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RESEARCH NOTE

APNEA TRAINING EFFECTS ON SWIMMING COORDINATION

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ABSTRACT

Lemaître, F, Seifert, L, Polin, D, Juge, J, Tourny-Chollet, C, and Chollet, D. Apnea training effects on swimming coordination. *J Strength Cond Res* 23(6): 1909–1914, 2009—Triathletes and elite breath-hold divers show an adaptive response to hypoxia induced by repeated epochs of breath holding. We hypothesized that hypoxic training could also improve swimming coordination. Before and after a 3-month breath-hold training program, 4 male swimmers performed a maximal incremental test on bicycle and a 50-m front crawl race at maximal speed without breathing so that interarm coordination could be assessed. Swim velocity, stroke rate (SR), stroke length (SL), and the arm stroke phases were calculated from video analysis. Arm coordination was quantified in terms of an index of coordination (IdC) based on the time gap between the propulsive phases of each arm. After apnea training, the forced expiratory volume in 1 second was higher (4.85 $\pm$ 0.78 vs. 4.94 $\pm$ 0.81 L, $p < 0.05$), with concomitant increases in $\dot{V}O_2$peak, minimal arterial oxygen saturation, and respiratory compensation point values (W and W·kg^{-1}) during the incremental test. Swimming performance was not improved (clean velocity and time on 50 m); however, SR was decreased and SL and IdC were increased. These results indicate that apnea training improves effectiveness at both peak exercise and submaximal exercise and can also improve swimming technique by promoting greater propulsive continuity.

KEY WORDS motor control, crawl, breath holding, hypoxia

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INTRODUCTION

Apnea training leads to well-known adaptations in metabolic response, i.e., increased apnea duration and lung volumes (24), accentuated bradycardia and vasoconstriction during apnea, and attenuated blood acidosis and oxidative stress (15,16,). Breath-hold divers have shown blunted chemosensitivity to hypercapnia and hypoxia compared with nondivers (9,13), which may result in a lower ventilatory response to submaximal exercise and less dyspnea during exercise. These changes have been shown to be beneficial for the breath-hold divers and for sportsmen (15). Moreover, breathing while swimming disturbs propulsive continuity, causing catch-up coordination: a time gap between the 2 arm propulsions (19,26). In nonexpert swimmers in particular, long inhalations affect stroke phase organization and arm coordination (7,18,19). Although the usual recommendation to sprinters is to minimize the number of breaths (1–3 breaths for 50 m), it is difficult not to breathe because of the high energy expenditure needed to reach the extremely high sprint intensities.

METHODS

Experimental Approach to the Problem

We thus hypothesized that apnea training would induce adaptive metabolic responses to hypoxemia, which (a) would be sufficient to induce significant changes in lung volumes and the exercising respiratory system and (b) would improve swimming coordination and thus propulsive continuity. The aim of the present study was therefore to determine the benefits of a 3-month apnea training program in 4 swimmers who had no experience of apnea.

Subjects

Table 1 presents the baseline anthropometric characteristics and sports activities per week as assessed by questionnaire. The initial group of this study consisted of 10 local swimmers. All swimmers that participated in this study have a regional competitive level (time for a 100-m front crawl: 58.7 s) participated in this study. They had responded to an invitation to take part in the study and were selected on the basis of the regularity of their swimming practice, which consisted of

Table 1. Anthropometric characteristics and respiratory parameters of swimmers before and after training.†‡

	Before	After
Age (y)	20.5 ± 1.7	20.5 ± 1.7
Height (cm)	178.7 ± 10.5	178.7 ± 10.5
Body mass (kg)	75.0 ± 7.1	75.1 ± 6.1
Fat mass percentage (%)	17.6 ± 4.5	17.3 ± 4.6
Sports activities ($h \cdot wk^{-1}$)	1.4 ± 0.5	1.6 ± 0.9
Swimming training ($h \cdot wk^{-1}$)	5.8 ± 3.0	5.9 ± 4.0
FVC (L)	5.61 ± 1.06	5.72 ± 1.13
FEV_1(L)	4.85 ± 0.78	4.94 ± 0.81*

†FVC = forced vital capacity; FEV_1= forced expiratory volume in 1 second.
‡Values are expressed as mean ± *SD*. Significant difference between the 2 groups, $p < 0.05$.

2 hours of swimming pool training 4 times per week. The training program takes place in second left in season and do not include hypoxic training. All swimmers were without any apnea experience. At the time of recruitment, 3 subjects with diagnosed asthma, histories of asthma, or asthma suspected from medical and lung function examination were excluded from the study. Three swimmers were lost for the follow-up study. Among them, 2 had stopped swimming, citing loss of interest and economic reasons and did not wish to have to follow anymore the apnea training program and 1 had moved to foreign countries. The subjects who dropped out after the first lung function test were not significantly different in terms of baseline characteristics, lung function, or swimming training parameters from those who carried on with the study. All subjects were nonsmokers and refrained from caffeinated beverages and heavy meals on the day of the experiment. Ventilatory parameters (forced vital capacity and forced expiratory volume in 1 second, FEV_1) were assessed 1 hour before the maximal incremental test using a spirometer (Microquark; Cosmed, Rome, Italy). For each ventilatory parameter, the best value from 3 consecutive maneuvers differing by no more than 5% was chosen (22). Before each test, a calibration was performed and values were expressed in body temperature and pressure with saturated water (BTPS) conditions.

Procedures

Maximal Incremental Test. One week before and after the apnea training program, the subjects performed a maximal incremental test ($\dot{V}O_2$peak test) on cycle ergometer according to the standards and guidelines for exercise testing (12). The cycling exercise began at a power output of 25 W for 6 minutes and was increased 50 W every 2 minutes until the subjects could not continue at the selected power output. The test was considered to be maximal according to the standard criteria for terminating exercise (12). The workloads (W and $W \cdot kg^{-1}$) corresponding to the respiratory compensation point (RCP) were also determined. Respiratory compensation point was determined using the criteria of an increase in both $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ and a decrease in $P_{ET}CO_2$ (28,29). Respiratory compensation point was detected by 2 independent observers. If there was disagreement, the opinion of a third investigator was sought.

Apnea Training. The program consisted of the repetition of 30-second apnea epochs separated by 30 seconds of breathing room air during 1 hour of steady state cycling exercise at 30% of their maximal oxygen uptake ($\dot{V}O_2$peak), in line with previous studies (15,16). Apneas were performed without prior hyperventilation, after a deep but not maximal inspiration with the chest relaxed (to avoid Muller and Valsalva maneuvers), and with visual information on the time. For 3 consecutive months, 1-hour apnea training sessions were held 3 times a week. The whole protocol was approved by the local ethics committee, and informed written consent was obtained from all subjects.

Swimming Test. In a 25-m pool, each swimmer swam 50 m at his maximal velocity. The swimmer started in the water without diving and had to cover the distance with breath holding. After 3 months of apnea training, the swimmer repeated the 50-m swim at maximal velocity with breath holding. The target time for the 50 m was based on the official competition time during the study period. After each test, all swimmers were informed of their performance, which was expected to be within ±2.5% of the target time. If the target time was not met, the subjects repeated the test after a 4-minute rest. Rating of perceived exertion (RPE) and rating of perceived dyspnea (RPD) were made at rest before and at the end of each swim trial (2,6).

Equipment and Calculations. All exercise ($\dot{V}O_2$peak test and the apnea protocol) was performed with the same electrically braked cycle ergometer (Ergometrics ER 800; Jaeger, Hoechberg, Germany). A breath-by-breath gas analyzer (CPX/D Cardiopulmonary Exercise System; Medical Graphics, St Paul, MN) measured oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$), end-tidal carbon dioxide pressure ($P_{ET}CO_2$), end-tidal oxygen pressure ($P_{ET}O_2$), ventilatory flow ($\dot{V}E$), breathing frequency (f_b), and tidal volume (V_T). Heart rate (HR) was recorded continuously during the maximal incremental test (Polar Accurex Plus; Polar Electro Oy, Kempele, Finland). SaO_2 was recorded continuously with a beat-by-beat pulse oximeter (Biox 3700; Ohmeda, Madison,

WI) placed on the index finger (5). Prior to the maximal test, the earlobe was prepared with a vasodilator cream to counteract vasoconstriction of the small skin vessels. It was then incised to sample arterialized blood, which was put into 10-μL heparinized capillary tubes. Lactate concentrations were measured 5 minutes after the end of the maximal test (Analox P-GM7). Moreover, lactate concentrations ([La]) were measured before, at 1 month, and then at the end of the training protocol with the same procedure. Temperature, barometric pressure, and humidity were measured in the laboratory just before each experimental session and did not differ between sessions.

Video Analysis and Arm Coordination. Two underwater video cameras (Sony compact FCB-EX10L, Paris, France) with rapid shutter speed (1 of 1000 seconds) were used (50 Hz), each fixed on a trolley that ran alongside the pool. One camera filmed the swimmer from the right, the other from the left. The trolleys were pulled by an operator at the same velocity as the swimmers, with each swimmer's head being the mark followed by the operator to control parallax. The right and left lateral views were synchronized and genlocked. From these views, the stroke rate (SR) (from hand entry at the first stroke to hand entry at the second stroke) and the index of coordination (IdC) were calculated, in accordance with the protocol of Chollet et al. (8). An external side view camera (50 Hz, TRV25; Sony), genlocked and mixed with the underwater right side view camera, filmed all trials of each swimmer from above the pool. This third camera measured the time over a distance of 10 m (from 10 to 15 m and from 15 to 20 m at lap 1 and from 35 to 40 m and from 40 to 45 m at lap 2) to obtain the clean velocity and SR. Plots delimited each zone of 5-m on the right and left sides of the pool. When the head of the swimmer reached the rope line of each zone entry, time was recorded. In average, there are 3 strokes per zone of 5 m. Therefore, stroke length (SL) was calculated from the velocity (V) and the SR for each zone of 5 m: $SL = (V/SR) \times 60$. Arm coordination was quantified using the IdC from the 4 phases composing 1 arm stroke (entry, pull, push, and recovery) (14). The IdC is defined as the time gap between the propulsion of the 2 arms (14). The IdC was expressed as a percentage of the mean duration of the stroke. When a lag time occurred between the propulsive phases of the 2 arms, the stroke coordination was called "catch-up" (IdC $< 0\%$). When the propulsive phase of one arm started when the other arm ended its propulsive phase, the coordination was called "opposition" (IdC $= 0\%$). When the propulsive phases of the 2 arms overlapped, the coordination was called "superposition" (IdC $> 0\%$). The average IdC was calculated for the 4 zones of 5 m. Finally, 12 strokes per swimmer were portioned in 4 zones of 5 m, which were used for the statistical analysis.

Statistical Analyses

All values are given as mean ± *SD*. A normal distribution (Ryan Joiner test) and the homogeneity of variance (Bartlett test) were verified and authorized parametric statistics (Minitab 14.10; Minitab SARL, Paris, France). When the normality test failed, a Wilcoxon test was used in each group to compare data before and after apnea training. Heart rate values were tested with repeated measure analysis of variance (ANOVA) and the other physiological parameters with a paired *t*-test. The changes in time on 50 m, V, SR, SL, and IdC were analyzed by 2-way ANOVA (fixed factor: test [2 levels]; random factor: subject [4 levels]). Pearson correlation coefficient was used to determine the

Table 2. Cardiopulmonary parameters at peak exercise and at the RCP during the maximal incremental test and coordination parameters in swimmers before and after apnea training.†‡

	Before	After	RMSD
RCP (W)	205.0 ± 25.2	272.5 ± 25.0*	40.1
RCP ($W \cdot kg^{-1}$)	2.73 ± 0.16	3.63 ± 0.23*	0.5
$\dot{V}O_2E/\dot{V}O_2O_{2RCP}$	28.4 ± 4.1	29.7 ± 3.1	3.2
$\dot{V}E/\dot{V}CO_{2RCP}$	22.8 ± 3.3	25.7 ± 2.9*	3.0
$P_{ET}CO_{2RCP}$ (mm Hg)	51.5 ± 8.4	44.5 ± 4.7*	6.8
$P_{ET}O_{2RCP}$ (mm Hg)	100.7 ± 6.0	105.7 ± 3.8	5.0
V ($m \cdot s^{-1}$)	1.55 ± 0.13	1.56 ± 0.12	0.12
SR (Hz)	0.85 ± 0.06	0.81 ± 0.06*	0.10
SL ($m \cdot stroke^{-1}$)	1.84 ± 0.10	1.93 ± 0.08*	3.44
IdC (%)	−11.30 ± 5.05	−8.90 ± 4.60*	4.52

†RMSD = the root mean square differences; RCP = respiratory compensation point; $\dot{V}E$ = ventilatory flow; $\dot{V}O_2$ = oxygen consumption; $\dot{V}CO_2$ = carbon dioxide production; $P_{ET}CO_2$ = end-tidal carbon dioxide pressure; $P_{ET}O_2$ = end-tidal oxygen pressure; V = average velocity; SR = stroke rate; SL = stroke length; IdC = index of coordination.

‡Values are expressed as mean ± *SD*. Significant difference between before and after training, $*p < 0.05$.

correlation between the data from the incremental test and the results from the coordination parameters. The root mean square differences (RMSD) were established for the displacement measures, in order to estimate the magnitude of the varying quantity, using the formula: RMSD = $\sqrt{(x_1^2 + x_2^2 + \ldots + x_n^2/N)}$, with x_1, $x_2 \ldots x_n$ = the n values of the variable studied and N = the number of values (20). The level of significance was set at 0.05.

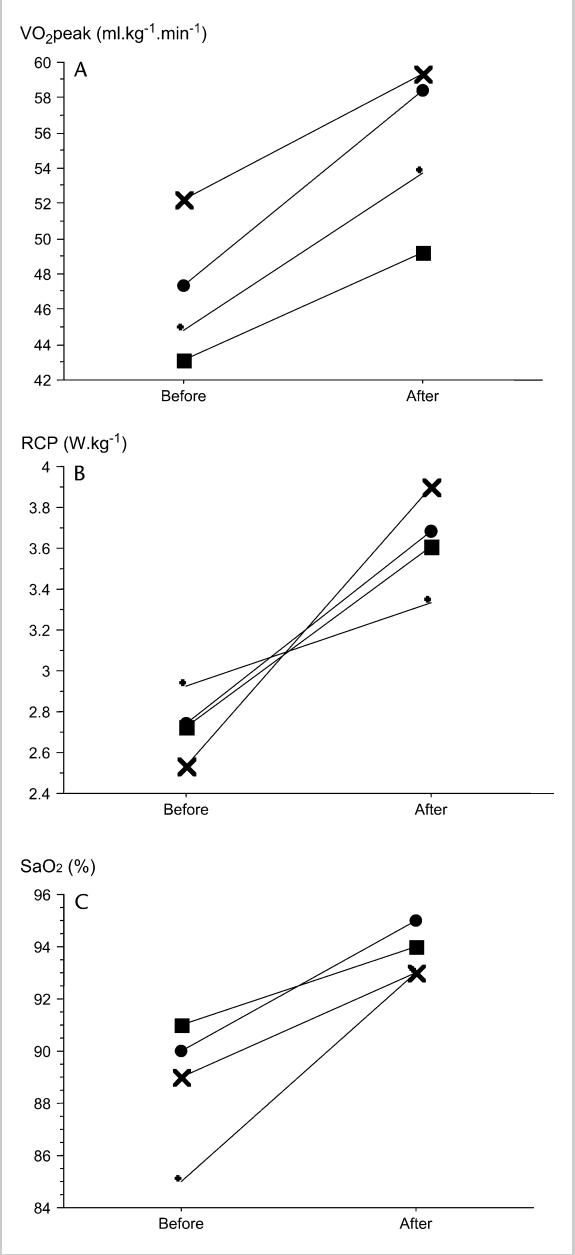

Figure 1. Individual changes in maximal oxygen consumption ($\dot{V}O_2$), oxygen consumption at the respiratory compensation point (RCP), and arterial oxygen saturation (SaO_2) before and after the apnea training.

Results

Anthropometric Characteristics, Lung Function Parameters at Rest, and Parameters at Peak Exercise

There were no between-period (before vs. after apnea training) differences in the anthropometric characteristics or ventilatory functions except for FEV_1, which was higher after training ($p < 0.05$, Table 1). There were no changes in the training parameters throughout the training period.

No significant changes were observed before vs. after apnea training for the peak exercise parameters ([Lapeak] [mM]: 14.3 ± 3.0 vs. 15.2 ± 1.96, ns respectively). However, $\dot{V}O_2$peak, minimal arterial oxygen saturation (SaO_2min), and RCP (W and W·kg^{-1}) increased after the training period (Figure 1). At RCP, $\dot{V}E/\dot{V}CO_2$ was increased and $P_{ET}CO_2$ was decreased ($p < 0.05$ for both) after apnea training, with no difference for the other parameters (Table 2).

Swimming Coordination and Assessments

The variability of the swimming parameters assessed by the RMSD (V, SR, SL, and IdC) was rather low, indicating a good reliability of our measures (0.12 m·s^{-1}; 0.10 Hz; 3.44 m·stroke^{-1}; 4.42%, respectively). The swimmers did not improve their performance (clean velocity and time on 50 m) between the 2 tests (time for test 1: 29.34 ± 2.26 seconds vs. test 2: 30.16 ± 2.16 seconds; ns); however, they improved their technique. They significantly decreased their SR ($F_{1,11} = 15.57$; $p < 0.05$) and increased their SL ($F_{1,11} = 12.41$; $p < 0.05$) and IdC ($F_{1,11} = 18.83$; $p < 0.05$), showing greater propulsive continuity between the 2 arms (Table 2).

The RPE and RPD scores decreased after apnea training (12.7 ± 1.9 vs. 10.0 ± 0.8 and 4.2 ± 0.9 vs. 2.5 ± 0.6; $p < 0.05$, respectively). Moreover, IdC and SL were associated with RPE and RPD scores after apnea training ($r = -0.992$, $p < 0.01$; $r = -0.906$, $p < 0.05$ and $r = -0.964$, $p < 0.05$; $r = -0.943$ $p < 0.05$, respectively).

Discussion

The main findings of the present study indicate that a 3-month period of apnea training induced significant improvements in FEV_1, $\dot{V}O_2$peak, and RCP values. In addition, the training led to improved swimming technique with greater propulsive continuity between the 2 arms and decreased RPE and RPD scores.

It has been suggested that swimmers are able to achieve greater lung volumes than either runners or control subjects, not because of greater inspiratory muscle strength but because they develop physically wider chests that contain an increased number of alveoli rather than alveoli of increased size (3). However, swimmers may have higher FEV_1 independent of stature and age in comparison with both land-based athletes and sedentary controls (11). Similar increased ventilatory function has also been described in trained breath-hold divers (10,21). Indeed, high breath-holding ability and swim training may facilitate the development of greater strength in the respiratory musculature. This

may have led to higher lung volumes in the swimmers of the present study. However, because no control group was used to compare the ventilatory parameters, we cannot determine whether the increased ventilatory capacity was explained by swim training alone or swimming and apnea training both. Furthermore, the swimmers of the present study presented a significant increase in $\dot{V}O_2$peak, SaO_2min, and RCP power after the training period. These results may indicate that after this period, the swimmers were more effective during submaximal exercise and at peak exercise. This improvement in ventilation was reflected by the level of SaO_2 and the perceived dyspnea, which decreased after training. Similar results were found in trained breath-hold divers, suggesting greater oxygen conservation in both static and dynamic conditions (10,23) due to splenic contractions (4). Although not directly measured in our investigation, our lower desaturation may imply these contractions. At the RCP, $\dot{V}E/\dot{V}CO_2$ was increased and $P_{ET}CO_2$ was decreased after apnea training, indicating a possible change in the ventilatory response to CO_2. The swimmers' apnea training may have displaced the CO_2 sensitivity threshold and reduced the central chemoreceptor sensitivity, thereby reducing the drive to breathe. Similar changes have been described in diving groups such as elite divers and underwater hockey players (9,13), as well as in swimmers but after hypoxic training conducted in swim sessions using a 7-stroke breathing pattern (14). Indeed, with this type of apnea training, the swimmers showed better tolerance to CO_2 than before training, which was characterized by a lower ventilatory response to submaximal exercise at RCP.

Apnea training enabled the swimmers to better support breath holding during the 50-m sprint, and consequently, stroke organization was less disturbed. After apnea training, fatigue appeared later and the disturbing effect of breathing on arm coordination disappeared (25). These "skilled" swimmers decreased their SR and increased their SL and IdC, showing greater propulsive continuity between the 2 arms after this specific training. Although the swimmers used a catch-up mode of arm coordination both before and after apnea training, this catch-up decreased because of the greater continuity. Similarly, although they did not improve their performance with training, their propulsions were more efficient because of the greater SL. Seifert et al. (26) for a 100 m and Alberty et al. (1) for a 200 m showed that throughout a race, nonelite swimmers increased their IdC but decreased their SL and SR, indicating a fatigue effect. In our case, the IdC increased, but SL did also, highlighting the beneficial effect of apnea and also swim training, as previously observed (28). Several authors (17) have shown that this change in stroke organization is due to a shorter catch and glide and a longer pull phase. Toussaint et al. (27) showed that such skilled swimmers exert less metabolic power in giving kinetic energy to masses of water, with the net result being more power devoted to propelling the swimmer forward. On the other hand, the high velocity reached during the swim trials, and thus with the intensity above the lactate threshold, may explain in part the decrease in SL that is usually observed. It is well known that when the velocity is slow or aerobic, swimmers can control the velocity and simultaneously keep the SL values at their constant high levels. When the intensity increases above the lactate threshold, the reduction in SL becomes progressively greater (17). This may be explained by the developing local muscle fatigue. In our study, the swimmers were able to decrease their SR and increase their SL and IdC to be more propulsive at the same race velocity. Although SaO_2 and HR were not recorded during the swim trials before and after the training program, we assume that the swimmers of the present study tried to maintain their speeds by decreasing SR and at the same time increasing SL. This assumption was confirmed by lower dyspnea after the apnea training, as indicated by the decrease in RPE score and its correlations with IdC and SL.

This study showed that a 3-month period of apnea training induced increases in FEV_1, $\dot{V}O_2$peak, and RCP values. In addition, the training led to improved swimming technique with greater propulsive continuity between the 2 arms.

Practical Applications

- Apnea training can develop greater propulsive continuity in front crawl and thus improve swimming technique. We suggest a program of repeated intervals of 30-second apnea epochs separated by 30 seconds of breathing room air during 1 hour of steady state cycling exercise at 30% of $\dot{V}O_2$peak.
- Repeated apnea interval training can also improve effectiveness at submaximal and peak exercise.
- To reduce the monotony of the training programs of sprint specialists, coaches may wish to use a mixed program with specific apnea training out of water on bicycle and in water.

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