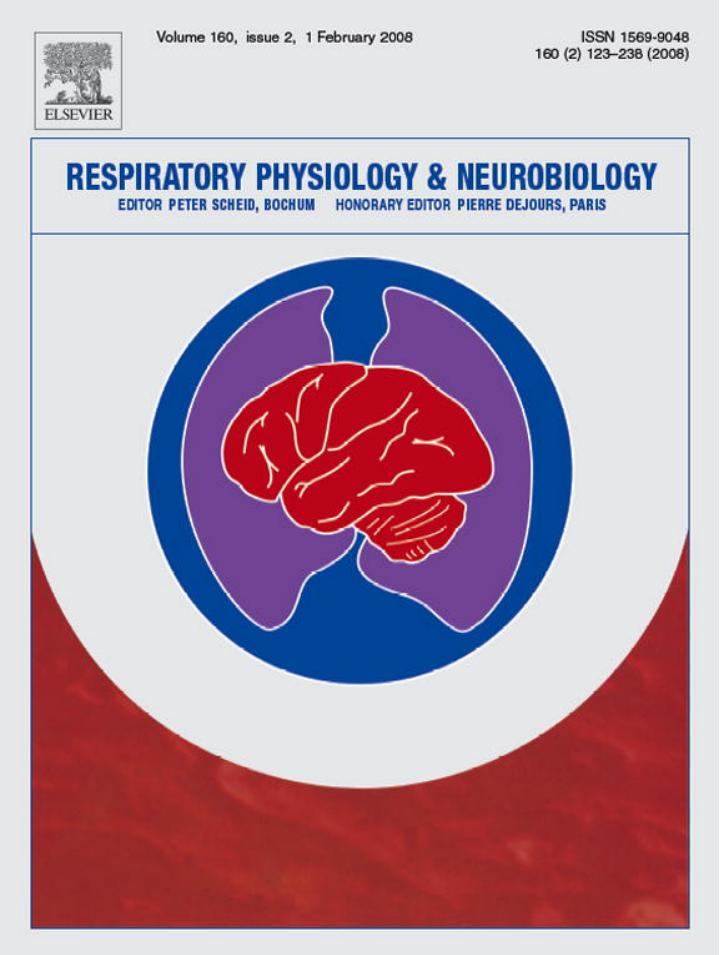

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Effects of a 4-week training with voluntary hypoventilation carried out at low pulmonary volumes

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Abstract

This study investigated the effects of training with voluntary hypoventilation (VH) at low pulmonary volumes. Two groups of moderately trained runners, one using hypoventilation (HYPO, $n=7$) and one control group (CONT, $n=8$), were constituted. The training consisted in performing 12 sessions of 55 min within 4 weeks. In each session, HYPO ran 24 min at 70% of maximal O_2 consumption ($\dot{V}_{O_2max}$) with a breath holding at functional residual capacity whereas CONT breathed normally. A $\dot{V}_{O_2max}$ and a time to exhaustion test (TE) were performed before (PRE) and after (POST) the training period. There was no change in $\dot{V}_{O_2max}$, lactate threshold or TE in both groups at POST vs. PRE. At maximal exercise, blood lactate concentration was lower in CONT after the training period and remained unchanged in HYPO. At 90% of maximal heart rate, in HYPO only, both pH (7.36 ± 0.04 vs. 7.33 ± 0.06; $p<0.05$) and bicarbonate concentration (20.4 ± 2.9 mmol L^{-1} vs. 19.4 ± 3.5; $p<0.05$) were higher at POST vs. PRE. The results of this study demonstrate that VH training did not improve endurance performance but could modify the glycolytic metabolism. The reduced exercise-induced blood acidosis in HYPO could be due to an improvement in muscle buffer capacity. This phenomenon may have a significant positive impact on anaerobic performance.

Keywords: Training with voluntary hypoventilation; Hypoxic training; Reduced breathing frequency; Hypoxemia; Hypercapnia; Exercise; Athletes

1. Introduction

Prolonged exposure to hypoxic conditions induces an increase in haemoglobin concentration and red blood cell mass (Berglund, 1992) which are determinant factors of endurance performance (Buick et al., 1980; Kanstrup and Ekblom, 1984). However, chronic hypoxia has also deleterious effects: decrease of training intensity which could lead to detraining, acute mountain sickness, difficulties in acclimatization (Boning, 1997), and at higher altitudes deterioration of muscle tissue (Green et al., 1989; Hoppeler et al., 1990).

To obtain beneficial effects of a prolonged exposure to hypoxia without undergoing the detrimental effects, new ways of hypoxic training have appeared these last few years such as the "living high training low" concept, first proposed by Levine and Stray-Gundersen (1997) or the "living low training high" method (LLTH) (Geiser et al., 2001). The latter consists in training under hypoxic conditions, generally in a hypobaric chamber or by breathing a hypoxic gas mixture, and remaining at sea level the rest of the time. Although improvements of aerobic (Melissa et al., 1997; Meeuwsen et al., 2001; Dufour et al., 2006) or anaerobic performance (Meeuwsen et al., 2001) have been reported at sea level, this way of training is still controversial. Both enzymatic activity, namely citrate synthase, and myoglobin content have also been found to increase (Terrados et al., 1990). However, the access to high altitude is impossible in many countries and the devices which create a hypoxic environment are quite expensive.

Nevertheless, it is possible to generate hypoxemia without being placed in a hypoxic environment, by voluntarily reducing the breathing frequency. While former studies did not report a significant arterial desaturation in these conditions (Craig, 1980; Dicker et al., 1980; Holmer and Gullstrand, 1980), a recent one reported that a severe hypoxemia [arterial O_2 saturation (Sa_{O_2})

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<88% (Dempsey and Wagner, 1999)] could be obtained by a prolonged expiration down to the residual volume (RV) (Woorons et al., 2007). Actually, to get a significant drop in Sa_{O_2}, two conditions have to be met: (1) the voluntary hypoventilation (VH) must be carried out at low pulmonary volumes, close to functional residual capacity (FRC) or RV (Yamamoto et al., 1987; Woorons et al., 2007). This likely leads to a greater inequality of ventilation perfusion ratio ($\dot{V}/\dot{Q}$) (Morrison et al., 1982), and therefore a widened alveolar-arterial difference for O_2 (PA_{O_2}-Pa_{O_2}). (2) VH should not be performed in supine position, like in swimming, where $\dot{V}/\dot{Q}$ is probably more homogeneous than in a sitting or standing position, which diminishes PA_{O_2}-Pa_{O_2} and consequently leads to a higher Sa_{O_2}.

Training with VH began to be used in swimming in the early seventies (Counsilman, 1975) and became later in this sport a more classical training method which remains frequently used nowadays. Using this method, trainers aim at reducing the oxygen availability and consequently expect a greater stimulation of anaerobic metabolism than during exercises with normal breathing. Nevertheless, the specific respiration of swimmers, carried out at high pulmonary volumes close to total lung capacity, and their supine position probably prevent them from hypoxemia. Thus, it is unlikely to get from VH training the same benefits as with the LLTH method. Unfortunately, to our knowledge, no study has ever investigated the effects of such a training method so far. Furthermore, it would be interesting to focus on other sports like running or cycling where VH training is not known to be performed and where hypoxemia is more likely to develop when voluntarily reducing the breathing frequency. In these conditions, as recently suggested (Woorons et al., 2007), one could actually have a greater stimulation of anaerobic metabolism as compared with exercise in normal breathing.

If VH training appeared efficient, it could open new perspectives in training methods. Thus, the objective of the present study was to assess the effects of a 4-week training with VH in runners using breath holding at FRC. We hypothesized that if VH training led to muscle adaptations, it could improve performance and induce changes in anaerobic and/or aerobic parameters.

2. Methods

2.1. Subjects

Fifteen male runners volunteered to participate in this study. A medical questionary as well as a full medical examination revealed that the subjects presented no sign or a history of cardiovascular or respiratory disease. A full interview concerning the training and competitive history of the runners showed that they had a moderate and homogenous level of performance and had been training three to four times a week for at least 1 year. Their training was performed at mixed intensities with about 80–85% of the training sessions at low and moderate intensities [60–85% of maximal O_2 consumption ($\dot{V}_{O_2max}$)] and 15–20% at high intensity (90–100% $\dot{V}_{O_2max}$). The runners were divided into two groups: voluntary hypoventilation group (HYPO, $n = 7$) and control group (CONT, $n = 8$). Both groups were well matched regarding the training history and $\dot{V}_{O_2max}$ of the subjects. The characteristics of HYPO (mean ± S.D.) were: age 27.1 ± 6.0 years, height 180.8 ± 4.5 cm, weight 75.6 ± 8.4 kg and $\dot{V}_{O_2max}$ 54.1 ± 2.4 mL kg^{-1} min^{-1}. Runners in CONT were 30.6 ± 3.9-year-old, 178.3 ± 5.8 cm in height, 69.6 ± 5.5 kg in weight, and had a $\dot{V}_{O_2max}$ of 54.0 ± 4.7 mL kg^{-1} min^{-1}. All the participants 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.

2.2. Study design

The experiment consisted in performing 12 training sessions within 4 weeks and to train three times a week. The duration of each training session was about 55 min. The sessions took partly place in an outdoor athletics track and were all supervised in order to rigorously check the work of all the runners. For both groups, every training was organised as follows:

(1) 15 min at 60% of the velocity corresponding to $\dot{V}_{O_2max}$ ($v\dot{V}_{O_2max}$),
(2) 24 min at 70% $v\dot{V}_{O_2max}$,
(3) 15 min at 60% $v\dot{V}_{O_2max}$.

HYPO had to carry out the second part of the training with a 4-s breath holding at FRC: after a normal expiration of a few $1/10^e$ s, the subjects hold their breath at FRC the remaining time and then briefly inspired at the end of the fourth second.

In order to precisely control the time of expiration/inspiration we used the stride frequency calculated at the running speed of the experiment. We then assessed the stride number in 4 s. The inspiration was carried out within one or two strides. To avoid the harmful effects of hypoventilation induced hypercapnia and especially headaches, hypoventilation was not performed continuously for 24 min but divided into four 5-min epochs, each separated by 1-min epochs in normal breathing and at the same running intensity. Every 5-min epoch was organised in the same way as in a previous study (Woorons et al., 2007): 15 s in normal breathing followed by 45 s with a breath holding at FRC.

2.3. Measurements

Four days before and 2 days after the training period, the subjects performed a $\dot{V}_{O_2max}$ test in a laboratory. Two days after the laboratory tests, a time to exhaustion test (TE) at 100% $v\dot{V}_{O_2max}$ was undertaken outdoor.

2.3.1. Training

For all the subjects, we measured arterial oxygen saturation with a pulse oximeter (Sp_{O_2}) (Pulsox 3i, Konica Minolta, Japan) during the second part of every training session. The data were monitored and recorded second by second, then transferred on a computer at the end of each session. We used this device to check that the subjects performed the respiratory technique efficiently and managed to get a significant arterial desaturation. End-tidal carbon dioxide pressure (PET_{CO_2}) was not measured in the present study, to control the level of hypercapnia. However,

we assessed and presented the data of PET_{CO_2} during exercise with VH in our previous study (Woorons et al., 2007).

2.3.2. $\dot{V}_{O_2max}$ test

To measure $\dot{V}_{O_2max}$, the subjects performed an incremental exhaustive running test on a motorized treadmill (h/p/cosmos mercury med 4.0, Jaeger, Nussdorf-Traunstein, Germany). All the subjects had previously run on a treadmill at least once. Nevertheless, before starting the test, they were asked to run for 3 min on the apparatus at 8, 9 and 10 km h^{-1} (1 min for each speed). This made them re-familiarize with the apparatus and was used for the warming up as well. After a 3-min period of rest in standing position on the apparatus, the test began at a speed of 11 km h^{-1} for 3 min. The speed was then incremented by 1 km h^{-1} at each following stage. All the stages had a duration of 3 min and were separated by a 1-min period of rest in standing position without any movement. The slope of the treadmill was established at 1% and kept constant during the whole test. We opted for this strategy rather than raising the slope in order to have a precise measure of $v\dot{V}_{O_2max}$. The subjects were strongly encouraged to run until exhaustion, i.e. they could no longer follow the speed. Thereafter, they recovered by walking at 5 km h^{-1} for 3 min.

2.3.2.1. Cardirorespiratory parameters. Oxygen consumption ($\dot{V}_{O_2}$), minute ventilation ($\dot{V}_E$), end-tidal O_2 pressure (PET_{O_2}) and PET_{CO_2} 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. Expired gases were collected into a metabograph (Oxycon, Jaeger, Wuerzburg, Germany). A 12-lead electrocardiogram as well as the cardiac frequency (fH) were recorded continuously. For assessment of $\dot{V}_{O_2max}$, at least two of the three criteria were met: (1) an age predicted maximum cardiac frequency (fH_{Tmax}) (220-age) in excess of 90% (2) $R \geq 1.10$ and (3) a plateau (≤150 mL increase) in $\dot{V}_{O_2}$ with an increase in workload.

Sp_{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 the accuracy of which 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. The breath-by-breath measurements, fH and Sp_{O_2} were averaged over 30-s intervals.

2.3.2.2. Arterialized blood gases, lactate concentration and venous sampling. Three capillary blood sampling (95 μL) were drawn from a prewarmed earlobe with a vasodilating capsaicin (1) at rest in seating position, (2) during exercise at an intensity corresponding to 90% of fH_{Tmax} and (3) at the second minute of recovery. Blood gases were immediately analysed for arterial oxygen pressure (Pa_{O_2}), arterial carbon dioxide pressure (Pa_{CO_2}), Sa_{O_2}, pH, arterial $P50_a$ and lactate ([La]), bicarbonate (HCO_3^-) and haemoglobin (Hb) concentration (Radiometer ABL800 Flex, Copenhagen). We also performed a fingertip blood sample (35 μL) at the end of each stage and at maximal exercise to obtain further values of [La] (Radiometer ABL800 Flex). We established the lactate kinetics, and the lactate threshold (LT) was assessed *a posteriori* by two independent observers. LT was defined as the velocity at which an increase in [La] corresponding to at least 1 mmol L^{-1} occurred between 3 and 5 mmol L^{-1} (Aunola and Rusko, 1992). Thus, for every subject, at PRE and POST, we first identified the velocity at the breakpoint and then took into account the cardiorespiratory parameters and [La] at this velocity. Finally, a venous blood sample was drawn at rest from an antecubital vein to assess venous P50 ($P50_v$).

2.3.3. Time to exhaustion test (TE) at 100% $v\dot{V}_{O_2max}$

The test was carried out on an outdoor 400 m athletics track. Following a 10-min warm-up at 60% $v\dot{V}_{O_2max}$, the runners had to sustain an intensity of 100% $v\dot{V}_{O_2max}$ as long as possible. The running velocity was controlled thanks to an audiovisual mark system. The subjects were verbally encouraged to run until exhaustion.

2.4. Statistical analyses

We first checked that the data had a normal distribution. We then used a two-way ANOVA for repeated measures in order to assess the differences between HYPO and CONT and between the tests before (PRE) and after (POST) the training period within each group. A Pearson linear regression analysis was performed to determine whether there was a relationship between variables. Statistical analyses were performed by using Statistica 5.5 software. All the data are expressed as the mean ± S.E. The level of significance was established at $p < 0.05$ for all statistics.

3. Results

3.1. Training

All the subjects of HYPO demonstrated a severe hypoxemia during their training. Fig. 1 presents the mean Sp_{O_2} in HYPO and CONT during a training session.

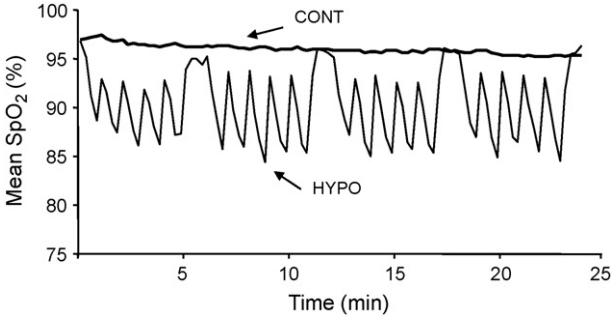

Fig. 1. Mean arterial O_2 saturation (Sp_{O_2}) measured during a training session in the control (CONT) and the voluntary hypoventilation (HYPO) group (standard error is not shown for more clarity).

Table 1
Maximal parameters during the treadmill test

	PRE	POST
$\dot{V}_{O_2max}$ (mL kg^{-1} min^{-1})		
HYPO	54.1 ± 2.4	53.4 ± 3.2
CONT	54.0 ± 4.7	52.9 ± 5.0
V_{PEAK} (km h^{-1})		
HYPO	16.5 ± 0.5	16.9 ± 0.4
CONT	16.8 ± 0.4	16.9 ± 0.5
$\dot{V}_E$ (L min^{-1})		
HYPO	151.3 ± 6.9	149.9 ± 5.9
CONT	147.9 ± 6.4	143.5 ± 5.4
fH (bpm)		
HYPO	196.3 ± 2.9	195.3 ± 2.9
CONT	194.1 ± 3.8	193.0 ± 3.5
Sp_{O_2} (%)		
HYPO	93.4 ± 0.6	93.3 ± 0.9
CONT	92.4 ± 3.2	91.9 ± 3.8
PET_{CO_2} (mmHg)		
HYPO	33.5 ± 1.3	35.4 ± 1.9
CONT	31.5 ± 0.9	34.3 ± 1.1
PET_{O_2} (mmHg)		
HYPO	116.4 ± 1.4	118.0 ± 1.8
CONT	118.1 ± 0.7	118.2 ± 0.8

Values are mean ± S.E. $\dot{V}_{O_2max}$, maximal oxygen consumption; V_{PEAK}, velocity at exhaustion; $\dot{V}_E$, expired minute volume of gas at body temperature and pressure saturated; fH, cardiac frequency; Sp_{O_2}, arterial O_2 saturation; PET_{CO_2}, end-tidal carbon dioxide pressure; PET_{O_2}, end-tidal O_2 pressure; PRE, before the training period; POST, after the training period.

Table 2
Parameters at lactic threshold

	PRE	POST
$\dot{V}_{O_2}$ (mL kg^{-1} min^{-1})		
HYPO	44.4 ± 0.8	43.7 ± 0.9
CONT	45.7 ± 1.6	45.8 ± 1.8
% $\dot{V}_{O_2max}$		
HYPO	82.7 ± 2.1	82.3 ± 2.0
CONT	82.1 ± 1.4	83.2 ± 1.7
Velocity (km h^{-1})		
HYPO	13.5 ± 0.3	13.5 ± 0.3
CONT	13.4 ± 0.3	13.4 ± 0.3
$\dot{V}_E$ (L min^{-1})		
HYPO	90.2 ± 5.0	88.8 ± 3.1
CONT	90.1 ± 6.2	94.8 ± 3.1
fH (bpm)		
HYPO	176.8 ± 3.7	175.5 ± 2.6
CONT	175.0 ± 5.4	174.0 ± 3.8
Sp_{O_2} (%)		
HYPO	94.2 ± 0.6	94.8 ± 0.7
CONT	94.3 ± 0.8	95.7 ± 0.8[a]
PET_{CO_2} (mmHg)		
HYPO	48.0 ± 1.8	47.6 ± 1.3
CONT	42.3 ± 1.8	41.1 ± 0.8
PET_{O_2} (mmHg)		
HYPO	100.2 ± 1.7	103.5 ± 1.8[a]
CONT	104.6 ± 2.0	108.0 ± 0.8

Values are mean ± S.E. $\dot{V}_{O_2}$, oxygen consumption; $\dot{V}_E$, expired minute volume of gas at body temperature and pressure saturated; fH, cardiac frequency; [La], blood lactate concentration; Sp_{O_2}, arterial O_2 saturation; PET_{CO_2}, end-tidal carbon dioxide pressure; PET_{O_2}, end-tidal O_2 pressure; PRE, before the training period; POST, after the training period.
[a] Significant difference from PRE ($p < 0.05$).

3.2. Cardiorespiratory variables and [La] values at exhaustion and at LT

There was no difference between groups in any parameter both at PRE and POST (Tables 1 and 2). $\dot{V}_{O_2max}$ was not different within each group at POST compared with PRE (Table 1).

At maximal exercise, we did not find any change between POST and PRE for $\dot{V}_E$, fH, Sp_{O_2}, PET_{CO_2} and PET_{O_2} in either HYPO or CONT (Table 1). On the other hand, [La] was lower at POST in CONT and unchanged in HYPO (Fig. 2).

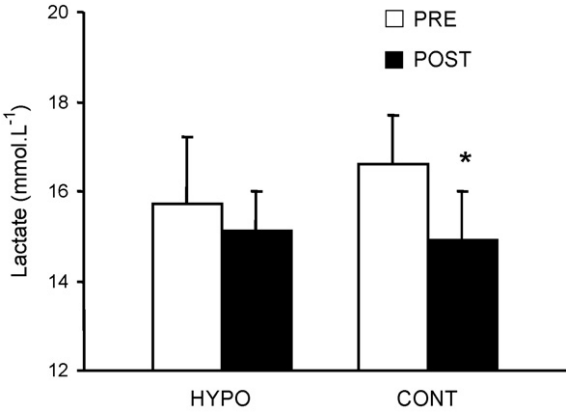

Fig. 2. Blood lactate concentration at maximal exercise of the $\dot{V}_{O_2max}$ test in voluntary hypoventilation (HYPO) and control (CONT) group before (PRE) and after (POST) the training period; *significant difference from PRE ($p < 0.05$).

At LT (Table 2), $\dot{V}_{O_2}$, $\dot{V}_E$, fH, PET_{CO_2} and PET_{O_2} remained unchanged in both groups after the training period. Sp_{O_2} was higher in CONT at POST vs. PRE but was not different in HYPO. There was a significant difference in [La] between PRE and POST in HYPO (4.6 ± 0.4 vs. 3.9 ± 0.2 mmol L^{-1}; $p < 0.01$) but not in CONT (4.8 ± 0.3 vs. 4.9 ± 0.4 mmol L^{-1}).

3.3. Arterialized blood gases and venous sampling

The results are presented in Table 3 and Fig. 3.

At rest, 90% fH_{Tmax} and 2-min recovery, there was no difference between groups in any parameter at either PRE or POST.

At rest, $P50_v$ was not different before and after the training period both in HYPO (25.7 ± 1.3 vs. 26.0 ± 2.4 mmHg) and CONT (25.9 ± 0.9 vs. 26.2 ± 1.9 mmHg). We did not find any difference between POST and PRE in any parameter for both groups.

At 90% fH_{Tmax}, Pa_{O_2}, Pa_{CO_2}, Sa_{O_2}, Hb and $P50_a$ remained unchanged at POST in both groups. pH and HCO_3^- were higher at POST in HYPO ($p < 0.05$) and were not different in CONT. There was no difference in [La] in both groups between tests despite a tendency to a lower [La] in HYPO ($p = 0.07$).

Table 3
Arterialized blood gases

	Rest		90% fH_{Tmax}		Recovery	
	PRE	POST	PRE	POST	PRE	POST
Pa_{O_2} (mmHg)						
HYPO	87.3 ± 1.1	85.6 ± 2.9	82.0 ± 2.7	82.5 ± 2.7	89.9 ± 1.6	88.2 ± 4.5
CONT	90.9 ± 2.4	87.7 ± 1.9	78.1 ± 2.9	83.0 ± 1.8	91.3 ± 3.2	93.7 ± 3.3
Pa_{CO_2} (mmHg)						
HYPO	39.6 ± 0.9	39.4 ± 1.0	38.6 ± 1.5	38.7 ± 1.7	30.9 ± 0.7	30.2 ± 2.0
CONT	37.3 ± 1.2	36.7 ± 0.6	36.6 ± 1.1	35.8 ± 1.0	27.8 ± 1.4	28.6 ± 1.2
Sa_{O_2} (%)						
HYPO	96.0 ± 0.1	95.9 ± 0.3	95.0 ± 0.3	95.1 ± 0.3	94.7 ± 0.2	94.1 ± 0.5
CONT	96.5 ± 0.2	95.9 ± 0.2	94.2 ± 0.4	94.7 ± 0.3	94.3 ± 0.3	94.6 ± 0.3
pH						
HYPO	7.41 ± 0.01	7.42 ± 0.01	Fig. 3	Fig. 3	7.22 ± 0.02	7.23 ± 0.02
CONT	7.42 ± 0.01	7.43 ± 0.01	Fig. 3	Fig. 3	7.22 ± 0.02	7.22 ± 0.02
HCO_3^- (mmol L^{-1})						
HYPO	25.0 ± 0.4	25.3 ± 0.2	Fig. 3	Fig. 3	12.4 ± 0.8	12.5 ± 1.0
CONT	24.0 ± 0.6	24.2 ± 0.4	Fig. 3	Fig. 3	11.1 ± 0.8	11.6 ± 0.8
[La] (mmol L^{-1})						
HYPO	0.9 ± 0.1	0.9 ± 0.1	Fig. 3	Fig. 3	14.5 ± 1.4	14.0 ± 0.9
CONT	1.0 ± 0.2	0.9 ± 0.1	Fig. 3	Fig. 3	15.7 ± 1.1	13.8 ± 1.0[a]
Hb (g dL^{-1})						
HYPO	15.6 ± 0.3	15.3 ± 0.4	16.5 ± 0.4	16.4 ± 0.3	16.6 ± 0.4	16.5 ± 0.4
CONT	15.6 ± 0.3	15.7 ± 0.3	16.1 ± 0.3	16.3 ± 0.3	16.2 ± 0.2	16.2 ± 0.4
$P50_a$ (mmHg)						
HYPO	25.9 ± 0.3	24.8 ± 0.5	25.8 ± 1.2	25.7 ± 0.6	29.7 ± 0.5	29.4 ± 1.0
CONT	25.3 ± 0.3	24.7 ± 0.4	25.8 ± 0.8	26.0 ± 0.9	31.5 ± 0.7	31.1 ± 0.9

Values are mean ± SE. Pa_{O_2}, arterial oxygen pressure; Pa_{CO_2}, arterial carbon dioxide pressure; Sa_{O_2}, arterial O_2 saturation; HCO_3^-, bicarbonate concentration; [La], blood lactate concentration; Hb, haemoglobin concentration; $P50_a$, P_{O_2} for Sa_{O_2} = 50%; fH_{Tmax}, age predicted maximum cardiac frequency; PRE, before the training period; POST, after the training period.

[a] Significant difference from PRE ($p < 0.05$).

At 2-min recovery, there was no difference in any parameter at POST compared with PRE in HYPO. In CONT, [La] was lower at POST ($p < 0.01$) whereas all the other parameters remained unchanged.

3.4. Performance indices

There was no difference between HYPO and CONT in $v\dot{V}_{O_2max}$, TE and the velocity at LT both at PRE and POST. Within each group, we did not find any change after the training period in the velocity at LT (Table 2) and $v\dot{V}_{O_2max}$ (HYPO: 16.1 ± 0.5 vs. 16.3 ± 0.5 km h^{-1}; CONT: 16.4 ± 0.5 vs. 16.4 ± 0.5 km h^{-1} at PRE and POST respectively). The maximal velocity reached during the $\dot{V}_{O_2max}$ test (V_{PEAK}) remained unchanged at POST in CONT whereas it tended to be higher in HYPO ($p = 0.07$) (Table 1). There was a significant correlation between the change (Δ) in V_{PEAK} and ΔHCO_3^- at 90% fH_{Tmax} in HYPO ($r = 0.90$; $p < 0.01$) (Fig. 4). TE at 100% $v\dot{V}_{O_2max}$ was not different before and after the training period both in HYPO (427 ± 43 vs. 471 ± 52 s) and CONT (467 ± 48 vs. 465 ± 33 s).

4. Discussion

To our knowledge, this study was the first to investigate the effects on performance of a training with VH at low pulmonary volumes. The experiment consisted in training 12 sessions within 4 weeks and to carry out a breath holding at FRC for 24 min at each session. This respiratory technique was expected to induce a severe arterial desaturation (Yamamoto et al., 1987; Woorons et al., 2007) which, when including in a training programme, could mimic the effects of the LLTH method. Furthermore, this study focused on runners since exercise-induced hypoxemia may be greater in running than in cycling (Rice et al., 2000) and does not seem to occur in swimming when reducing the breathing frequency (Dicker et al., 1980; Holmer and Gullstrand, 1980). The minimal Sp_{O_2} fell below 88% in all subjects during training, even though there were some individual differences and that the oximeter may not perfectly reflect the actual Sa_{O_2} when used in outdoor conditions. While some runners maintained their Sp_{O_2} at about 87–88% during VH, others, and particularly two of them, regularly demonstrated a spectacular arterial desaturation, reaching a Sp_{O_2} down to 78–79%. Such a Sp_{O_2} could be obtained at simulated altitudes of about 1800–2800 m in trained subjects exercising at 70% of $\dot{V}_{O_2max}$ (Woorons et al., 2006).

Contrary to one of the hypotheses we made, VH training failed to induce any improvement in aerobic performance. We did not report any change in $\dot{V}_{O_2max}$, $v\dot{V}_{O_2max}$, the velocity at LT or TE at 100% $v\dot{V}_{O_2max}$. Furthermore, as expected regarding the

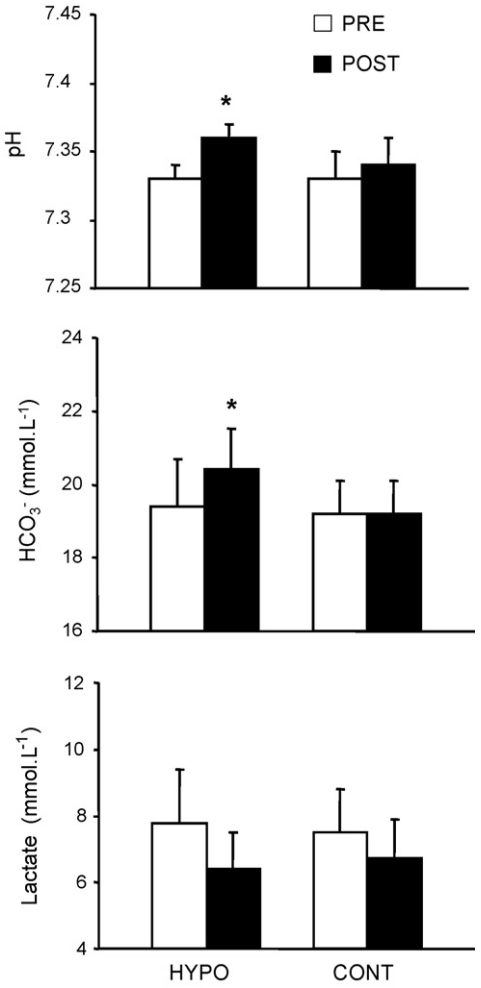

Fig. 3. pH, blood bicarbonate (HCO_3^-) and lactate concentration at 90% of age predicted maximum cardiac frequency in voluntary hypoventilation (HYPO) and control (CONT) group before (PRE) and after (POST) the training period; *significant difference from PRE ($p < 0.05$).

short exposure time to hypoxia (Vallier et al., 1996; Emonson et al., 1997), there was no alteration in haemoglobin concentration. Some studies investigating the effects of training under hypoxic conditions found an increase in endurance performance at sea level (Melissa et al., 1997; Meeuwsen et al., 2001; Dufour et al., 2006) while other studies failed to find any improvement (Vallier et al., 1996; Ventura et al., 2003; Truijens et al., 2003). It is possible that the intensity of exercise in the context of LLTH should be at least at the second ventilatory threshold to get significant changes (Dufour et al., 2006). However, it seems difficult to ask the subjects using VH, regarding the exercise hardness, to perform this respiratory technique at higher intensities. The combined stress of exercising at high intensity and hypoventilating at the same time would be difficult to endure by athletes and may induce overtraining.

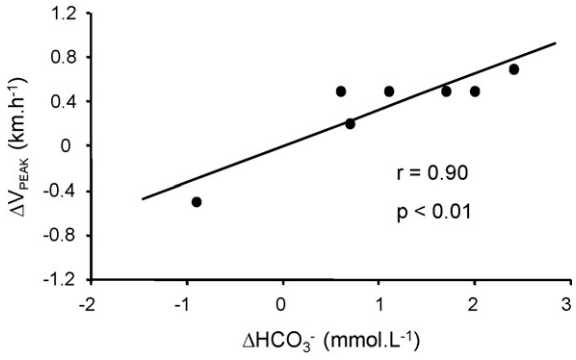

Fig. 4. Relationship between the change in maximal velocity reached during the $\dot{V}_{O_2max}$ test (ΔV_{PEAK}) and the change in blood bicarbonate concentration (ΔHCO_3^-) at 90% of age predicted maximum cardiac frequency in HYPO.

There are differences in the level of hypoxia between VH training and LLTH. In the former method, arterial O_2 desaturation is not undergone continuously during exercise since there is an alternance of normal and reduced breathing frequency periods. Thus, the time spent in hypoxia is lower in VH training than LLTH. One could therefore think that increasing the frequency and time of VH may be a solution to get an improvement in aerobic capacity. However, in our study, VH was included in every session during the whole training period and once again it appeared risky to increase the total duration of VH. Some of the subjects indeed complained from being physically and mentally tired at the end of the experiment. This makes us think that the time duration and weekly frequency we chose for the work with VH was probably maximal. Moreover, some studies have reported significant changes at the cellular level even after short time exposures (30 min) to hypoxia performed three to five times a week (Terrados et al., 1990; Geiser et al., 2001). One could also point out that the degree of arterial desaturation may not be severe enough. However, LLTH seems to be actually effective at a moderate rather than at a high degree of hypoxia (Terrados et al., 1990; Meeuwsen et al., 2001). Finally, if VH training did not improve aerobic performance at sea level, it might increase $\dot{V}_{O_2max}$ in hypoxia, as shown for LLTH (Geiser et al., 2001; Dufour et al., 2006).

A significant change reported in the present study concerns the lactate concentration. While [La] was unchanged in HYPO at maximal exercise and at 2-min recovery after training, CONT demonstrated a lower [La]. A first hypothesis explaining this phenomenon could be that the training of CONT may have induced a detraining of anaerobic glycolysis. The running intensity was indeed of 70% $v\dot{V}_{O_2max}$ at the most, that is to say a moderate one which unlikely stimulates anaerobic glycolysis. Even though the subjects mostly trained at low or moderate intensity before engaging the experiment, they also performed a small part of their training at high intensities, where [La] is higher. Therefore, a lower [La] could be expected at the end of the experiment. Surprisingly, this drop in [La] did not occur in HYPO. It is therefore possible, as recently suggested (Woorons et al., 2007), that a training with VH may actually enhance the lactate concentration in the working muscles and finally help to maintain the power of glycolytic metabolism. Furthermore, it has already been shown that performing exercises with similar intensities and Sp_{O_2} can induce higher [La] than exercises with normal Sp_{O_2} (Peltonen et al., 1999). Nevertheless, a placebo effect cannot be ruled out. The subjects could not obviously be blinded in this kind of experiment. It is possible that, knowing they were testing an original training method, there was a

psychological effect which made them go further during the test.

The reduced exercise-induced blood acidosis in HYPO at 90% fH_{Tmax} was an interesting and unexpected modification induced by VH training. The higher pH was accompanied by a higher HCO_3^- and a tendency to a lower [La]. These results could reflect an improvement in the buffer capacity, even though pH, [La] and HCO_3^-concentration were unchanged in HYPO after 2 min of recovery and therefore probably at exhaustion. Thus, one could argue that training with VH delays the metabolic acidosis but does not decrease it at maximal exercise. Previously, an improved buffer capacity had been found especially after a high intensity training (Edge et al., 2006a) or after training at altitude (Mizuno et al., 1990). Extra cellular, mainly Hb and HCO_3^-, as well as intra-cellular components, such as proteins, phosphates, amino acids or HCO_3^-, are involved in the buffering system. In the present study, if VH had altered blood HCO_3^-, this would probably have been noted at rest but there was no difference in this parameter between PRE and POST. Moreover, we reported no change in Hb after training. We therefore suggest that the factors participating in the weaker blood acidosis after VH training may have an origin within the muscular cell.

A delayed acidosis could be interesting for anaerobic performance. Hydrogen ions may accumulate more slowly and allow the athletes to continue to exercise longer or at a higher intensity for a given distance. Although we did not directly measure anaerobic performance, one could think that a better buffer capacity should have had an impact on both V_{PEAK} and TE at 100% $v\dot{V}_{O_2max}$. However, training did not significantly change these parameters. Nevertheless, TE is a poor indicator of anaerobic performance since it may more depend on the level of LT than on the accumulation of muscle metabolites (Edge et al., 2006b). On the other hand, V_{PEAK}, which is frequently reached beyond $v\dot{V}_{O_2max}$, may reflect a contribution of anaerobic glycolysis. While we did not report any significant mean improvement in HYPO, V_{PEAK} was increased from 0.5 km h^{-1} on average in six among the seven subjects of this group. The only subject who did not enhance his performance was the one who complained the most from being tired at the end of the experiment. Furthermore, the correlation we found between ΔV_{PEAK} and ΔHCO_3^- at 90% fH_{Tmax} in HYPO, argues in favor of a better anaerobic performance induced by an improved buffering capacity following VH training. Finally to assess the effects on anaerobic performance a specific test like a Wingate or a 400 or 800 m run would certainly be useful.

VH training also differs from LLTH since it creates hypercapnia. An intermittent exposure to hypercapnia during exercise may induce some adaptations at the cellular or vascular level. It could be expected that repeated exercises with hypercapnic acidosis might lead to physiological adaptations limiting the blood acidosis. Unfortunately, to our knowledge, no study has ever investigated the effects of exercise under intermittent exposure to hypercapnia so far. When dealing with VH, one could make a parallel with what happens in apnea which also generates asphyxia (hypercapnia plus hypoxia) (Clanton and Klawitter, 2001). A study reported that a breath hold training carried out at 30% $\dot{V}_{O_2max}$ in triathletes led to a lower blood acidosis during dynamic apnea (Joulia et al., 2003). The authors attributed this phenomenon to a reduced production of lactic acid by exercising muscles and/or the increase of its catabolism by other tissues. However, it is difficult to know whether it was the consequence of hypoxemia, hypercapnia or both. Likewise, it is difficult to distinguish the respective role played by hypoxia and hypercapnia during VH training.

The results of this study provide interesting information concerning the possible use of VH training. It is probably useless for endurance athletes given the hardness of such training and the absence of benefit. On the other hand, athletes involved in short time duration activities or even in intermittent sports, where anaerobic glycolysis plays a major role, may find interest to carry out a VH training. Furthermore, it is sometimes traumatizing to repeatedly perform exercises at high intensities in order to stimulate anaerobic glycolysis. Regular training with VH could thus reduce the risk of injury by exercising at moderate intensities. Nevertheless, there is still a lot to investigate concerning the practical implication of VH training. The optimal weekly frequency and duration per session have to be determined. Furthermore, as mentioned before, hypoventilation induces hypercapnia. An elevated Pa_{CO_2} generally produces an increase in cerebral blood flow leading to headaches and a higher pressure of cerebrospinal fluid (West, 1997). Therefore, the time duration at which hypoventilation can be performed continuously without undergoing the harmful effect of hypercapnia is also of interest. Anyway, for more safety, it is certainly wiser not to perform continuously the work with VH but alternate with periods of normal breathing like in the present study. Finally, an interesting question concerns the combination of VH with the other forms of training.

In summary, this study showed that a 4-week training with voluntary hypoventilation carried out at low pulmonary volumes did not alter the markers of aerobic performance such as $\dot{V}_{O_2max}$, lactate threshold or time to exhaustion at 100% $\dot{V}_{O_2max}$. After VH training, lactate at maximal exercise did not decrease and exercise-induced blood acidosis at 90% fH_{Tmax} was reduced. This latter phenomenon may have a significant positive impact on anaerobic performance which should be confirmed by further studies.

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