Swimmers can train in hypoxia at sea level through voluntary hypoventilation

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\textbf{A B S T R A C T}

This study used an innovative technique of pulse oximetry to investigate whether swimmers can train under hypoxic conditions through voluntary hypoventilation (VH). Ten trained subjects performed a front crawl swimming series with normal breathing (NB), VH at high (VH\textsubscript{high}) and low pulmonary volume (VH\textsubscript{low}). Arterial oxygen saturation was continuously measured via pulse oximetry (SpO\textsubscript{2}) with a waterproofed forehead sensor. Gas exchanges were recorded continuously and lactate concentration ([La]) was assessed at the end of each test. In VH\textsubscript{low}, SpO\textsubscript{2} fell down to 87\% at the end of the series whereas it remained above 94\% in VH\textsubscript{high} during most part of the series. Ventilation, oxygen uptake and end-tidal O\textsubscript{2} pressure were lower in both VH\textsubscript{high} and VH\textsubscript{low} than in NB. Compared to NB, [La] significantly increased in VH\textsubscript{low} and decreased in VH\textsubscript{high}. This study demonstrated that swimmers can train under hypoxic conditions at sea level and can accentuate the glycolytic stimulus of their training if they perform VH at low but not high pulmonary volume.

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1. Introduction

In the early seventies, a new training method appeared in competitive swimming. This method, still widely used, consists in reducing breathing frequency with fewer breaths relative to arm strokes (i.e. inhale every 5, 7, or 9 strokes instead of 2–3 single arm strokes). At the beginning, it was thought that holding one’s breath during exercise would decrease the oxygen (O\textsubscript{2}) availability to the muscles and could therefore simulate the effects of altitude training (Cousiliman, 1975). For this reason, this method was called “hypoxic training”. It was also expected that, because of the lower O\textsubscript{2} supply, this kind of training would improve the anaerobic metabolism (Cousiliman, 1975; Bonen, 1979). However, in the following years, some studies showed that a decrease in breathing frequency during exercise provoked only a hypercapnic effect (Dicker et al., 1980; Holmer and Gullstrand, 1980). Furthermore, other studies failed to find any increase in anaerobic metabolism since blood lactate concentration ([La]) was not different (Hsieh and Hermiston, 1983; Yamamoto et al., 1987; Town and Vanness, 1990), or even lower (Holmer and Gullstrand, 1980) when compared with normal breathing. Despite these findings, breath holding has become a classical training method in swimming which continues to be called erroneously “hypoxic training” by many coaches or swimmers.

In the mid 2000s, a new approach of exercising with voluntary hypoventilation (VH) was proposed by our laboratory. We postulated that when VH is performed in the classical way, which is at high pulmonary volume like in swimming, the alveolar O\textsubscript{2} stores are enhanced and gas exchanges are facilitated which therefore prevents obtaining a hypoxemic effect (Woorons et al., 2007). On the contrary, we hypothesized that VH at low pulmonary volume, that is at or below functional residual capacity (FRC), should lower the alveolar O\textsubscript{2} partial pressure (P\textsubscript{A\textsubscript{O}2}) and induce a greater heterogeneity of the ventilation to perfusion ratio (V/Q), thus increasing the alveolar to arterial difference for O\textsubscript{2} (D(A–a)O\textsubscript{2}). This hypothesis was verified in cycling and running where the arterial O\textsubscript{2} saturation (SaO\textsubscript{2}) fell down to 87\% on average (Woorons et al., 2007, 2008, 2010, 2011). The severe hypoxemia induced by VH at FRC also led to a greater muscle deoxygenation and therefore likely to tissue hypoxia (Woorons et al., 2010). This phenomenon was probably responsible for the higher [La] we reported for the first time in the said study and which reflects a greater solicitation of anaerobic glycolysis. In all the above mentioned studies, the changes were systematically accompanied by respiratory acidosis.
under the effect of the elevated carbon dioxide (CO₂) pressures. Thus it appears that in terrestrial sports, the main feature of exercise with VH at low lung volume is to provoke a combined lactic and respiratory acidosis as the result of both hypoxic and hypercapnic effect.

So far, it is not known whether a swimming exercise performed with VH at low pulmonary volume could induce a hypoxic effect. First, in front crawl swimming in particular, gas exchange may be better than in cycling or running because V/Q matching is improved in prone position (Mure and Lindahl, 2001). Therefore, both arterial O₂ pressure and SaO₂ might not decrease as much as in terrestrial sports. Second, the measure of SaO₂ used to be technically problematic in an aquatic environment. However for the first time, thanks to an innovative equipment based on a waterproofed forehead sensor, it becomes possible to continuously measure arterial O₂ saturation by pulse oximetry (SpO₂) in swimmers and then precisely assess the effects of VH.

Training under hypoxic conditions thanks to VH and without leaving sea level could be interesting for swimmers. It has been shown that VH training reduces blood and probably muscle acidosis (Woorons et al., 2008) which could delay the onset of fatigue. Therefore, the goal of the present study was to determine and compare the effects of VH at low and high pulmonary volume on SpO₂ in swimmers through the validation of a novel technique of measurement using a waterproofed forehead sensor. We expected a significant drop in SpO₂ only with VH at low lung volume but hypothesized that the degree of hypoxemia would not be severe (i.e. SpO₂ < 88% [Dempsey and Wagner, 1999]).

2. Methods

2.1. Subjects

Ten subjects including six competitive swimmers (1 woman, 5 men) and 4 male triathletes were recruited to participate in this study. Two of the swimmers were involved in national competitions and trained 6–10 times a week. The four remaining swimmers had a regional level and trained 3–5 times a week. The triathletes carried out 2–4 swimming sessions a week and had also a regional level in this discipline. The characteristics (mean ± SD) of the subjects were age 29.2 ± 8.4 years, height 180.6 ± 5.0 cm and weight 75.1 ± 8.7 kg. All the subjects 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 Ile de France II, Paris, France.

2.2. Protocol

The whole experiment was conducted in the city of La Madeleine (59110) in the northern France (altitude = 29 m). The tests took place in a 25-m swimming pool with a water temperature of 27 °C.

**Fig. 1.** Description of the voluntary hypoventilation technique at high (A) and low (B) pulmonary volume.

**Pre-experiment session:** Before beginning the experiment, the subjects came once or twice to the pool to familiarize with the equipment as well as VH technique. For some of them, it was necessary to train breathing through a snorkel so that they could feel more relaxed afterwards. Furthermore, since a standard flip turn could not be carried out, the subjects had to learn making an open turn with the equipment to keep it safe and in place. All the subjects had already performed VH at high pulmonary volume (VH_high) during their training since it is the classical method used for decades in swimming. This breathing technique consists, just after inhaling, to hold one’s breath for a few seconds and then to rapidly exhale the air before the next inhalation (Fig. 1a). On the other hand, VH at low pulmonary volume (VH_low) requires to hold one’s breath after exhaling down to about FRC and then exhaling the remaining air just before the inhalation (Fig. 1b). Prior to the experiment, the subjects had never carried out VH_low during their training so it was necessary to familiarize them to what could be called the “exhale-hold” technique. We especially aimed to determine the number of arm strokes over which the subjects had to exhale before the breath holding. Even though the pulmonary volumes were not controlled, it was possible, thanks to several learning exercises, to get close to FRC given the fact that at this pulmonary volume it remains about 1 l to reach the residual volume. Depending on the subjects, the exhalation was made over 2 or 3 arm strokes.

When the subjects were accustomed to both the equipment and the VH technique, they first carried out a 400-m front crawl swimming at maximal speed. Then, in three other sessions separated by 48–72 h, they completed one series of ten 50-m front crawl swimming at 95% of their 400 m speed under the following randomized conditions: normal breathing (NB), VH_high and VH_low. During each 50 m, the speed was verified with an auditory signal at 12.5 m intervals. A visual mark was placed at the bottom and in the middle of the pool so that the swimmers could adjust their velocity when hearing the auditory signal. Between each 50 m, they rested motionlessly for 12 s along the wall, breathing normally. The characteristics of the series were determined in collaboration with a confirmed swimming coach. For VH_high and VH_low, we asked the swimmers to hold their breath as long as possible but to take care not to go to asphyxia in order to successfully complete the whole series. Subjects were instructed to refrain from strenuous exercise 24 h prior each testing session.

2.3. Measurements

2.3.1. SpO₂ and heart rate

SpO₂ and heart rate (HR) were measured via the pulse oximeter Nellcor N-595 (Pleasanton, CA, USA) with the adhesive forehead sensor Max-Fast (Nellcor, Pleasanton, CA, USA). To enable the utilization of the equipment in an aquatic environment, we waterproofed the sensor by wrapping it in an adhesive plastic film. The sensor was connected to the oximeter that was kept out of the
water. Before the beginning of each test, the sensor site was cleaned with an alcohol wipe to remove skin oils and was prewarmed with a vasodilating cream (Disalgyl®, Monin, France) to increase perfusion. The sensor was then placed above the supraorbital ridge of the subjects and secured with the personal latex swimming cap of the subjects (Fig. 2). It was important that the cap fitted the swimmer because too much or not enough pressure on the sensor can produce inaccurate SpO₂ measurements (Shelley et al., 2005). For each swimmer, we reused the same Max-Fast sensor over all sessions and placed it on the same site, which was identified thanks to pen marks made over the skin. Both SpO₂ and HR data were analyzed in the last 15-s of each 50-m. To insure that the waterproofing of the sensor and the conditions of measurements of the present study provided accurate and reliable estimations of arterial oxygen saturation measured by direct blood sampling (i.e. SaO₂), we validated this new technique of pulse oximetry in our laboratory (see supplementary data).

2.3.2. Gas exchange

During all the tests, gas exchange was recorded through a K4b² remote breath-by-breath portable system (Cosmed, Rome, Italy) reported as reliable for expired gas measurements (McLaughlin et al., 2001). The device was connected to the snorkel system (Aquatrainer, Cosmed, Rome, Italy) developed by Keskinen et al. (2003). With this Aquatrainer module, gas exchange is measured using inspiratory and expiratory flows. The connection of the inlet and outlet tubes to the K4b² turbine (50 ml) through a connecting unit (140 ml) allows inspiratory and expiratory gases to mix to a small extent at the beginning of both the expiration and inhalation. The distance between the snorkel mouthpiece and the K4b² turbine unit was 128 cm and the volumes of both the outlet and inlet tubes were 825 ml. This system has been found as a valid tool for collecting expired gas for breath-by-breath analysis, comparable to the standard facemask (Keskinen et al., 2003; Rodríguez et al., 2008). Before each test, we performed the standardized calibration procedures as recommended by the manufacturer (Cosmed, K4b²). These included air calibration, turbine calibration with a standard 3000-ml syringe, gas calibration with a certified commercial gas preparation (O₂: 16%, CO₂: 5%) and delay calibration to ensure accurate readings during the testing and to check the alignment between the gas flow and gas concentrations. The breath-by-breath measurements were performed for: tidal volume (Vt), respiratory frequency (f), expired ventilation (Ve), oxygen consumption (VO₂), carbon dioxide production (VCO₂), end-tidal O₂ pressure (PETO₂) and end-tidal carbon dioxide pressure (PETCO₂). The ventilatory equivalent (Ve/VO₂) was calculated. Data were analyzed in the last 15-s of each 50-m.

2.3.3. Rating of perceived exertion (RPE) and [La] 

Just after the end of each test, RPE was obtained using the Borg scale. Then, two minutes later, a blood sample (5 μl) was taken from the earlobe of the subjects to obtain [La]. The sample was collected with a portable blood lactate analyzer (Lactate Pro). This lactate analyzer has been shown to display a good reliability and accuracy when compared to a laboratory-based analyzer (Tanner et al., 2010).

2.4. Statistics

All the results are expressed as mean±SD. To determine whether there was a difference in oximetry variables and gas exchange between the three breathing modalities at the end of each 50 m, we performed a two-way analysis of variance (ANOVA) with repeated measures. When a significant main effect was found, the Bonferroni post hoc test was carried out. We also performed a one-way ANOVA followed by a Bonferroni post hoc test to compare [La] and RPE in the three series. Student’s t-tests were used to determine whether there was a difference in the changes (Δ) in variables (relative to NB exercise) between V̇H high and V̇H low at the end of the series. The following relationships were tested by performing Pearson linear regression tests: SpO₂/EETO₂, SpO₂/PETO₂, ΔSpO₂/Δ[La], ΔVO₂/Δ[La] and ΔV̇E/ΔV̇O₂. The level of significance was set at p < 0.05.

3. Results

3.1. 400-m front crawl swimming

The main results (mean±SD) at the end or over the last 15-s of all the tests were: performance time 341±37 s, V̇O₂ 3.62±0.58 L min⁻¹, V̇E 122±25 L min⁻¹, HR 179±9 bpm, SpO₂ 95±1.9% and [La] 8.2±2.1 mmol L⁻¹.

3.2. 50-m series

3.2.1. Oximetry measures

The mean SpO₂ of the whole series (Fig. 3a) as well as SpO₂ in each 50-m (Fig. 3b) were lower in V̇H low than in both V̇H high and NB, and lower in V̇H high than in NB. Cardiac frequency was lower in V̇H high, than in NB from the 3rd 50-m and lower in V̇H high than in V̇H low from the 6th 50-m. There was no difference in HR between V̇H low and NB during the whole series. At the end of the test, HR was 147±19 bpm in NB, 141±20 bpm in V̇H high and 145±21 bpm in V̇H low. ΔSpO₂ was greater and ΔHR lower in V̇H low than in V̇H high (Table 1).

3.2.2. Gas exchange

The results are presented in Fig. 4 and Table 1. Respiratory frequency, V̇E, V̇O₂, V̇E/V̇O₂, V̇CO₂ and PETO₂ were lower and V̇T and PETCO₂ were higher in both V̇H high and V̇H low than in NB from the beginning to the end of the series. PETO₂ was higher and PETCO₂ was lower in V̇H low than in V̇H high from the 6th 50-m. V̇E, V̇O₂, V̇CO₂ and V̇E/V̇O₂ were lower in V̇H high than in V̇H low during most part of
Fig. 3. Mean arterial O₂ saturation (SpO₂) (A) and SpO₂ in each 50-m (B) during the series performed with normal breathing (NB, □) and voluntary hypoventilation at high (VHₜₜₚ, ■) and low (VHₕₕₜ, ○) pulmonary volumes. Significantly different from *NB, †VHₜₜₚ. Values are mean ± SD; p < 0.05.

Fig. 4. Gas exchange recorded in the last 15 s of each 50-m during the series performed with normal breathing (□) and voluntary hypoventilation at high (■) and low (○) pulmonary volumes. fₑ, respiratory frequency; Vₜ, tidal volume; Vₑ, expired minute ventilation; Vₑ/Vₒ₂, ventilatory equivalent for O₂; Vₒ₂, oxygen uptake; Vₐₜ, carbon dioxide production; PₑO₂, end-tidal O₂ pressure; PₑCₐₐₜ, end-tidal carbon dioxide pressure. Significantly different from *NB, †VHₜₜₚ. Values are mean ± SD; p < 0.05.
the series. Finally, $f_R$ was lower in $VH_{high}$ than in $VH_{low}$ during the whole series whereas there was no difference in $V_t$ between both breathing techniques. Relative to NB exercise, the change in all gas exchange variables in the last 15 s of the series were significantly different between $VH_{low}$ and $VH_{high}$ (Table 1). In both VH techniques, we found a significant correlation between $PET_{CO_2}$ and $SPO_2$ ($VH_{low}$: $r = -0.76$, $p < 0.05$; $VH_{high}$: $r = -0.71$, $p < 0.05$; $n = 10$) and between $PET_{CO_2}$ and $SPO_2$ ($VH_{low}$: $r = 0.70$, $p < 0.05$; $VH_{high}$: $r = 0.68$, $p < 0.05$; $n = 10$). $\Delta V_t$ was also significantly correlated to $\Delta V_e$ at the end of exercise in both $VH_{low}$ ($r = 0.81$, $p < 0.05$) and $VH_{high}$ ($r = 0.73$, $p < 0.05$).

3.2.3. $[La]$ and RPE

$[La]$ was higher in $VH_{low}$ than in both $VH_{high}$ and NB and lower in $VH_{high}$ than in NB (Fig. 5). The changes in $[La]$ are reported in Table 1. There was a significant negative correlation between $\Delta [La]$ and $\Delta SPO_2$ when we pooled the data of $VH_{low}$ and $VH_{high}$ ($r = -0.61$, $p < 0.05$; $n = 20$). On the other hand, we did not find any correlation between $\Delta [La]$ and $\Delta V_t$ in both or pooled VH technique. RPE was greater in $VH_{low}$ than in both $VH_{high}$ and NB, and higher in $VH_{high}$ than in NB (Fig. 5).

4. Discussion

For the first time, this study demonstrated that $SPO_2$ can drop dramatically when swimmers exercise at sea level with VH at low pulmonary volume, which can be described as the exhal-e-hold technique. $SPO_2$ was measured using an oximetry technique based on a waterproofed forehead sensor that we validated for both the accuracy and the reliability. On the other hand, when swimmers performed the classical technique of hyperventilation used since the beginning of the seventies (i.e. “inhale-hold”), $SPO_2$, despite a slight decrease, could not lead to hypoxemia.

In $VH_{low}$, $SPO_2$ was 89% on average during the whole series while it dropped down to 87% at the end of exercise. This latter result runs counter to the hypothesis we made since $SPO_2$ below 88% is considered as severe hypoxemia (Dempsey and Wagner, 1999). A similar level of arterial desaturation, which corresponds to what could be obtained at altitudes above 2000 m during submaximal exercise (ANCHISI et al., 2001; WOORONS et al., 2006), had already been found in cycling or running with VH at FRC (WOORONS et al., 2008, 2010, 2011). On the other hand, no study had ever reported hypoxemia in swimmers performing VH. Since the continuous measurement of $SPO_2$ was not possible in swimming, the former studies estimated $SPO_2$ from respiratory variables (DICKER et al., 1980; HOLMER and GULLSTRAND, 1980). They concluded that exercise with VH did not lead to a significant arterial desaturation. Nonetheless, in these studies, VH was always carried out at high pulmonary volume. Even though $SPO_2$ could have been measured in swimmers, it is unlikely that a great arterial desaturation would have been obtained with this breathing technique. In the present study, we have shown that there was only a slight decrease in $SPO_2$ when using the classical VH method at high pulmonary volume. Indeed, in $VH_{high}$, $SPO_2$ was 93% at the end of exercise and remained above 94% during most part of the series. Therefore, it can be concluded that the method used for decades by swimmers does not lead to hypoxemia since for being considered as so, $SPO_2$ should have been equal to or below 92% during exercise (POWERS et al., 1989).

It has been shown that when VH down to residual volume is carried out during a cycling exercise, the resulting severe hypoxemia is caused by three factors: a decrease in $P_{O_2}$, a right shift of the oxygen dissociation curve (ODC) and a greater $D(A-a)O_2$ (WOORONS et al., 2007). In the present study, the lower $P_{ETCO_2}$ in $VH_{low}$ than in NB from the beginning to the end of exercise and the significant relationship between $PETCO_2$ and $SPO_2$ in $VH_{low}$ confirm the role of $P_{A CO_2}$. Furthermore, the higher $PETCO_2$ throughout exercise and the relationship between $PETCO_2$ and $SPO_2$ suggest that a right shift of ODC could have also played a role in the fall of $SPO_2$ in $VH_{low}$. Although pH was not measured in the present study, it is well known that elevated $PETCO_2$ leads to respiratory acidosis and therefore accentuates arterial desaturation as we previously showed during exercise with VH (WOORONS et al., 2007). It is however surprising that in $VH_{high}$, despite a lower $P_{ETO_2}$ and a greater level of hypercapnia than in $VH_{low}$, $SPO_2$ remained much higher. Thus, during VH exercise, the quality of the pulmonary gas exchange, depending on the lung volume at which breath holding occurs, seems to be the determinant factor for inducing a great arterial desaturation. A study has shown that when apnea is performed at rest, the lower the initial lung volume the greater the degree of hypoxemia, probably because of an increased heterogeneity of $V/Q$ (FINDLEY et al., 1983). The same hypothesis has been sustained.

Table 1

<table>
<thead>
<tr>
<th>Change (Δ) in variables (%) relative to normal breathing at the end of exercise.</th>
<th>$VH_{high}$</th>
<th>$VH_{low}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$ΔSPO_2$ (%)</td>
<td>$-4.7 \pm 2.7$</td>
<td>$-10.1 \pm 5.1$</td>
</tr>
<tr>
<td>$ΔHR$ (%)</td>
<td>$-4.0 \pm 8.1$</td>
<td>$-1.3 \pm 7.1$</td>
</tr>
<tr>
<td>$Δ[La]$ (%)</td>
<td>$-15.8 \pm 13.5$</td>
<td>$+3.1 \pm 35.1$</td>
</tr>
<tr>
<td>$ΔV_t$ (%)</td>
<td>$-67.0 \pm 9.6$</td>
<td>$-50.2 \pm 19.9$</td>
</tr>
<tr>
<td>$ΔV_e$ (%)</td>
<td>$+41.0 \pm 26.4$</td>
<td>$+26.6 \pm 33.1$</td>
</tr>
<tr>
<td>$ΔV_{CO_2}$ (%)</td>
<td>$-55.0 \pm 5.8$</td>
<td>$-41.1 \pm 10.4$</td>
</tr>
<tr>
<td>$ΔPETCO_2$ (%)</td>
<td>$-26.5 \pm 11.4$</td>
<td>$-14.8 \pm 10.8$</td>
</tr>
<tr>
<td>$ΔPETO_2$ (%)</td>
<td>$-28.6 \pm 7.9$</td>
<td>$-21.2 \pm 7.3$</td>
</tr>
<tr>
<td>$ΔPETO_2$ (%)</td>
<td>$-30.4 \pm 10.5$</td>
<td>$-22.3 \pm 14.9$</td>
</tr>
<tr>
<td>$ΔPETCO_2$ (%)</td>
<td>$+55.3 \pm 18.1$</td>
<td>$+41.3 \pm 24.8$</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

$VH_{high}$ voluntary hyperventilation at high pulmonary volume; $VH_{low}$ voluntary hyperventilation at low pulmonary volume; $P_{ETO_2}$, arterial oxygen saturation; HR, heart rate; [La], blood lactate concentration; $f_R$, respiratory frequency; $V_t$, tidal volume; $V_t$, expired ventilation; $V_{CO_2}$, oxygen consumption; $V_{CO_2}$, carbon dioxide production; $PETCO_2$, end-tidal CO$_2$ pressure; $PETO_2$, end-tidal O$_2$ pressure.

* Significantly different from $VH_{high}$. Values are mean ± SD; $p < 0.05$.

<Figure 5: Blood lactate concentration and rate of perceived exertion (RPE) at the end of the series performed with normal breathing (NB) and voluntary hyperventilation at high ($VH_{high}$) and low ($VH_{low}$) pulmonary volumes. Significantly different from *NB, $VH_{high}$. Values are mean ± SD; $p < 0.05$.>
to explain the large increase in D(A-a)O2 during VH exercise at low pulmonary volume (Yamamoto et al., 1987; Woorons et al., 2007). On the other hand, it is likely that at high pulmonary volume, close to total lung capacity, the homogeneity of V/Q is improved. This could explain why the fall of SpO2 was attenuated during VHhigh, despite a low PdO2 and high CO2 pressures.

Either with the exhale-hold or the inhale-hold technique, the large drop of Fk led to a decrease in itsp despite the compensating increase in itsq. It is noticeable that Fk and itsp were even lower in VHhigh than in VHlow. Thus, the higher the pulmonary volumes at the point of breath holding, the greater the fall in Fk and consequently itsp. During exercise with VHhigh, swimmers took a breath every 8 arm strokes on average. Such a low breathing frequency was also sustained previously by swimmers using the classical VH technique (Holmer and Gullstrand, 1980; Town and Vanness, 1990; West et al., 2005). On the other hand, during VHlow, swimmers inhaled every 4 or so strokes which represented 5 and 6 arm strokes. This fall in itsp might be the main reason for the decrease in VO2, that occurred in both VH techniques and that has already been reported in swimmers performing VH (Holmer and Gullstrand, 1980; Town and Vanness, 1990; West et al., 2005). During aquatic exercise, the respiratory system is challenged due to increased hydrostatic pressure which likely causes a greater work of breathing (Ray et al., 2008). Therefore, the energy cost of the respiratory muscles is probably higher than during terrestrial exercise for a given Vt. The large drop of itsp during VHlow, and even more during VHhigh, may have reduced the energy cost of the respiratory muscles and consequently should have strongly impacted on the whole body VO2. This hypothesis is reinforced by the significant correlation found between Δitsp and ΔVO2 in both VHlow and VHhigh. The decrease in VO2 during VH exercise might also be partly due to a reduced drag and/or a better stroke mechanics through an improvement in body position as suggested before (Holmer and Gullstrand, 1980). However, if it were so, this would occur only when VH is performed at high pulmonary volume since, according to Archimedes’ principle, a reduced amount of air in the lungs decreases swimmer’s buoyancy.

The increase in [La] during VHlow constitutes another interesting and original finding of this experiment. So far, all the studies that investigated the glycolytic effects of VH exercise in swimming failed to find any higher [La] than exercise with NB (Holmer and Gullstrand, 1980; Hsieh and Hermiston, 1983; Town and Vanness, 1990; West et al., 2005). This is probably because they all used the inhale-hold technique that does not elicitor arterial hyperoxemia. Indeed, it seems that the determinant factor for elevating [La] during VH exercise is the hypoxic effect. In a recent study, we demonstrated that the greater increase in [La] that was for the first time reported during a prolonged cycling exercise with VH at FRC was caused by the resulting severe arterial desaturation (Woorons et al., 2010). The decrease in oxygen delivery induced a greater muscle deoxygenation and probably tissue hypoxia, which could have led to a greater anaerobic response to exercise. In the present study, since the drop of SpO2 in VHlow was quite the same as in the former study, the same phenomenon probably occurred. This hypothesis is supported by the significant relationship between Δ[La] and ΔSpO2 in pooled VHlow and VHhigh. It is even likely that the difference in lactate concentration was greater within muscles since lactate release to the blood may be reduced in the presence of hypercapnia (Ehrsam et al., 1982; Graham et al., 1986). On the other hand, it is remarkable that [La] was lower in VHhigh than in NB. This decrease in [La] may be due to the large fall of VO2, and therefore to a lower relative exercise intensity, although we did not find any relationship between Δ[La] and ΔVO2. Previously, a lower [La] associated with a decreased VO2 had also been reported in swimmers performing VH at high pulmonary volume (Holmer and Gullstrand, 1980). Thus, the present results clearly show that swimmers can accentuate the metabolic acidosis and can increase the solicitation of anaerobic glycolysis if they perform the exhale-hold instead of the inhale-hold technique.

In summary, this study demonstrated that swimmers can actually train under hypoxic conditions at sea level or without simulating altitude if they perform VH at low pulmonary volume, namely the exhale-hold technique. SpO2 was estimated thanks to a pulse oximetry technique based on a waterproof forehead sensor that displayed both a good accuracy and reliability. We also demonstrated that using the exhale-hold technique, swimmers can elevate [La] and can therefore accentuate the glycolytic stimulus of their training. On the contrary, it was clearly shown that the classical method used for about forty years in swimming and which consists in holding one’s breath at high pulmonary volume does not lead to hypoxemia and reduces the solicitation of anaerobic glycolysis. Since interesting physiological adaptations have been found after training with VH at FRC in runners (Woorons et al., 2008), it would be worth investigating whether swimmers can improve their performance if they switch their breathing pattern from the inhale-hold to the exhale-hold technique during VH training.

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The authors declare that they have no conflict of interest.

The authors declare that this research has been carried out in accordance with “The Code of Ethics of the World Medical Association” for experimtents involving humans.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.resp.2013.08.022.

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