}$ in the large arterial desaturation induced by PE during exercise regarding the strong correlation we found between $P_{A0_2} - P_{a0_2}$ and $S_{O_2}$, and $P_{SO_2}$ and $S_{O_2}$. The wider $P_{A0_2} - P_{a0_2}$ we reported during PE was probably caused first by a greater inequality of $V/Q$, which is the consequence of the low pulmonary volumes (Morrison et al., 1982). A greater diffusion limitation could be another contributing factor. Wagner et al. (1986) reported that a $V_{E}$ above 2.7 L min$^{-1}$ could lead to a diffusion limitation and the $V_{O_2}$ reached at the end of exercise in the present study was about 3.5 L min$^{-1}$ during PE70 versus 3.12 L min$^{-1}$ for NB70.

The results of this study show some significant changes caused by a PE down to RV, which could make this respiratory technique interesting to use as a training method. A PE-induced severe hypoxemia, if undergone repeatedly, may lead to some adaptations especially at the cellular level since the time spent in hypoxia seems too short to provoke cardiovascular or haematological adaptations (Eckardt et al., 1989; Engfred et al., 1994; Vallier et al., 1996; Emonson et al., 1997). This way of training could thus be quite similar to the LLTH method which was reported to induce modifications at the cellular level and enhance aerobic (Melissa et al., 1997; Meeuwsen et al., 2001; Dufour et al., 2006) and anaerobic performance (Meeuwsen et al., 2001). A greater citrate synthase activity and myoglobin content could for instance be expected (Terrados et al., 1990) with a subsequent increased force production at a given oxygen consumption.
improvement in aerobic capacity. However, unlike LLTH, PE also generates hypercapnia. Regularly repeating exercises with hypercapnic acidosis may be more serious than performing a single 5-min exercise. On the other hand, in addition to the hypoxic effect, repetitive exposure to hypercapnic acidosis may induce further adaptations at the cellular or vascular level, limiting for instance the blood acidosis during exercise. Thus, it would be worth investigating the effects of this training method on performance.

While this study investigated the effect of PE during a 5-min exercise, the training duration should obviously be longer to get the specific adaptations mentioned above. However, both hypoventilation-induced hypercapnia and exercise hard- ness should make us cautious concerning the time of training. Thus, we suggest that integrating this respiratory technique in three sessions a week, with a hypoventilation time representing about 20–30 min per session could be sensible and would be a good compromise. Furthermore, to avoid the harmful effects of hypercapnia, the work should be organised in series separated by periods of normal breathing. For instance, after a warm-up of about 15 min, the athletes could perform four to five series of 5-min exercise (15 “NB-45” PE) each separated by 1 or 2 min of normal breathing. Further research would be useful to define the optimal duration and repetition of this kind of training.

In summary, we demonstrated that repeated prolonged expira- tions carried out down to residual volume during a submaximal exercise led to a drop of $S_{O_2}$ down to 87% and was also accom- panyed by a marked hypercapnia. The severe arterial desaturation was caused by a $P_{A_{O_2}}$ decrement, a widened $P_{A_{O_2}} - P_{A_{CO_2}}$ and a right shift of ODC. We also reported a greater $\dot{V}_{O_2}$ and $f_{i}$ during PE than NB70 maybe caused by the greater activity of the respir- atory muscles and the adrenergic system. Finally, the prolonged expiration led to a greater blood acidosis, mainly hypercapnic and possibly also linked to a greater muscle acidosis.

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