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Repeated sprint training in hypoxia induced by voluntary hypoventilation in swimming

Submission type: Original Investigation

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ABSTRACT

Purpose: Repeated-sprint training in hypoxia (RSH) has been shown as an efficient method for improving repeated sprint ability (RSA) in team-sport players but has not been investigated in swimming. We assessed whether RSH with arterial desaturation induced by voluntary hypoventilation at low lung volume (VHL) could improve RSA to a greater extent than the same training performed under normal breathing (NB) conditions. **Methods:** 16 competitive swimmers completed six sessions of repeated sprints (two sets of 16×15 m with 30 s send-off) either with VHL (RSH-VHL, n=8) or with NB (RSN, n=8). Before (pre-) and after (post-) training, performance was evaluated through an RSA test (25m all-out sprints with 35 s send-off) until exhaustion. **Results:** From pre- to post-, the number of sprints was significantly increased in RSH-VHL (7.1 ± 2.1 vs 9.6 ± 2.5; $p<0.01$) but not in RSN (8.0 ± 3.1 vs 8.7 ± 3.7; $p=0.38$). Maximal blood lactate concentration ($[La]_{max}$) was higher at post- compared to pre- in RSH-VHL (11.5 ± 3.9 vs 7.9 ± 3.7 $mmol.l^{-1}$; $p=0.04$) but was unchanged in RSN (10.2 ± 2.0 vs 9.0 ± 3.5 $mmol.l^{-1}$; $p=0.34$). There was a strong correlation between the increases in the number of sprints and in $[La]_{max}$ in RSH-VHL only ($R=0.93$; $p<0.01$). **Conclusion:** Repeated sprint training in hypoxia induced by voluntary hypoventilation at low lung volume improved repeated sprint ability in swimming, probably through enhanced anaerobic glycolysis. This innovative method allows inducing benefits normally associated with hypoxia during swim training in normoxia.

Key words: RSH, swim, low pulmonary volume, saturation

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INTRODUCTION

Over the last few years, a novel approach of hypoxic training has been investigated by several studies.[1-3] This method, the so-called repeated-sprint training in hypoxia (RSH), consists of the repetition of short 'all-out' exercise bouts (<30 s) interspersed with incomplete recoveries under hypoxic conditions. Even though experimental confirmation is needed, RSH efficiency has been suggested to rely on the increased muscle perfusion induced by the vasodilatory compensation associated to the hypoxia-induced reduced oxygen content. It is now considered a different method than hypoxic training (IHT).[4,5] RSH also differs from IHT in the sense that the training intensity is maximal and therefore enables to maintain high fast-twitch fibres (FT) recruitment. Thus, although it has recently been reported that the RSH model may not improve sea-level performance more than the same training performed in normoxia,[6,7] it could anyway lead to a greater improvement when carried out under certain conditions.[1,2] In particular, this method has been shown to be effective for improving the repeated-sprint ability (RSA). The number of sprints completed during an RSA test to exhaustion was greater in cycling[1] (38%) and in double poling skiing[2] (58%) after as few as six[2] or eight[1] training sessions of RSH. On the other hand, no improvement in RSA was found in rugby players after RSH.[3]Ref_1Ref_2ref_3ref_4ref_1ref_4ref_2

One of the main difficulties inherent to hypoxic training is the access to normobaric hypoxic facilities or devices, either for practical and financial reasons or due to the characteristics of the sport. Some equipment[8] allow training in running, cycling or even in team-sports while in swimming, exercising in normobaric hypoxia is quite problematic. Within the last ten years, several studies have reported that it was possible, thanks to a technique of voluntary hypoventilation at low lung volume (VHL), to train under hypoxic conditions without leaving sea-level or using devices that mimic altitude conditions.[9-14] During biking or running exercises carried out with VHL, it has been shown that pulse

oxygen saturation (SpO_2) could drop under 88%,[11,13] a level considered as severe hypoxaemia[15] and equivalent to an altitude above 2000-m.[9] Furthermore, very recently, a study demonstrated that even in swimming, using VHL, or the so called "exhale-hold" technique", could lead to a strong arterial desaturation and therefore could enable every swimmer to train under hypoxic conditions[14].ref_5ref_6ref_7ref_8ref_9ref_10

Implementing RSH through the VHL technique (RSH-VHL) in order to improve RSA could be interesting for some aquatics sports. In swimming for instance, performance has been shown to be closely related to the capacity to maintain high exercise intensities during training.[16] Moreover, in water polo, like in all terrestrial team sports, the ability to repeat intense exercise bouts for sustained periods is essential for overall performance.[17] However, it is important to note that when exercising with VHL, the time spent at large arterial desaturation is shorter than during the same exercise performed under real hypoxic conditions.[18] This is due to the fact that periods with normal breathing, which make SpO_2 raise up again, must be interspersed with the hypoventilation periods. Consequently, during VHL exercise, the "hypoxic dose" is lower than when exercising in an environment impoverished in O_2. However, in previous RSH studies, the overall hypoxic dose was also very low. It is therefore unclear how the intermittent desaturation pattern occurring with VHL would have an impact on the extent of the physiological adaptations linked to the oxidative pathway after RSH-VHL. On the other hand, it is also noteworthy that VHL exercise elevates the partial pressures of carbon dioxide (CO_2) within the body which in turn increases blood bicarbonate concentrations.[10,12] This may have consequences on buffering capacity and could be beneficial for pH regulation and the anaerobic metabolism capacity, as previously reported.[11] Such adaptations could be interesting for RSA since the limitation of the energy available from the anaerobic glycolysis and the intramuscular accumulation of hydrogen ions,

as well as the increase in extracellular potassium, are amongst the key factors responsible for fatigue during repeated-sprint exercise.[19]

So far, no study has ever investigated the effects of RSH in swimming. Also, VHL training has never been performed at maximal velocity. The goal of the present study was therefore to determine whether six sessions of RSH-VHL carried out in swimming could have positive effects on repeated-sprint (RS) performance. We hypothesized that such training would increase RSA to a greater extent than the same training carried out under normal breathing conditions.

METHODS

Subjects

Sixteen highly-trained swimmers (9 men, 7 women) were selected to participate in this study. Their main characteristics are presented in Table 1. Eleven of the swimmers had a regional level whereas the other five competed at a national level. At the time of the experiment, all swimmers had at least 9 hours per week of training in the pool plus 3 hours of strength and conditioning training. The subjects were all non-smokers, lowlanders and not acclimatized or recently exposed to altitude. None of them had ever used VHL training before the study. During the protocol, the subjects were asked to avoid any exposure to an altitude of more than 1500-m. Prior to the experiment, all swimmers carried out a medical test and none of them had any sign of respiratory, pulmonary or cardiovascular disease. Written voluntary informed consent was obtained from the subjects (or their parents it they were minors) before participation and the study was approved by the institutional ethics committee.

Study design

The experimental protocol consisted in performing one testing session before (pre-) and after (post-) a specific RS training period of two weeks (six specific sessions). The subjects were matched into pairs for gender and performance level and then randomly assigned to the group with RS training in normoxia (RSN, n=8) or in hypoxia induced by VHL (RSH-VHL, n=8) (Table 1). Before the experiment, a lead-in period was carried out over four sessions in order to standardize the swimmers' training and familiarize them with VHL technique as well as the equipment used for the measurements. All testing and training sessions were conducted in the same 25-m swimming pool (altitude: 417-m; water temperature: 27°C).

Training protocol

Within six specific swimming sessions, and over a 2-week period, swimmers had to complete, after a 1500-m standardized warm-up, two sets of 16×15 m all-out front crawl sprints, each 15-m repetition starting every 30 s. Both sets were separated by 20 min of active recovery at low intensity. The RSN group performed the two sets with normal breathing (NB) while the RSH-VHL group completed the sets with VHL. To perform the VHL technique, which was well described in a previous study,[14] swimmers were asked to exhale down to functional residual capacity or a little below just before starting each 15-m sprint. Then they had to push off the wall, glide and swim by trying to hold their breath up to the end of the 15 m. If they were not capable to do so, they were allowed to take an inhalation after exhaling the remaining air from the lungs and reproduce the same exhale-hold procedure to finish the sprint. At the end of each 15-m sprint, the swimmers of both groups completed the remaining 10 m at low pace while breathing normally and then recovered passively along the wall till the next sprint. The specific swimming sessions including the RS were separated by 48-72 h.

The other training sessions of the week were generally performed at low intensity, near the first ventilatory threshold.

Testing protocol

Two days before and two days after the two-week training period, swimmers of both groups participated in one testing session including an RSA test. For the 48 h prior to each testing session, subjects refrained from high-intensity training and exhaustive activity and were requested to sleep at least 8 h the night before. Furthermore, they were asked to maintain their usual diet during the intervention period, to avoid caffeine and alcohol in the 24 h preceding the measurements and to arrive at the testing sessions in a rested and hydrated state, at least 3 h postprandial. Each subject performed the two testing sessions at the same time and day of the week. After the same 1500-m standardized warm-up as during the RS training sessions, they performed a single 25-m all-out sprint followed, after 3 min of recovery at low intensity, by a second 25-m sprint. Reference velocity (RV) was calculated from the best time recorded over the two sprints. The RSA test started after a 10-min swim period at low intensity. It consisted of the repetition of 25-m all-out front crawl sprints with NB. Each 25-m repetition started every 35 s so that the (passive) recovery period was about 20 s. To avoid any protective pacing strategy, subjects were requested to reach at least 98% of the RV over the first sprint, which was the case in all of them. Then, over the following sprints, task failure was declared when swimmers did not reach, for the second time, at least 94% of RV. Subjects were given very strong verbal encouragement over the whole test to complete as many sprints as possible. They were never told about the criterion for task failure and were never given any indication on the number of sprints performed.

Measurements

Testing data

Performance

During the RSA test, time performance of each sprint was simultaneously measured by two experienced timekeepers. It is important to mention that in multiple experienced timers, the error in hand timing has been shown to be very low (e.g., mean error corresponding to -0.04 - 0.05 s) and intraclass correlation values between hand and electronic timing is high (~0.98-0.99).[20] The mean value of both measurements was recorded and sprint velocity (SV) was calculated *a posteriori*. Maximal SV (SV_{max}) and the total number of sprints test were assessed at pre- and post- in both groups.

Fatigue score

To assess fatigue during the RSA test, we used the percentage decrement score (S_{dec}) which has been shown to be the most valid and reliable measure for quantifying fatigue in this kind of test.[19] The following formula was used:

- S_{dec} (%) = (100×(total sprint time/ideal sprint time))-100
    - Where :
        - Total sprint time = sum of sprint times from all sprints.
        - Ideal sprint time = number of sprints×fastest sprint time.

Blood lactate concentration ([La]) and rating of perceived exertion (RPE)

Just after the end of the RSA test, we asked the swimmers to evaluate RPE using the Borg scale (0-10).[21] Then at the first and third minute following the test, blood sample (5 µl) was collected at the earlobe to measure [La] (Lactate Pro, Akray, Japan). The highest values of the two samples was recorded as the maximal [La] ($[La]_{max}$).

Training data

To assess and compare the physiological effects of the two RS modalities (i.e. with VHL vs NB), we performed several measurements during one set of 16×15 m all-out sprint in each subject. SpO_2 and heart rate (HR) were continuously measured via a pulse oximeter (Nellcor N-595, Pleasanton, CA, USA). We used the adhesive forehead sensor Max-Fast (Nellcor, Pleasanton, CA, USA) which was waterproofed to allow its utilization in the pool as validated and fully described previously.[14] Time performance of each sprint was also measured during the same set to obtain SV_{max} and S_{dec}. Finally, we assessed [La] and RPE at the end of the set.

Statistics

Data are presented as mean ± SD. The effect of treatment (RSH-VHL vs RSN) and time (pre- vs. post-) was assessed for each of the variables using a two-way ANOVA for repeated measures. When a significant effect was found, the Bonferroni post hoc procedure was performed to localize the difference. Pearson linear regression analysis was performed to find any potential linear relationship between the change in the number of sprints and the change in $[La]_{max}$ during the RSA test. ANOVA for repeated measures and Student *t*-tests were also used to compare the variables measured during the training sessions in both groups. All analyses were made with Sigmastat 3.5 software (Systat Software, CA, USA). Null hypothesis was rejected at $p<0.05$.

RESULTS

All subjects of both groups completed the six specific training sessions within the two-week period. The total amount of swim training performed between the two testing sessions was not different between RSN and RSH (number of training sessions: 11.4±0.5 vs

11.4±0.5; total duration: 18.5±1.7 h vs 18.6±1.8 h; total distance: 41.5±4.0 km vs 42.9±5.4 km).

Testing data

Performance and fatigue score

The results are presented in Table 2 and Figure 1. There was no difference in RV between groups both at pre- and post-. Likewise, during the RSA test, the total number of sprints, SV_{max} and S_{dec} were not different between RSN and RSH-VHL at the two testing sessions. The two-way Anova showed a significant time effect for the number of sprints ($p = 0.015$). The post-hoc test revealed a significant increase in RSH-VHL ($p < 0.01$) but not in RSN ($p = 0.38$). On the other hand, despite a significant time effect for SV_{max} ($p=0.04$), the post-hoc test showed no difference in this variable at post- compared to pre- in the two groups. Finally RV increased in both groups ($p=0.03$) after the training period whereas there was no change in S_{dec}.

Blood lactate concentration and RPE

Due to technical problems, [La] could not be measured in two subjects (one in each group). The two-way ANOVA showed no group effect for $[La]_{max}$ and RPE at the end of the RSA test. On the other hand, there was a time effect for $[La]_{max}$ which was higher at post- compared to pre- in RSH-VHL ($p=0.04$) and unchanged in RSN ($p=0.34$) (Figure 2). There was a significant correlation between the change in the number of sprints and the change in $[La]_{max}$ over the RSA test in RSH-VHL ($R=0.93$; $p<0.01$; $n=7$) but not in RSN ($R=0.53$; $p=0.23$; $n=7$). From pre- to post-, there was no difference in RPE in either group.

Training data

Of interest is the large decrease in SpO_2 observable from the start of the set for each sprint in RSH-VHL (Figure 3). There was no difference between both groups in any of the

variables measured during one set of 16×15 m except for S_{dec} (Table 3) which was lower in RSH-VHL than in RSN (p=0.03) and time at various level of SpO_2 (Table 4).

DISCUSSION

This study was the first to investigate the effects of a particular form of RSH in swimming, in which hypoxia was induced by hypoventilation. It was also the first to use VHL during repeated-sprint exercise. The main finding was that six sessions of RSH-VHL could significantly improve RSA performance in swimming (i.e. number of sprints) while the same training carried out in normoxia and under normal breathing conditions did not modify RSA. A second original result was that RSH-VHL, unlike RSN, led to a higher $[La]_{max}$ and therefore probably to a greater energy supply from the anaerobic glycolysis.

The 35% increase in the number of sprints after RSH-VHL in swimming is in line with the results reported very recently after RSH in cyclists (38%).[1] On the other hand, competitive cross country skiers were capable of performing 58% more sprints during an RSA test after only six sessions of RS under normobaric hypoxia.[2] The authors explained the greater efficiency of RSH in skiers than in cyclists by the fact that the upper arm muscles contain a high proportion of FT.[22,23] Yet the physiological adaptations induced by RSH that are determinant for performance have been shown to take place in FT.[5] In swimming, propulsion is generated mainly from the upper-body muscle groups[24] known for their high FT proportion.[25] However, the mean power related in front crawl[26] could be lower than in double poling.[2] This may partly explain why, in the present study, the increase in the number of sprints to exhaustion was less than in skiers[2] and closer to the results reported in cyclists.[1]

In addition, the work-to-rest ratio is known for being paramount for the effectiveness of RS, especially in hypoxia,[27] since it determines to a large extent[28] the oxidative vs glycolytic contribution and the type of muscle fibre that are recruited. In previous studies

where RSH was efficient,[1,2] the ratio was 1:2. In the present study, sprint and recovery durations were ~9 s and ~21 s respectively during training sessions and ~13 s and ~22 s during the RSA tests. Therefore the ratio was close to 1:2 and similar to the above-mentioned studies.[1,2] However, one cannot rule out that the benefits induced by RSH-VHL might have been different with another ratio. Since the RSH studies that used a 1:5 ratio did not report any RSA improvement,[3,6,7] it is doubtful that such low ratio could lead to a more effective RSH intervention. On the other hand, slightly increasing the recovery during RSH-VHL (ratio ~1:3) might have helped maintain a maximal intensity during the all set. It is indeed noticeable that swimmers who performed the set with VHL had a lower S_{dec}, and therefore a reduced fatigue than the RSN group. This was probably the consequence of a conservative pacing in order to finish the set that was perceived as challenging.

The data recorded during training demonstrate that it is possible to create an intermittent hypoxic condition when using VHL during a swimming RS session. On average, over the whole set, SpO_2 dropped below 84% in most of the sprints (Figure 3**)**. Such an arterial desaturation had never been reported so far by studies dealing with VHL exercise. The minimum level of SpO_2 was even slightly lower than what was reported in a recent RSH study.[2] However, it is important to note that swimmers reached their minimum saturation level for only a few seconds during each sprint. Furthermore, unlike in RSH (i.e. in normobaric hypoxia), the subjects were not in hypoxic conditions during the recovery periods since they had to breathe normally. Thus, over the whole set (including the periods of recovery with NB), the mean SpO_2 was 94.5%. Therefore, although high levels of desaturation were reached, the overall duration sustained at SpO2 < 88% was very low (Table 4) which might have impacted on the physiological adaptations.

In particular, it is questionable whether RSH-VHL could lead to a large improvement in muscle perfusion, as suggested after RSH.[1,2] This adaptation has been proposed as a

determinant factor of RSA on the indirect basis (near infrared spectroscopy; NIRS) of increased variation of muscle deoxygenation/reoxygenation during sprint/recovery. Indeed, an enhanced O_2 availability speeds up the rate of Pcr resynthesis,[29] which has been shown to be paramount in the maintenance of power production during RS.[30] Unfortunately, because there is no near infrared spectroscopy method suitable for aquatic movements, it was not possible to verify whether the hypoxic effect of RSH-VHL may have increased muscle oxygenation as much as after RSH. However, on the basis of recent findings, the higher the severity of the hypoxic stress may not be the better for improving RS performance as simulated altitudes of 4000m or above may negatively impact on the quality of training.[31,32] Anyway, according to the fact that the performance gain obtained after both approaches was rather similar, if the extent of the physiological adaptations aforementioned had been lower after RSH-VHL than after RSH, this should have been compensated by other factors.

Interestingly, a physiological change that occurred only after RSH-VHL and that has not been reported so far in any of the RSH studies is an increase in $[La]_{max}$. This finding is noteworthy because it suggests that using VHL instead of normobaric hypoxia to create hypoxic conditions could enhance the anaerobic glycolysis. Recently it was shown that high-intensity swimming exercise with VHL induced a higher [La] than the same exercise carried out with NB.[14] This phenomenon was probably the result of the fall in tissue oxygenation that occurs during this kind of exercise and that consequently leads to a greater reliance upon non-oxidative metabolism.[12] This is certainly the reason why repeatedly performing exercises with VHL has positive effects on the anaerobic glycolysis, as suggested before.[11,14] In the present study, based on the significant relationship between the change in $[La]_{max}$ and in the number of sprints, it is likely that enhanced maximal glycogenolytic and glycolytic rates played a role in the RSA improvement. This assumption is reinforced by a latest published study which has found that VHL training at supramaximal intensity could significantly

improve performance in swimming,[33] partly through an increased anaerobic glycolysis activity. However, in the context of repeated-sprint exercise, the contribution of anaerobic glycolysis has to be tempered since it is dependent upon exercise mode, muscle group/composition, sprint duration and work-to-rest-interval.[30]

One may argue that the outcome of this study was limited by the fact that swimmers were not blinded. It could for instance be assumed that the increase in both RS performance and $[La]_{max}$ were the result of a placebo effect instead of physiological modifications. This problem is recurrent when dealing with voluntary hypoventilation since the studies cannot be conducted single-or double-blind. However, while a psychological effect cannot be ruled out in the present study, it is important to note that swimmers were not aware or given any information about the effects of VHL training. Another limitation of the present study was that few physiological variables were measured, mainly because of the constraints of the aquatic environment and the maximal velocity of the swimmers. Consequently, it was difficult to fully determine the factors that led to an improvement in RS performance after RSH-VHL. Since VHL, unlike RSH, also leads to hypercapnia and elevated blood bicarbonate concentration,[10,12] it is possible that other physiological adaptations, such as better buffering capacity, have been involved in the increased performance. In particular, it would have been interesting to assess whether RSH-VHL could increase the capacity for pH regulation, a physiological adaptation that has already been reported after RSH[1] or VHL training[11] and that was shown to be an important factor of RSA.[34]

Practical applications

A greater capacity to repeat short bouts of exercise at maximal velocity could be valuable in most aquatics sports, such as swimming or water polo. In the former, training intensity has been shown to be the key factor for improving performance in elite swimmers[16]

and it is well established that RSA constitutes an important factor of performance in team sports.[17] Using VHL to implement an RSH could thus be useful also in these sports. However, few criteria seem to be useful for performing RSH-VHL. First, the effectiveness of RS, especially in hypoxia, seems to depend on a work-to-rest ratio close to 1:2. Second, it is probable that shorter bouts and sets would be more efficient and less psychologically demanding. Finally, an adequate dosage and combination with the other forms of training is required.[18]

In summary, this study showed for the first time that RSH-VHL could be an effective method for improving RSA in swimming. The increase in performance occurred after only six sessions over a two-week period. RSH-VHL also led to a greater anaerobic glycolysis activity, which seems to have played a role in the improved RSA. Further studies are needed to assess whether similar results could be obtained in non-aquatic sports and to determine the physiological modifications induced by this novel approach of hypoxic training.

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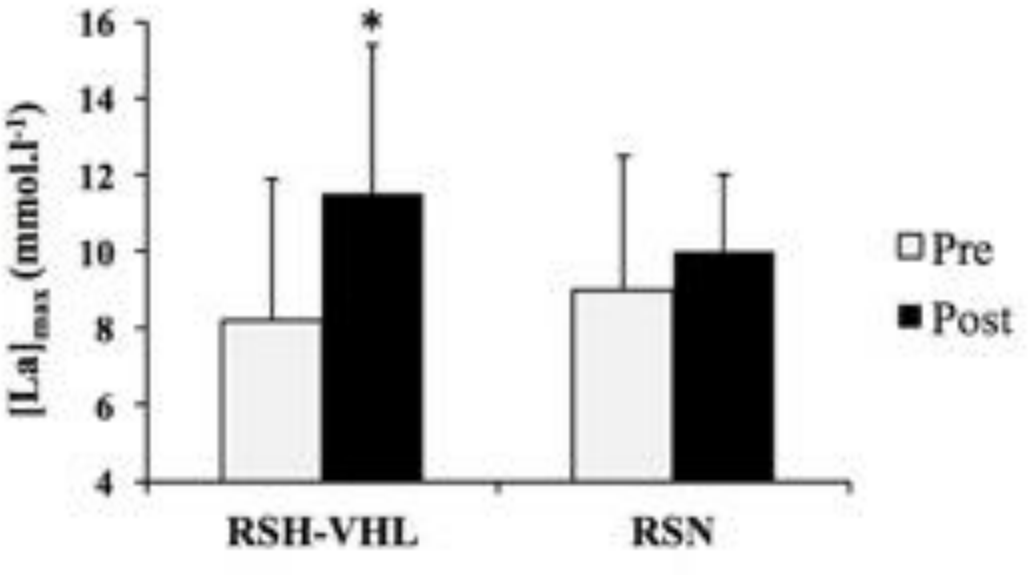

Figure 1: Average velocity in successive sprints during the repeated-sprint test before (pre-) and after (post-) repeated-sprint training in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) or in normoxia with normal breathing (RSN). ** $p < 0.01$ for difference with pre-; # $p < 0.05$ for difference with pre- sprint 7.

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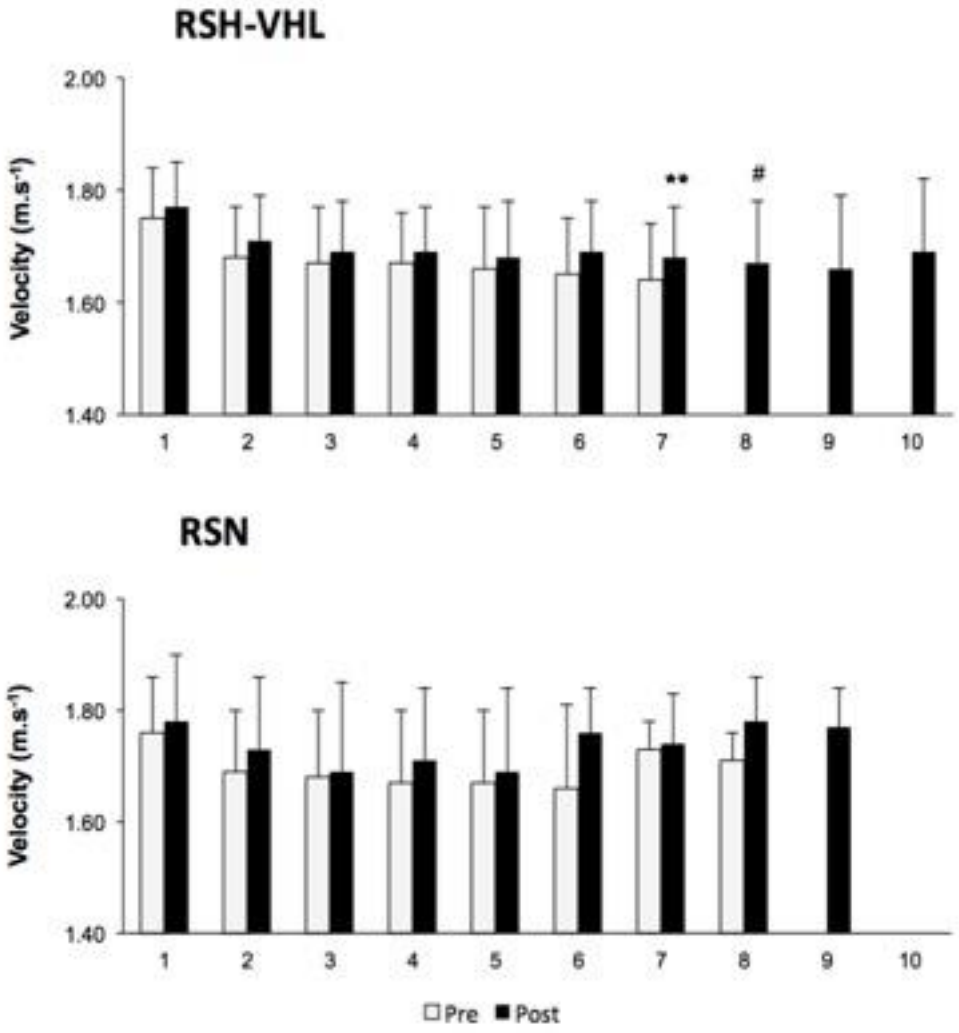

Figure 2: Maximal blood lactate concentration ($[La]_{max}$) measured after the repeated-sprint test before (pre-) and after (post-) repeated-sprint training in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) or in normoxia with normal breathing (RSN). * $p < 0.05$ for difference with pre-.

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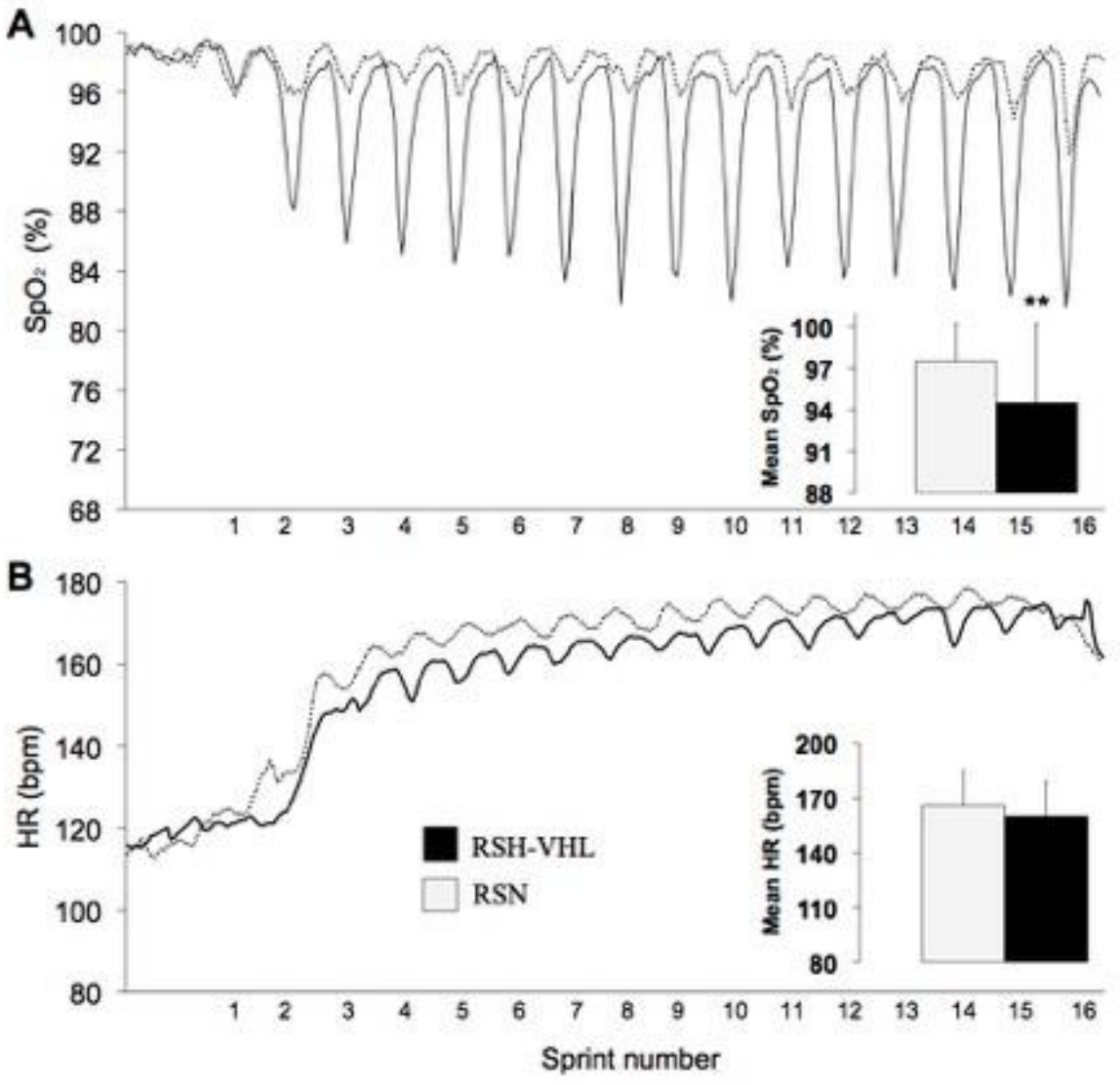

Figure 3: *Curves* : kinetics of arterial oxygen saturation (SpO_2) (A) and heart rate (HR) (B) during one set of 16 x 15-m swimming sprints performed in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL, straight line) or in normoxia with normal breathing (RSN, dotted line). *Bars* : mean SpO_2 (A) and HR (B) over the whole set in RSN and RSH-VHL. ** $p < 0.01$ for difference with RSN.

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Table 1 – Subjects characteristics

	RSH-VHL	RSN
Number of subjects	8	8
Age (year)	15.6 ± 1.8	15.5 ± 1.9
Gender	4 female / 4 male	3 female / 5 male
Height (cm)	168 ± 6	174 ± 11
Weight (kg)	55 ± 8	64 ± 10
Level (FINA points 2014)	481 ± 106	442 ± 86

Values are mean ± SD.

Table 2 – Data of the repeated-sprint test before (pre-) and after (post-) repeated sprint training in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) and in normoxia (RSN).

	RSH-VHL		RSN	
	pre-	post-	pre-	post-
RV ($m.s^{-1}$)	1.77 ± 0.09	1.80 ± 0.08*	1.77 ± 0.11	1.80 ± 0.12*
Total number of sprints	7.1 ± 2.1	9.6 ± 2.5**	8.0 ± 3.1	8.7 ± 3.7
SV_{max} ($m.s^{-1}$)	1.75 ± 0.09	1.77 ± 0.08	1.76 ± 0.10	1.79 ± 0.11
S_{dec}	4.7 ± 0.8	4.9 ± 1.4	4.9 ± 1.5	4.4 ± 1.0
RPE (0–10)	8.3 ± 1.3	8.6 ± 1.1	8.6 ± 0.8	9.2 ± 0.7

Values are mean ± SD. RV, reference velocity; SV_{max}, maximal sprint velocity; S_{dec}, percentage decrement score; RPE, rate of perceived exertion (Borg 0-10 scale). *p<0.05, **p<0.01 for difference with pre-.

Table 3 – Training data recorded during one set of 16 x 15-m sprints performed in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) and in normoxia (RSN).

	RSH-VHL	RSN
First sprint velocity ($m.s^{-1}$)	1.76 ± 0.09	1.89 ± 0.15
SV_{max} ($m.s^{-1}$)	1.82 ± 0.09	1.90 ± 0.15
S_{dec}	1.0 ± 3.7*	4.6 ± 1.8
$[La]_{max}$ ($mmol.l^{-1}$)	5.7 ± 2.2	4.4 ± 1.9
RPE (0–10)	8.5 ± 1.0	8.2 ± 1.3
HR (bpm)	157 ± 21	162 ± 23

Values are mean ± SD. SV_{max}, maximal sprint velocity ; S_{dec}, percentage decrement score ; $[La]_{max}$, maximal blood lactate concentration after repeated sprint set; RPE, rate of perceived exertion (Borg 0-10 scale) ; HR, heart rate. *p<0.05 for difference with RSN.

Table 4 – Arterial oxygen saturation (SpO_2) values recorded during one set of 16 x 15-m sprints performed in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) and in normoxia (RSN).

		Time (s)		Training (%)	
		RSH-VHL	RSN	RSH-VHL	RSN
SpO_2 > 88%	Time at SpO_2 > 98%	102 ± 0.4**	216 ± 0.3	88%	100%
	95% < Time at SpO_2 ≤ 98%	324 ± 0.9**	402 ± 0.8		
	92.5% < Time at SpO_2 ≤ 95%	56 ± 0.8**	6 ± 0.6		
	90% < Time at SpO_2 ≤ 92.5%	24 ± 0.7**	2 ± 0.0		
	88% < Time at SpO_2 ≤ 90%	40 ± 0.6**	0		
SpO_2 ≤ 88%	85% < Time at SpO_2 ≤ 88%	38 ± 0.9**	0	12% (80 s)	0%
	82.5% < Time at SpO_2 ≤ 85%	34 ± 0.7**	0		
	80% < Time at SpO_2 ≤ 82.5%	8 ± 0.4**	0		
	Time at SpO_2 ≤ 80%	0	0		

Values are mean ± SD. *p<0.05, **p<0.01 for difference with RSN.