

# Hypoventilation Training at Supramaximal Intensity Improves Swimming Performance

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<sup>1</sup>Pluridisciplinary Research Unit “Sport Health and Society” (URePSSS), EA 7369, Lille University, FRANCE; <sup>2</sup>Association for Research and Promotion of Hypoventilation Training (ARPEH), Lille, FRANCE; <sup>3</sup>Laboratory “Hypoxia and Lung,” EA 2363, Paris 13 University, Bobigny, FRANCE; and <sup>4</sup>Laboratory Mobility, Aging and Exercise (MOVE), EA 6314, Faculty of Sport Sciences, Poitiers University, FRANCE

## ABSTRACT

WOORONS, X., P. MUCCI, J. P. RICHALET, and A. PICHON. Hypoventilation Training at Supramaximal Intensity Improves Swimming Performance. *Med. Sci. Sports Exerc.*, Vol. 48, No. 6, pp. 1119–1128, 2016. **Purpose:** This study aimed to determine whether hypoventilation training at supramaximal intensity could improve swimming performance more than the same training conducted under normal breathing conditions. **Methods:** For a 5-wk period, 16 triathletes (12 men and 4 women) were asked to include one supramaximal set of 12 to 20 × 25-m front crawl swimming twice a week into their usual swimming session, performed either with hypoventilation at low lung volume (VHL group) or with normal breathing (CONT group). Before (Pre) and after (Post) training, all triathletes performed all-out front crawl trials for 100, 200, and 400 m. **Results:** Time performance was significantly improved in VHL in all trials (100 m:  $-3.7 \pm 3.7$  s [ $-4.4\% \pm 4.0\%$ ]; 200 m:  $-6.9 \pm 5.0$  s [ $-3.6\% \pm 2.3\%$ ]; 400 m:  $-13.6 \pm 6.1$  s [ $-3.5\% \pm 1.5\%$ ]) but did not change in CONT. In VHL, the maximal lactate concentration ( $+2.35 \pm 1.3$  mmol·L<sup>-1</sup> on average) and the rate of lactate accumulation in blood ( $+41.7\% \pm 39.4\%$ ) were higher at Post than at Pre in the three trials, whereas they remained unchanged in CONT. Arterial oxygen saturation, heart rate, breathing frequency, and stroke length were not altered in both groups at the end of the training period. On the other hand, stroke rate was higher at Post compared with Pre in VHL but not different in CONT. The measurements of gas exchange for the 400-m trial revealed no change in peak oxygen consumption as well as in any pulmonary variable in both groups. **Conclusion:** This study demonstrated that VHL training, when performed at supramaximal intensity, represents an effective method for improving swimming performance, partly through an increase in the anaerobic glycolysis activity. **Key Words:** BREATH HOLDING, HYPOXIA, HYPERCAPNIA, EXERCISE, SWIMMERS

In a recent study, it was reported that swimmers could train under hypoxic conditions at sea level through voluntary hypoventilation at low lung volume (VHL), or the so-called exhale-hold technique (37). During submaximal swimming exercise with VHL, arterial oxygen saturation fell as low as 87%, a level considered as severe hypoxemia (4). Under these conditions, there was also an increased lactate concentration, revealing a greater glycolytic activity as compared with the same exercise performed with normal breathing. Such results, already reported in terrestrial activities (36), were original in swimming. Conversely, in studies in which swimmers applied hypoventilation at high lung volume (i.e., inhale-hold), the classic technique used since the 1970s, no hypoxic effect occurred, and lactate concentration was not different (32,34) or even lower than exercise

with normal breathing (15,37). On the other hand, it is noticeable that, irrespective of the lung volume, exercising with hypoventilation always caused a hypercapnic effect, that is, elevated alveolar and arterial blood partial pressure of carbon dioxide (5,15,16,32,34,35,38).

Surprisingly, few studies have investigated the effects of this kind of training. An improved performance has already been reported after several weeks of training with reduced breathing frequency in swimming, but the changes were not greater than those in the group who trained with normal breathing (17,21). This outcome is probably because these studies used hypoventilation at high lung volume. Yet hypercapnia alone may not represent a sufficient stimulus to induce highly beneficial adaptations to performance, as confirmed by another study that dealt with reduced breathing frequency in biking (18). On the other hand, one could expect more significant alterations when hypercapnia is combined with hypoxia. In runners, 4 wk of moderate-intensity training with VHL enabled blood and probably muscle acidosis to be delayed (39), which could be favorable for anaerobic performance.

With regard to its hypoxic effect, VHL training can be considered as an intermittent hypoxic training (IHT) (25). Although some studies have demonstrated that IHT could be more effective for improving sea-level aerobic or anaerobic

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performance than the same training performed in normoxia (8,24,31), many others failed to find so (9,14,26,29,33). However, thoroughly analyzing the literature dealing with IHT, we found that intensity *per se* seems to be the key factor for improving performance (10). The majority of the controlled studies that reported advantageous effects used exercise intensities equal to or higher than the second ventilatory threshold (8,31). Furthermore, some remarkable effects have been highlighted these last 2 yr after training with repeated sprint (i.e., short all-out exertions) in hypoxia (RSH) (11,13), which could be considered as a superior form of IHT. Thus, one could assume that such results could be reproduced through VHL training at high intensity, especially because the intermittent hypercapnic effect may play an additional role in the physiological adaptations leading to improved performance.

To date, to the best of our knowledge, the studies that have investigated the effects of hypoventilation training did not use high-intensity exercises. In swimming, most of the competitive trials are performed at intensities beyond maximal oxygen consumption ( $\dot{V}O_{2max}$ ), thus primarily involving the anaerobic metabolism and stimulating the glycolytic fast-twitch fibers. Because it seems that the effectiveness of the IHT/RSH approach is dependent on the maintenance of high fast-twitch fiber recruitment (10), the aim of the present study was to ascertain the effects of a 5-wk training with VHL at supramaximal intensity (i.e., beyond  $\dot{V}O_{2max}$ ) on swimming performance. We hypothesized that performance could be improved for distances of 100 to 400 m because of increased anaerobic glycolysis, but we did not rule out positive changes at the aerobic level.

## METHODS

**Subjects.** Sixteen triathletes (12 men and 4 women) were recruited to participate in this study. Thirteen of them belonged to the same triathlon team (Wasquehal, northern France), whereas the remaining three were individual competitors. Two of the subjects were male–male dizygotic twins who ranked in the top 10 best 16-yr-old triathletes in

France. Ten competed at a regional or departmental level. The last four were recreational triathletes. On average, the weekly training volume of the participants was two or three training sessions in swimming, two or three training sessions in running, and two training sessions in cycling, corresponding to approximately 8 h·wk<sup>-1</sup>. Within the 4 or 5 wk preceding the experiment, all of the subjects performed one or two high-intensity swimming sessions per week to improve their swimming performance. This level of performance would be representative of that of recreational or regional performances. All participants were sea-level residents and unacclimatized to altitude. They were asked to avoid any exposure to an altitude higher than 1500 m for the entire period of the study. Furthermore, none of them had used hypoventilation training in the previous few months of the study. All the subjects were informed about the nature, conditions, and risks of the experiment and gave their written informed consent. The study was approved by the ethical committee of Ile de France II, Paris, France.

**Study design.** All the testing and training sessions were conducted in two different 25-m swimming pools located in northern France: La Madeleine (altitude = 29 m, water temperature = 28°C) and Wasquehal (altitude = 20 m, water temperature = 27°C). Before beginning the experiment, the subjects came once or twice to the swimming pool to familiarize with the VHL technique and/or to learn swimming with the equipment used for the measurements. For some of them, it was necessary to train swimming while breathing through a snorkel (used for ventilatory recordings) and also making an open turn with the equipment because a standard flip turn could not be made. The experimental protocol (Fig. 1) consisted of performing 10 carefully supervised training sessions, each including a set of supramaximal-intensity exercise, for a 5-wk period. One week before and 1 wk after the training period, all of the subjects participated in two testing sessions separated by 48–72 h. At the end of the first two testing sessions, subjects were matched into pairs for gender, performance level in swimming, and training history. The subjects were then randomly assigned to either the hypoventilation group (VHL) (two women and six men,  $n = 8$ ,

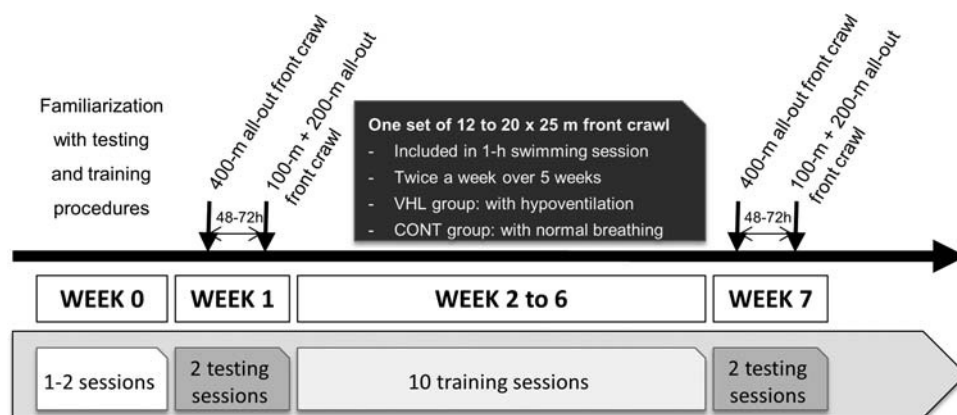


FIGURE 1—Experimental design of the study.

age =  $32.5 \pm 10.7$  yr, height =  $174.9 \pm 6.4$  cm, weight =  $66.0 \pm 6.4$  kg, and  $\dot{V}O_{2\text{peak}} = 51.7 \pm 11.3$  mL·min<sup>-1</sup>·kg<sup>-1</sup> [mean  $\pm$  SD]) or the control group (CONT) (two women and six men,  $n = 8$ , age =  $33.5 \pm 9.3$  yr, height =  $177.6 \pm 11.3$  cm, weight =  $68.1 \pm 12.9$  kg, and  $\dot{V}O_{2\text{peak}} = 51.04 \pm 10.8$  mL·min<sup>-1</sup>·kg<sup>-1</sup>).

**Training sessions.** Twice a week during the 5-wk period, one set of 12 to 20  $\times$  25 m front crawl swimming was included in the regular 1-h training sessions of the subjects. The number of 25-m repetitions was progressively increased during the training period according to the rating of perceived exertion (RPE). Depending on the performance level of the athletes, the turnaround time for each 25 m was set at 30 or 35 s so that the resting period was always between 10 and 15 s. It was established in collaboration with the swimming coach of the team that such resting duration enabled the subjects to maintain a supramaximal speed, i.e., equal or faster than the speed of a 200-m front crawl. It is important to note that the subjects were never asked to perform the 25 m at maximal velocity. The feature of the present study was therefore distinguishable from that of the RSH studies (11,13).

CONT performed the entire set with normal breathing, whereas the VHL group completed the set with hypoventilation at low lung volume. This breathing technique was well described elsewhere (37). Briefly, just before starting each lap, the athletes had to exhale down to functional residual capacity or a little below but without reaching the residual volume. They had then to push off the wall, glide, and swim by holding one's breath until a strong urge to breathe was felt. At that time, after exhaling the remaining air, they were allowed to take an inhalation and reproduce the same exhale-hold procedure till the end of the lap. It is important to note that the subjects were not recommended to hold their breath for as long as possible to avoid asphyxia and to maintain a high swimming velocity throughout the set.

During the entire training, the 1-h swimming sessions were adapted by the coach so as to include the supramaximal set. Each session generally began with a 10-min warm-up. Then, half of the time, the supramaximal set was preceded or followed by a 20- to 30-min aerobic workload. Finally, a 10-min cooldown period ended the session. In accordance with the swimming coach, the subjects received no technical information and did not perform any technical swimming exercise during the entire training period.

**Testing sessions at baseline (Pre) and after (Post) the training period.** Subjects were asked to refrain from any training the day before each testing session and from high-intensity training 48 h before the testing sessions. The participants were also asked to refrain from caffeine or alcohol 48 h before all testing sessions. At the first testing session of both Pre and Post, the subjects performed 400-m front crawl swimming at maximal speed. At the second training session, they first performed an all-out 100-m front crawl followed by an all-out 200-m front crawl approximately

40 min later. The 40-min period separating both trials included 10 min of active recovery in the water preceded and followed by 5–10 min and 20–25 min of rest, respectively. Before each of the three trials, the participants completed a standardized 10-min warm-up at low to moderate intensity plus two to three sprints for 15–20 m. It is important to note that the time trials were conducted differently from normal swimming events because of the equipment carried by the participants for the physiological measurements. The subjects performed the trials one at a time and started in the water instead of the starting blocks. Furthermore, they were instructed to perform an “open turn” alternatively rightward and leftward in order not to twist the cable connected to the measurement devices. Obviously, performance was significantly lower than that during a normal swimming event in these conditions. In the pilot experiment and based on another study (37), we established that the time per lap was 1.5 to 2 s longer. It is also important to mention that each subject always completed the same trial in the same swimming pool, at the same water temperature, and at the same time and day of the week.

**Gas exchange.** Gas exchange was recorded during the 400-m all-out trial through a K4b<sup>2</sup> remote breath-by-breath portable system (Cosmed, Rome, Italy). The device was connected to a snorkel system (Aquatrainer, Cosmed) developed by Keskinen et al. (19). With this Aquatrainer module, gas exchange is measured using inspiratory and expiratory flows. The connection of the inlet and outlet tubes to the K4b<sup>2</sup> 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 expiration and inhalation. The distance between the snorkel mouthpiece and the K4b<sup>2</sup> turbine unit was 128 cm, and the volumes of both the outlet and the inlet tubes were 825 mL. Before each test, we performed the standardized calibration procedures as recommended by the manufacturer (K4b<sup>2</sup>, Cosmed). These procedures included air calibration, turbine calibration with a standard 3000-mL syringe, gas calibration with a certified commercial gas preparation (O<sub>2</sub>: 16%, CO<sub>2</sub>: 5%), and delay calibration to ensure accurate readings during the testing and to check the alignment between the gas flow and the gas concentrations. The breath-by-breath measurements were recorded continuously for tidal volume ( $V_T$ ), breathing frequency ( $B_f$ ), expired ventilation ( $\dot{V}_E$ ), oxygen consumption ( $\dot{V}O_2$ ), carbon dioxide production ( $\dot{V}CO_2$ ), end-tidal O<sub>2</sub> pressure (PETO<sub>2</sub>), and carbon dioxide pressure (PETCO<sub>2</sub>). The ventilatory equivalent ( $\dot{V}_E/\dot{V}O_2$ ) was calculated. Data were averaged and analyzed for the 30-s training period corresponding to the highest values of  $\dot{V}O_2$  ( $\dot{V}O_{2\text{peak}}$ ).

**Arterial oxygen saturation and heart rate.** Arterial oxygen saturation (SpO<sub>2</sub>) and heart rate (HR) were continuously measured during the 100- and 200-m all-out trials via the pulse oximeter Nellcor N-595 (Pleasanton, CA, USA) with the adhesive forehead sensor Max-Fast (Nellcor; Pleasanton). The forehead sensor was waterproofed for use in an aquatic environment, as validated and fully described

previously (37). The same care and procedures were applied to ensure a good quality of the signal and accurate measurements. Both SpO<sub>2</sub> and HR data were analyzed in the last 15 s of each trial.

**Blood lactate concentration and rate of lactate accumulation in blood.** Between the third and the fourth minutes after each trial, two blood samples (5 μL) were collected from the earlobe of the subjects to obtain blood lactate concentration ([La]). The samples were collected with a portable blood lactate analyzer (Lactate Pro). To improve the accuracy and reliability of the measurements, maximal [La] ([La]<sub>max</sub>) was determined by averaging the values of the two successive blood samples. On the basis of the works of di Prampero and Ferretti (6), we also calculated the rate of lactate accumulation in blood ([La]<sup>Rt</sup>) from the ratio of [La]<sub>max</sub> to the duration of the exercise for each swim trial. This rate is considered to be directly proportional to the rate of lactate accumulation in 1 kg of body mass (6).

**Stroke length, stroke rate, and breathing frequency.** The entire 100- and 200-m swim trials of each subject were filmed (video camera; Samsung, South Korea) to calculate *a posteriori* the time, the number of strokes, and the number of breaths for each lap. Average stroke length, stroke rate (SR), and B<sub>F</sub> were then determined.

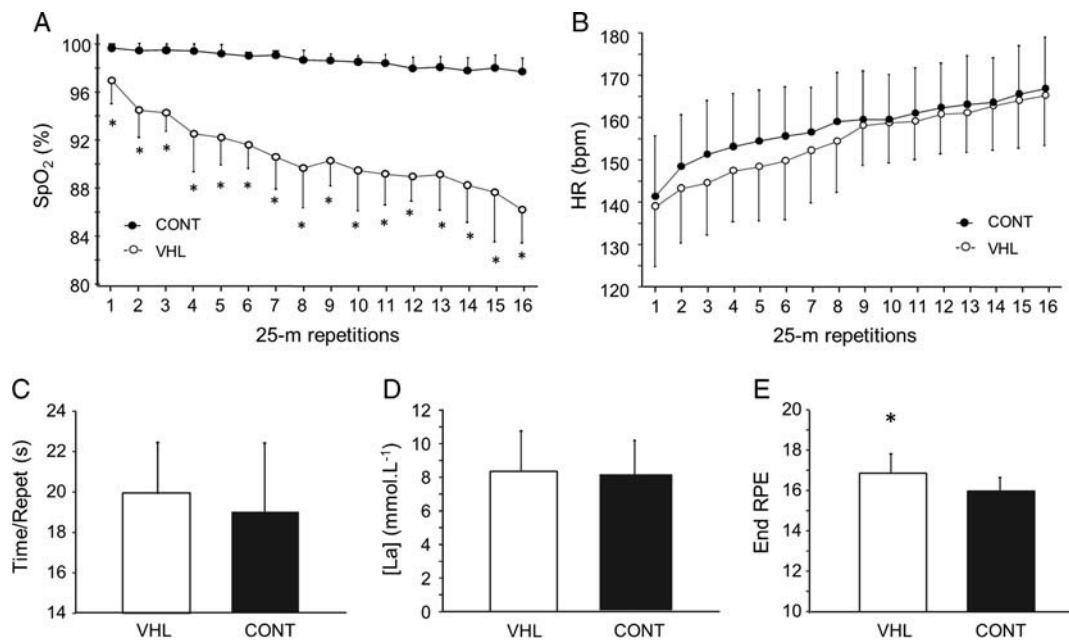
**Evaluation of training stimulus.** The participants were asked to report their daily physical training into a detailed logbook during the entire training period. The logbook included duration, distance, and RPE of each training session. For each subject of both groups, we quantified the total training stimulus using the method developed by Foster et al. (12), which consists of multiplying the RPE of the global training session by its duration. During the 5-wk

period, we also assessed the average time and the end-exercise RPE of each supramaximal set in all subjects. Furthermore, at the third and fourth week of the training period, we measured SpO<sub>2</sub>, HR, [La], and RPE during one set of 16 × 25 m in each subject. Thus, it was possible to compare the stimulus and the physiological effects of the supramaximal set between CONT and VHL.

**Statistical analyses.** Data were first tested for distribution normality and variance homogeneity. The effect of treatment (VHL vs CONT) and time (Pre vs Post) was then assessed for each of the variables by using a two-way repeated-measures ANOVA. When a significant effect was found, the Newman-Keuls *post hoc* procedure was performed to localize the difference. We used Student's *t*-tests for determining whether there was a difference between groups in the change in performance (Δperf) and in the change in [La]<sub>max</sub> (Δ[La]<sub>max</sub>) and [La]<sup>Rt</sup> (Δ[La]<sup>Rt</sup>). The Pearson linear regression analysis was performed to find any potential linear relationship between Δperf on the one hand and Δ[La]<sub>max</sub> and Δ[La]<sup>Rt</sup> on the other hand. Repeated-measures ANOVA and Student's *t*-tests were also used to compare the variables measured during training in both groups. All statistical analyses were performed with Statistica software (StatSoft Inc., Tulsa, OK), and the level of significance was chosen for *P* < 0.05. Values are presented as mean ± SD.

## RESULTS

Ten subjects completed the entire 10 supramaximal sets. In VHL, two subjects missed one session and one subject missed two sessions. In CONT, three subjects missed one



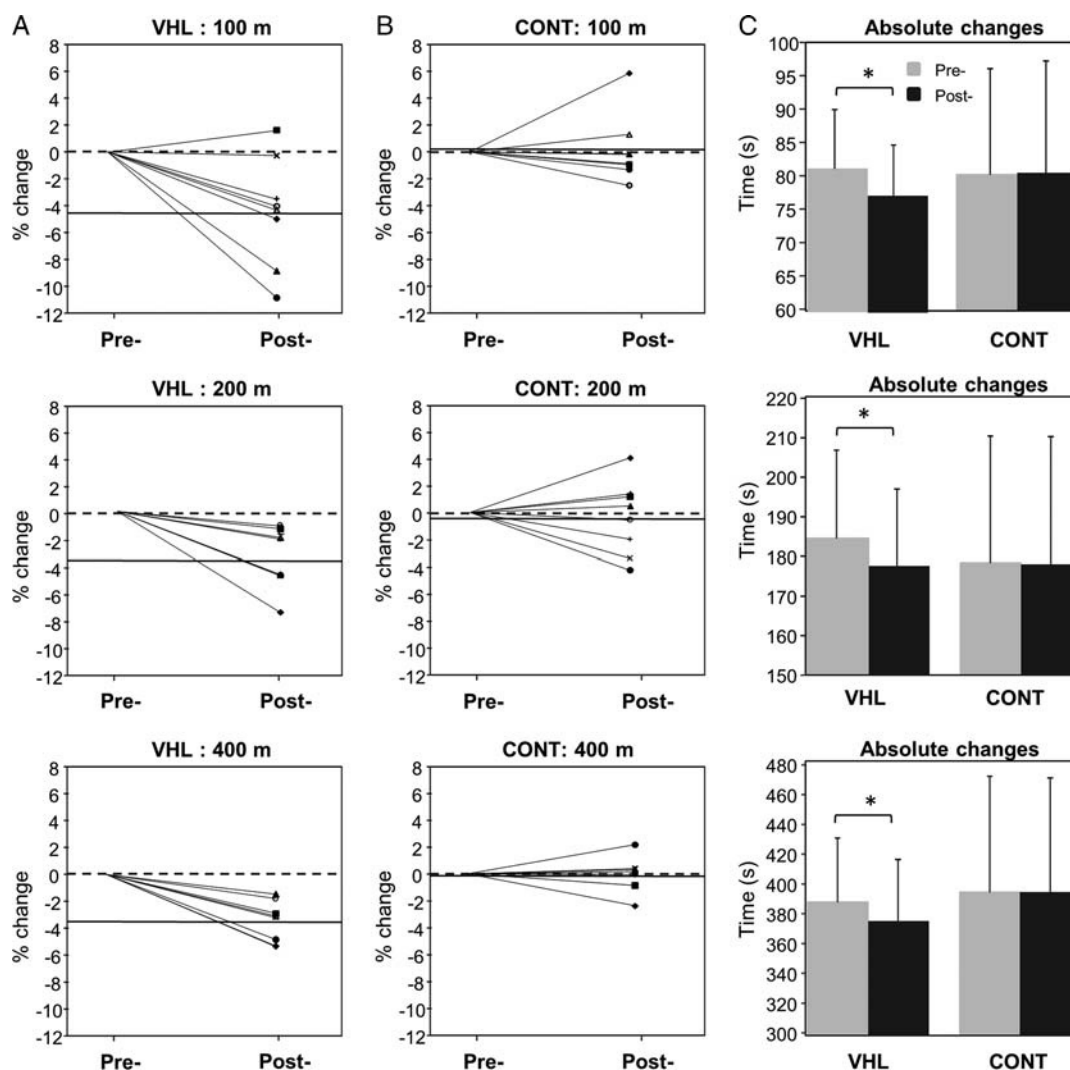
**FIGURE 2**—Arterial oxygen saturation (SpO<sub>2</sub>) (A), heart rate (HR) (B), mean time per repetition (C), blood lactate concentration ([La]) (D), and RPE (E) measured during or at the end of one set of 16 × 25 m performed at supramaximal intensity with hypoventilation (VHL) or normal breathing (CONT). \*Significantly different from CONT, *P* < 0.05.

session. Furthermore, one subject of CONT could not complete the 400-m trial at the first testing session because of his inability to breathe through the snorkel at maximal intensity. The results of CONT for this trial are presented for seven subjects.

**Training data.** For the entire supramaximal sets, there was no difference between VHL and CONT in the mean number of repetitions per set ( $14.9 \pm 0.8$  vs  $15.3 \pm 0.3$ ), time per repetition ( $20.1 \pm 2.3$  vs  $19.7 \pm 3.2$  s), and recovery between repetitions ( $12.9 \pm 0.4$  vs  $12.1 \pm 1.5$  s). On the other hand, the mean end-set RPE was significantly higher in VHL than that in CONT ( $16.4 \pm 0.9$  vs  $14.7 \pm 1.2$ ,  $P < 0.05$ ). The measurements performed during one set of  $16 \times 25$  m showed that  $SpO_2$  was lower in VHL than that in CONT throughout the set, whereas HR and mean time per repetition were not different between groups (Fig. 2A–C). The mean time spent at different degrees of hypoxemia (4) during the set was always different between groups (VHL vs CONT):

mild hypoxemia ( $SpO_2 = [92\%–94\%]$ ),  $117.7 \pm 23.1$  s ( $23.5\% \pm 3.9\%$  of the set) vs  $0.75 \pm 2.1$  s ( $0.16\% \pm 0.4\%$ ); moderate hypoxemia ( $SpO_2 = [88\%–91\%]$ ),  $88.7 \pm 39.3$  s ( $17.8\% \pm 8.1\%$ ) vs 0 s (0%); severe hypoxemia ( $SpO_2 < 88\%$ ),  $55.0 \pm 53.4$  s ( $10.6\% \pm 10.4\%$ ) vs 0 s (0%). At the end of the set, there was no difference in [La] between both groups, whereas RPE was higher in VHL than that in CONT (Fig. 2D and E). The total training stimulus (expressed in arbitrary unit [AU]) during the entire period was not different between VHL ( $24,451 \pm 10,562$  AU) and CONT ( $27,680 \pm 13,556$  AU).

**Performance.** The results of the ANOVA showed a time–interaction effect in all trials. Performance was always improved at Post compared with Pre in VHL, although it did not change in CONT (Fig. 3). Furthermore,  $\Delta perf$  was always greater in VHL than that in CONT in the three trials (100 m:  $-3.7 \pm 3.7$  s [ $-4.4\% \pm 4.0\%$ ] vs  $+0.24 \pm 2.5$  s [ $+0.14\% \pm 2.7\%$ ]; 200 m:  $-6.9 \pm 5.0$  s [ $-3.6\% \pm 2.3\%$ ] vs



**FIGURE 3**—Percent change in 100-m (top), 200-m (middle), and 400-m (bottom) time trial performances for each individual subject of the hypoventilation (VHL) (A) and control group (CONT) (B) and absolute changes for all subjects together (C), before (Pre) and after (Post) the training period. Horizontal solid lines represent group changes. Horizontal dashed lines are the zero level. \*Significant differences between Pre and Post,  $P < 0.05$  (repeated-measures ANOVA).

$-0.69 \pm 5.7$  s [ $-0.4\% \pm 2.9\%$ ]; 400 m:  $-13.6 \pm 6.1$  s [ $-3.5\% \pm 1.5\%$ ] vs  $-0.27 \pm 6.5$  s [ $-0.04\% \pm 1.4\%$ ]).

**Gas exchange.** At  $\dot{V}O_{2peak}$ , there was no difference in any of the gas exchange variables either between groups at Pre and Post or between Pre and Post within each group (Table 1).

**SpO<sub>2</sub> and HR.** The results showed no difference in both SpO<sub>2</sub> and HR between groups or between Pre and Post during the last 15 s of each of the three trials (Table 2).

**Blood lactate concentration and rate of lactate accumulation in blood.** There was a time–interaction effect for all trials. In VHL,  $[La]_{max}$  and  $[La]^{Rt}$  were higher at Post than at Pre in the 100-, 200-, and 400-m trials, whereas there was no difference in CONT (Fig. 4). At Pre,  $[La]_{max}$  and  $[La]^{Rt}$  were not different between VHL and CONT in the three trials, whereas they were significantly higher at Post in VHL for the 100- and 400-m trials. In the 200-m trial,  $[La]_{max}$  tended to be greater at Post in VHL than that in CONT ( $P = 0.06$ ). Further,  $\Delta[La]_{max}$  was greater in VHL than that in CONT for the 100-m ( $+2.65 \pm 2.2$  mmol·L<sup>-1</sup> [ $+41.5\% \pm 45\%$ ] vs  $+0.04 \pm 1.1$  mmol·L<sup>-1</sup> [ $+1.7\% \pm 14.3\%$ ]), 200-m ( $+1.79 \pm 1.3$  mmol·L<sup>-1</sup> [ $+24.6\% \pm 22.7\%$ ] vs  $+0.4 \pm 1.1$  mmol·L<sup>-1</sup> [ $+4.6\% \pm 13.3\%$ ]), and 400-m ( $+2.61 \pm 1.7$  mmol·L<sup>-1</sup> [ $+42.2\% \pm 43.8\%$ ] vs  $-0.2 \pm 0.6$  mmol·L<sup>-1</sup> [ $-2.41\% \pm 7.9\%$ ]) trials.  $\Delta[La]^{Rt}$  was also greater in VHL than that in CONT in all trials (Fig. 4). Finally, we found a significant relationship in VHL between  $\Delta perf$  and  $\Delta[La]_{max}$  ( $R = -0.75$ ,  $P < 0.05$ ) and between

TABLE 2. SpO<sub>2</sub>, HR, B<sub>f</sub>, SL, and SR values for the 100- and 200-m trials.

	100 m		200 m	
	Pre	Post	Pre	Post
SpO <sub>2</sub> (%)				
VHL	98.1 ± 2.0	98.3 ± 1.7	97.0 ± 2.0	96.7 ± 2.2
CONT	98.7 ± 1.0	98.9 ± 1.0	98.5 ± 0.9	98.2 ± 0.9
HR (bpm)				
VHL	165.1 ± 12.8	164.2 ± 12.4	165.5 ± 13.6	165.7 ± 11.8
CONT	163.0 ± 9.60	162.7 ± 11.5	163.7 ± 11.2	162.9 ± 12.0
B <sub>f</sub> (cycles per minute)				
VHL	27.8 ± 6.6	26.2 ± 7.5	26.6 ± 6.6	26.7 ± 6.4
CONT	27.9 ± 3.6	28.3 ± 3.7	27.9 ± 2.4	27.8 ± 3.0
SL (m)				
VHL	1.07 ± 0.1	1.06 ± 0.1	1.03 ± 0.1	1.03 ± 0.1
CONT	1.13 ± 0.2	1.14 ± 0.2	1.08 ± 0.2	1.08 ± 0.2
SR (s·min <sup>-1</sup> )				
VHL	74.1 ± 7.7	78.0 ± 6.4*†	67.9 ± 5.4	70.9 ± 5.4*†
CONT	71.1 ± 8.9	70.4 ± 9.2	67.1 ± 7.2	67.2 ± 7.7

Values are presented as mean ± SD

Pre, before the training period; Post, after the training period; VHL, hypoventilation group ( $n = 8$ ); B<sub>f</sub>, breathing frequency; CONT, control group ( $n = 8$ ); HR, heart rate; SL, stroke length; SpO<sub>2</sub>, arterial oxygen saturation; SR, stroke rate.

\*Significantly different from Pre.

†Significantly different from CONT,  $P < 0.05$ .

$\Delta perf$  and  $\Delta[La]^{Rt}$  ( $R = -0.79$ ,  $P < 0.05$ ) for the 400-m trial but not for the two other trials. There was no such relationship in CONT.

**Stroke length, SR, and breathing frequency.** The results are presented in Table 2. In both groups, there was no difference in stroke length and B<sub>f</sub> at Post compared with Pre in the 100- and 200-m trials. There was also no difference in stroke length and B<sub>f</sub> between VHL and CONT at both Pre and Post in the two trials. On the other hand, SR was higher at Post than at Pre in VHL but not in CONT and higher in VHL than that in CONT at Post.

## DISCUSSION

This study was the first to investigate the physiological consequences of VHL training at supramaximal intensity in swimming as well as its effects on performance. The main result was that after 5 wk of such training, swimming performance was significantly improved for distances of 100, 200, and 400 m. On the other hand, the same training performed under normal breathing conditions did not alter performance in already well-trained triathletes. The increased  $[La]_{max}$  and  $[La]^{Rt}$  in the VHL group represents another original finding. It suggests that the performance improvement could be attributed, at least in part, to a greater activity of the anaerobic glycolysis.

So far, the studies that investigated the effects of hypoventilation training had not convincingly demonstrated that this method could be more effective for improving performance than training under normal breathing conditions. After 4 wk of VHL training in runners, Woorons et al. (39) reported only a tendency to an increase in maximal velocity reached during an incremental test ( $+2.4\%$ ). Furthermore, several weeks of training with hypoventilation at high lung volume did not improve swimming performance more than

TABLE 1. Gas exchange at  $\dot{V}O_{2peak}$ .

	Pre	Post
V <sub>T</sub> (L)		
VHL	2.43 ± 0.5	2.42 ± 0.5
CONT	2.46 ± 0.6	2.57 ± 0.6
B <sub>f</sub> (cycles per minute)		
VHL	46.1 ± 7.9	46.9 ± 8.9
CONT	43.8 ± 10.8	42.1 ± 6.6
$\dot{V}E$ (L·min <sup>-1</sup> )		
VHL	109.1 ± 20.6	110.5 ± 19.8
CONT	106.6 ± 31.7	108.2 ± 30.7
$\dot{V}O_{2peak}$ (L·min <sup>-1</sup> )		
VHL	3.40 ± 0.9	3.55 ± 0.8
CONT	3.39 ± 1.0	3.50 ± 1.0
$\dot{V}O_{2peak}$ (mL·min <sup>-1</sup> ·kg <sup>-1</sup> )		
VHL	50.6 ± 11.2	52.9 ± 9.3
CONT	50.1 ± 11.3	51.1 ± 9.7
$\dot{V}CO_2$ (L·min <sup>-1</sup> )		
VHL	3.23 ± 1.0	3.22 ± 0.9
CONT	3.41 ± 1.2	3.45 ± 1.2
$\dot{V}E/\dot{V}O_2$ (L·min <sup>-1</sup> )		
VHL	32.1 ± 5.9	30.7 ± 3.0
CONT	30.8 ± 4.0	30.6 ± 4.2
PETCO <sub>2</sub> (mm Hg)		
VHL	36.6 ± 5.7	36.2 ± 4.7
CONT	38.7 ± 3.9	38.8 ± 5.3
PETO <sub>2</sub> (mm Hg)		
VHL	113.0 ± 4.2	112.4 ± 5.3
CONT	111.4 ± 5.0	111.3 ± 5.0

Values are presented as mean ± SD.

Pre, before the training period; Post, after the training period; V<sub>T</sub>, tidal volume; B<sub>f</sub>, breathing frequency;  $\dot{V}E$ , expired ventilation;  $\dot{V}O_{2peak}$ , highest value of oxygen consumption recorded for 30 s;  $\dot{V}CO_2$ , carbon dioxide production;  $\dot{V}E/\dot{V}O_2$ , ventilatory equivalent; PETCO<sub>2</sub>, end-tidal carbon dioxide pressure; PETO<sub>2</sub>, end-tidal O<sub>2</sub> pressure; VHL, hypoventilation group ( $n = 8$ ); CONT, control group ( $n = 7$ ).

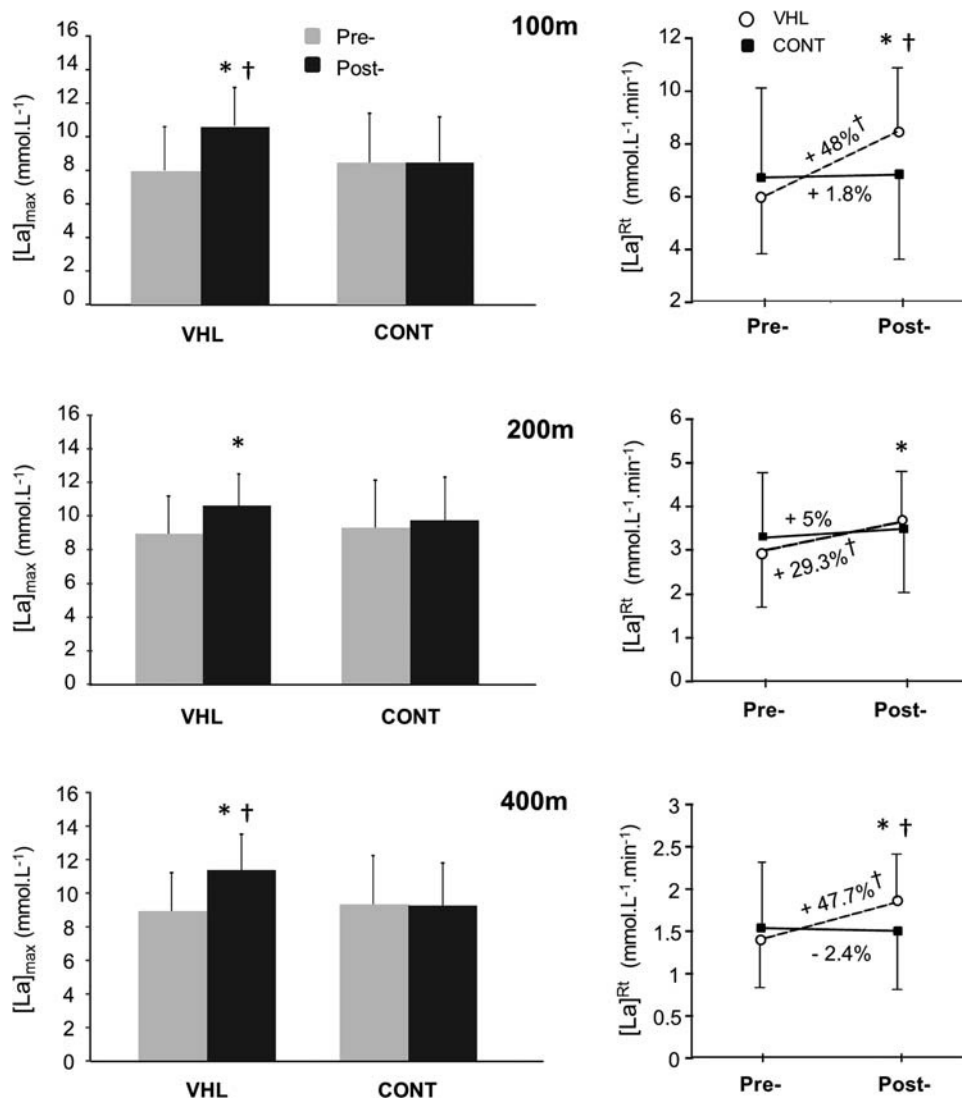


FIGURE 4—Maximal blood lactate concentration ( $[La]_{max}$ ) and rate of lactate accumulation in blood ( $[La]^{Rt}$ ) in the 100-, 200-, and 400-m trials Pre and Post training at supramaximal intensity with hypoventilation (VHL) or normal breathing (CONT). \*Significant differences between Pre and Post. †Significantly different from CONT,  $P < 0.05$  (repeated-measures ANOVA).

training with normal breathing (17,21) and had no effect on cycling performance (18). However, it is remarkable that in all these studies, hypoventilation training was performed at intensities that did not exceed  $\dot{V}O_{2max}$ . This may explain the lack of significant improvement in performance or the fact that the increase was not greater than training with normal breathing. A thorough review of the studies that dealt with IHT, to which VHL training can be related, showed that this approach was quite ineffective for sea-level performance when using low to moderate exercise intensities (10). In such conditions, the power output does not induce a sufficient stimulus for the active musculature, which probably leads to a downregulation of muscle structure and function (22). On the other hand, when hypoxia is associated with high-intensity exercises, the performance improvement seems to be greater than when training under normoxic conditions (8,11,13,31). In the present study, it is likely that the use of supramaximal intensity during VHL training

played a key role in the increase in swimming performance, especially because distances of 100 and 200 m are mainly glycolytic and require high swimming speeds. Therefore, on the basis of all the current knowledge, we suggest that for an effective VHL training, athletes should predominantly use exercise intensities at least as high as the intensity of their targeted competitive time.

During the 5-wk period of training, all swimming sessions were rigorously supervised. Quantitative data were collected at each session, and physiological measurements were performed during one supramaximal set in all subjects. Thus, it was possible to establish that, on average, both groups performed the entire sets at the same absolute exercise intensity. The mean number of repetitions per set and the recovery duration after each repetition were not different between groups. Furthermore, the data reported by the participants about their daily physical training showed no difference between VHL and CONT concerning the total

training stimulus during the entire period. Therefore, it may be concluded that the additional stimulus of VHL training associated with the supramaximal intensities of exercise was mainly responsible for the outcome of the present study.

The increases in  $[La]_{max}$  and  $[La]^{Rt}$  that were recorded in all swimming trials after VHL training represent an interesting result. Such findings had never been reported so far by studies dealing with hypoventilation training. However, it was already shown that after several weeks of training performed exclusively at moderate intensity, peak  $[La]$  could be maintained in subjects who trained with VHL, but it decreased when training was performed under normal breathing conditions (39). The authors concluded that VHL training could have positive effects on the anaerobic glycolysis. The results of the present study seem to demonstrate that when VHL training is performed at supramaximal intensity, the energy produced by the anaerobic glycolytic system is largely augmented.

This phenomenon was probably the consequence of the combined effect of hypoxia and hypercapnia. During exercise with VHL, the drop in  $O_2$  partial pressures associated with the raise in blood  $CO_2$  partial pressure ( $PCO_2$ ) provokes a large arterial  $O_2$  desaturation and, consequently, a fall in tissue oxygenation (36). In these conditions,  $[La]$  and therefore the anaerobic stimulus have been reported to be higher than during exercise with normal breathing (36,37). In the present experiment, data collected during VHL training confirmed the appearance of a severe hypoxemia ( $SpO_2 < 88\%$ ) during the 25-m repetitions. Besides, it is remarkable that the levels of  $SpO_2$  were even lower than those in previous studies (35,37,38), likely because of the high exercise intensities. Thus, it is likely that repeated bouts of tissue hypoxia undergone during the supramaximal sets with VHL induced adaptations leading to higher  $[La]^{Rt}$  and  $[La]_{max}$ . The higher  $[La]^{Rt}$  in particular is very reflective of the greater contribution of the anaerobic glycolysis (6). It may have been the consequence of an increased activity of the glycolytic enzymes, such as lactate dehydrogenase or phosphofructokinase, as reported after supramaximal-intensity IHT or RSH training (11,31). On the other hand, the increased  $[La]_{max}$  reflects an improved anaerobic capacity and may be due to a greater ability to tolerate high concentrations of lactate and high levels of acidosis, as reported after high-intensity training (20,27).

It is very likely that the greater anaerobic glycolysis induced by VHL training played a role in the increased performance. Previously, a higher maximal  $[La]$  close to the one observed in the present study ( $+2.2 \text{ mmol}\cdot\text{L}^{-1}$  vs  $+2.4 \text{ mmol}\cdot\text{L}^{-1}$ ) was associated with a higher peak power output ( $+12\%$ ) in elite road cyclists after 4 wk of training, including supramaximal-intensity exercises in hypoxia (31). This assumption is also supported by the significant relationships we found between both  $\Delta[La]^{Rt}$  and  $\Delta[La]_{max}$  and  $\Delta perf$  for the 400-m trial in the VHL group. However, it is surprising that such relationship did not exist for the 100- and 200-m trials considering their glycolytic feature.

Likewise, the fact that  $[La]_{max}$  was not higher in these trials than that in the 400 m is also surprising. These unexpected results are difficult to explain. It could be possible that our data did not reflect the actual  $[La]_{max}$  because the highest  $[La]$  can be reached between the fifth and the eighth minute of the recovery period in short competitive swimming events (2,7). Anyway, it is probable that other factors, in particular, better regulation of muscle acid-base balance, contributed to the performance improvement after VHL training.

Although metabolic acidosis was greater in the VHL group after the training period, no change occurred in any of the ventilatory parameters. Yet for compensating the greater acidosis, one may have expected an increase in both maximal  $\dot{V}E$  and  $\dot{V}E/\dot{V}O_2$  and a concomitant decrease in  $PETCO_2$ . This may be because in swimming, unlike terrestrial sports, the work of the respiratory muscles is greater because of hydrostatic pressures (28). The levels of maximal  $\dot{V}E$  can therefore be difficult to increase and may constitute a limiting factor in this sport. This argument is strengthened by the fact that for collecting gas exchange, the subjects had to breathe through a snorkel, which probably increases the work of breathing even more.

The role of an increased anaerobic glycolysis in the performance improvement after VHL training is reinforced by several factors. First,  $\dot{V}O_{2peak}$  was unchanged in VHL at Post compared with Pre. Although the maximal oxygen consumption of our subjects was not as high as in elite swimmers, one can reasonably assert that it was significantly enhanced by the high-intensity training performed within the few weeks preceding the experiment. Therefore, one can rule out an additional aerobic benefit induced by VHL training. Second, a decreased  $B_f$  associated with a greater  $PCO_2$  has already been reported in swimmers who trained several weeks with hypoventilation at high lung volume (17), possibly because of a lower sensitivity to hypercapnia (18). In swimming, the ability to reduce  $B_f$  could be interesting for the performance because turning the head to inhale increases drag and, consequently, the energy cost because of hydrodynamic disturbances and discontinuity in propulsive actions (23). The unaltered  $B_f$  in all swimming trials in the VHL group also rules out a contribution of hydrodynamic factors in the performance gain. Finally, it is noteworthy that stroke length, which depends on technical skill and is described as an index of motor effectiveness (3,30), was not different between Pre and Post in VHL. Thus, performance could be improved through an increased in SR, which was probably the result of anaerobic adaptations within the muscles fibers.

The fact that the experiment was not double or even single-blinded constitutes the main limitation of the present study. Because hypoventilation is a voluntary act, it is obviously not possible to blind the subjects or the investigators in this kind of research. Therefore, one cannot exclude that a placebo effect played a role in the performance improvement in VHL. In particular, the magnitude of this effect on performance has been shown to be between 1% and 5% in



majority (1). It is for instance possible to argue that the increased  $[La]_{max}$  was caused by psychological factors that may have led the subjects to push physiological limits. However, it is important to mention that we kept a neutral attitude toward all the participants of both groups throughout the experiment. Further, we did not present VHL training as an effective method to improve fitness level.

In conclusion, this study demonstrated that VHL training, when performed at supramaximal intensities, could represent an effective training method in swimming. The performance improvements that occurred for the 100-, 200-, and 400-m trials were probably the consequence of physiological adaptations induced by the strong combined and

intermittent effect of hypoxia and hypercapnia. Our results suggest that the performance gain after VHL training was partly due to an improved anaerobic glycolytic activity. However, the beneficial effects of this training method remain to be confirmed in elite or high-level swimmers.

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The authors declare that the results of the present study do not constitute endorsement by the American College of Sports Medicine.

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