

Physiological responses to repeated apneas in underwater hockey players and controls.

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Lemaitre F, Polin D, Foulia F, Boutry A, LePessot D, Chollet D, Tourny-Chollet C. Physiological responses to repeated apneas in underwater hockey players and controls. *Undersea Hyperb Med* 2007; 34(6):407-414. The aim of this study was to investigate the effects of short repeated apneas on breathing pattern and circulatory response in trained (underwater hockey players: UHP) and untrained (controls: CTL) subjects. The subjects performed five apneas (A1-A5) while cycling with the face immersed in thermoneutral water. Respiratory parameters were recorded 1 minute before and after each apnea and venous blood samples were collected before each apnea and at 0, 2, 5 and 10 minutes after the last apnea. Arterial saturation (SaO₂) and heart rate were continuously recorded during the experiment. Before the repeated apneas, UHP had lower ventilation, higher P_{ET} CO₂ (p<0.05) and lower P_{ET} O₂ than CTL (p<0.001). After the apneas, the P_{ET} O₂ values were always lower in UHP (p<0.001) than CTL but with no difference for averaged P_{ET} CO₂ (p=0.32). The apnea response, i.e., bradycardia and increased mean arterial blood pressure, was observed and it remained unchanged throughout the series in the two groups. The SaO₂ decreased in both groups during each apnea but the post-exercise SaO₂ values were higher in UHP after A2 to A5 than in CTL (p<0.01). The post-apnea lactate concentrations were lower in UHP than in CTL. These results indicate that more pronounced bradycardia could lead to less oxygen desaturation during repeated apneas in UHP. The UHP show a specific hypoventilatory pattern after repeated apneas, as well as a more pronounced cardiovascular response than CTL. They indeed showed no detraining of the diving response.

INTRODUCTION

The diving response is characterized by bradycardia, peripheral vasoconstriction, decreased cardiac output, and a gradual elevation in arterial pressure with consequent lactate accumulation in unperfused muscle, and elite apnea divers display a more pronounced response than novices (1). Apnea diving is a new sport that is usually characterized by

prolonged apneas at rest or at low exercise intensity. However, in several sports, such as judo, track and field, basketball and soccer, athletes are required to perform high intensity intermittent exercise. Intermittent training decreases the recovery period after submaximal exercise (2) and this effect has been explained by a smaller increase in lactate concentration, an improvement in the quantity of oxygen available to active muscles, and better oxygen extraction (3). Underwater hockey players

(UHP) undergo intensive intermittent training and, in addition, engage in activities that require repeated breath-holding maneuvers and controlled breathing patterns during training and competition. These players indeed show a higher tolerance to carbon dioxide than non-divers (4), characterized by a lower ventilatory response to any submaximal exercise and less dyspnea during exercise or the recovery between repeated apneas. However, the elevated PaCO₂ in these UHP, secondary to the relative hypoventilation, may also increase heart rate (HR) and reduce vasoconstriction to what is usually seen in apnea, leading to a reduced apnea time or greater reduction in PaO₂. Indeed, elite underwater hockey players are trained in short breath-holds during high intensity work. Such conditions are not likely to produce much hypoxia, and a diving response with vasoconstriction in these subjects may actually be disadvantageous physiologically; for example, a reduced apnea time. We therefore tested the hypothesis that underwater hockey training would lead to a blunting of the diving response with a diminished decrease in ventilation, HR, oxygen consumption, and vasoconstriction.

MATERIALS AND METHODS

The cohort

Twenty male subjects participated in this study. One group was composed of ten underwater hockey players (UHP), all members of a national underwater hockey team (National 1), and the other group comprised ten controls (CTL). The UHP responded to an invitation to take part in the study and were selected on the basis of the regularity of their underwater hockey practice and membership on the national team. Ten students composed the CTL group. They had no apnea experience and had a low volume of sports training (<5 hours.week⁻¹).

Table 1. Anthropometric characteristics, respiratory parameters and cardiopulmonary parameters at peak exercise during the maximal incremental test of underwater hockey players (UHP) and controls (CTL).

	UHP n= 10	CTL n= 10
Age (years)	25.7±5.2	23.5±2.5
Height (cm)	180.3±8.1	178.3±6.4
Body mass (kg)	82.9±14.1*	72.6±7.6
Sports activities (hours.week ⁻¹)	3.8±2.5	4.2±1.9
Hockey training (hours.week ⁻¹)	7.8±4.0	/
$\dot{V}O_{2peak}$ (ml.min ⁻¹ .kg ⁻¹)	50.10±3.45	45.50±5.40
P _{peak} (W.kg ⁻¹)	4.24±0.65	4.05±0.30
HR _{peak} (bpm)	186±9	185±5
RER _{peak}	1.22±0.08	1.28±0.07
[La] _{peak} (mM)	8.02±0.92*	9.75±1.15
FVC (l)	5.94±0.80	5.94±0.96
FEV ₁ (l)	4.86±0.81	4.85±0.60

Values are means ± SD. FVC: forced vital capacity; FEV₁: forced expiratory volume in one second; O_{2peak}: maximal oxygen uptake; P_{peak}: maximal power output; HR_{peak}: maximal heart rate; RER_{peak}: maximal respiratory exchange ratio; [La]_{peak}: maximal lactic acid blood concentration. Significant difference between the two groups, **: p<0.01.

All subjects were non-smokers and all refrained from caffeinated beverages and heavy meals on the day of the experiment. Table 1 presents the baseline anthropometric characteristics, sports activities per week as assessed by questionnaire, and ventilatory parameters (forced vital capacity: FVC, forced expiratory volume in 1 second: FEV₁) performed 1 hour before the maximal incremental test using a spirometer (Microquark, Cosmed, Italy). For each ventilatory parameter, the best value from three consecutive maneuvers differing by no more than 5% was chosen (5). Before each test, a calibration was performed and values were expressed in BTPS conditions. The whole protocol was approved by the local Ethics Committee and informed written consent

was obtained from all subjects.

Protocol

One week before the apnea protocol, the subjects (UHP and CTL) performed a maximal incremental test ($\dot{V}O_{2\text{ peak}}$ -test) on a cycle ergometer according to the standards and guidelines for exercise testing (6) (Table 1).

Apnea protocol

The subjects relaxed for about 20 minutes while equipment was attached and until stable resting heart rate was recorded. Five apneas (A1-A5) were then performed with the face immersed in thermoneutral water ($31^{\circ}\text{C} < \text{temperature} < 32^{\circ}\text{C}$) during upright leg cycling at 75 Watts for 45 sec, according to the protocol of a previous study (7). The apnea series was assumed to reproduce the repeated exercise of underwater hockey, but the duration of the apneas was chosen to allow the development of a complete and significant cardiovascular response during dynamic leg exercise (8, 9). The apneas were spaced 5 minutes apart to permit splenic contractions, which are a characteristic of the human diving response (10, 11) and demonstrated during intermittent hypoxia (10, 12). Apneas were performed without prior hyperventilation, after a deep but not maximal inspiration with the chest relaxed (to avoid Muller's and Valsalva maneuvers), and without any feedback of the apnea time from the investigators. Five seconds before the end of the apnea, the subjects lifted their heads above the water surface on the investigator's instruction, held the breath until the face was dried, and then terminated the apnea by maximal expiration through an open-circuit spirometer mouthpiece. Respiratory flow and volumes were recorded for 1 minute before and 1 minute after each apnea. The ambient air temperature was $26\text{-}27^{\circ}\text{C}$.

Equipment and calculations

All exercise ($\dot{V}O_{2\text{ peak}}$ -test) and the apnea protocol) was performed with the same electrically braked cycle ergometer (Ergometrics ER 800, Ergoline, Jaeger, Germany). A breath-by-breath gas analyzer (CPX/D Cardiopulmonary Exercise System, Medical Graphics, St Paul, MN, USA) allowed the measurements of: oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$), end-tidal carbon dioxide pressure ($P_{\text{ET}}CO_2$), end-tidal oxygen pressure ($P_{\text{ET}}O_2$), ventilatory flow ($\dot{V}E$), breathing frequency (f_b), and tidal volume (V_T). HR was recorded continuously (Polar Accurex plus, Polar Electro Oy, Kempele, Finland) and the average HR values were calculated for the period 90-30 sec before each apnea and at the end of the apneic tests for the last 10 sec. SpO_2 was recorded continuously with a beat-by-beat pulse oximeter (Biox 3700, Ohmeda, Madison, WI, USA) placed on the index finger (13) and an average value was calculated for the period 90-30 sec before each apnea and from a 10-sec period based on the nadir after each apnea. The subjects were prepared by rubbing the ear with a capsaicin ointment to enhance local blood flow (13). Blood pressure was measured 1 minute before and just after each apnea from the dominant arm using a semi-automated BP measurement device (Tensiometer model UB 401, A & D, Mississauga, Ontario). The tensiometer values were compared with the values recorded by sphygmomanometer before and after each apnea, and the results were in good agreement. The Mean Arterial Blood Pressure (MAP) was then calculated according to the following standard equation: $MAP = (SBP + 2DBP)/3$ where DBP is the Diastolic Blood Pressure and SBP is the Systolic Blood Pressure in mmHg. Prior to the experiment, 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- μ l

heparinized capillary tubes. Sampling was done at rest 2 minutes before A1, 2 minutes after A3 and A5, and then 5 and 10 minutes after the last dynamic apnea. Lactate concentrations were then measured immediately (Analox P-GM7), as were hematocrit and hemoglobin (Radiometer, ABL 725). Temperature, barometric pressure and humidity were measured in the laboratory just before each experimental session. All parameters were averaged over the five apneas and compared between UHP and CTL.

Statistical analysis

All values are given as means \pm SD. Normality was checked and for variables with a normal distribution, a one-way repeated-measure analysis of variance (ANOVA) was used to assess the changes in parameters with repeated apneas. A two-way repeated-measure ANOVA (i.e. group \times period) was also used to compare the values of each parameter before and after the apneas in the two groups (UHP and CTL), as well as the static apnea data with the post-exercise apnea data. A PLSD Fisher post-hoc test was then used for all pair wise comparisons of the mean responses in the two groups. Stepwise regression analyses were performed to determine which independent variables (HR min, % HR decrease, HR and MAP after each apnea) explained the desaturation changes during the repeated apneas. The results were considered significant at $p < 0.05$. All statistical analysis was performed with Statview (Abacus Concepts, Inc., Berkeley, CA, USA).

RESULTS

Anthropometric characteristics and parameters at peak exercise

UHP were heavier than CTL ($p < 0.05$) and no difference between groups was found for the other anthropometric characteristics (Table 1). There were no between-group differences

in ventilatory functions or incremental test parameters (Table 1), except for maximal lactic acid production ($[La]_{peak}$), which was lower in UHP than in CTL ($p < 0.01$).

Effect of repeated apneas

Breathing pattern: The $\dot{V}E$, $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ values, averaged across time, were lower in UHP than in CTL before the apneas (Table 2). After the apneas, only $\dot{V}E/\dot{V}O_2$ remained higher in UHP than CTL ($p < 0.05$).

Table 2. Cardiorespiratory responses before and after repeated apneas in underwater hockey players (UHP) and controls (CTL).

	Before (T0)		After (T1)	
	UHP	CTL	UHP	CTL
$\dot{V}E$ (l.min ⁻¹)	16.4 \pm 4.6**	19.2 \pm 5.4	52.2 \pm 14.3	51.8 \pm 13.4
$\dot{V}E/\dot{V}O_2$	35.4 \pm 7.0**	40.4 \pm 1.0	26.7 \pm 6.7*	23.0 \pm 5.7
$\dot{V}E/\dot{V}CO_2$	35.5 \pm 5.8*	38.7 \pm 7.4	30.8 \pm 4.5	31.1 \pm 5.9
$P_{ET}CO_2$ (mmHg)	35.0 \pm 3.7*	32.9 \pm 5.1	54.2 \pm 4.5	51.6 \pm 8.1
$P_{ET}O_2$ (mmHg)	100.0 \pm 13.5***	110.4 \pm 6.6	57.9 \pm 13.0**	66.20 \pm 12.9
HR (bpm)	78 \pm 9	78 \pm 18	81 \pm 14**	90 \pm 15
MAP (mmHg)	102 \pm 10***	94 \pm 7	100 \pm 16**	93 \pm 10
DBP (mmHg)	85 \pm 11***	77 \pm 8	71 \pm 18	67 \pm 13
SBP (mmHg)	136 \pm 11***	126 \pm 9	159 \pm 24*	146 \pm 13
SaO ₂ (%)	97 \pm 2	96 \pm 1	94 \pm 3*	92 \pm 2

End-tidal carbon dioxide pressure (PETCO₂), end-tidal oxygen pressure (PETO₂), ventilatory flow (E), oxygen consumption (O₂) and carbon dioxide production (CO₂), the mean arterial blood pressure (MAP); the diastolic blood pressure (DBP), the systolic blood pressure (SBP) and arterial oxygen saturation decreases (SaO₂ %). Values are means over the 5 apneas \pm SD. *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$.

Before the apneas, UHP had higher averaged $P_{ET}CO_2$ ($p<0.05$) and lower $P_{ET}O_2$ than controls ($p<0.001$) (Table 2). After the apneas, the $P_{ET}O_2$ values were always lower in UHP than controls ($p<0.001$) but with no difference for averaged $P_{ET}CO_2$.

Cardio-circulatory change: The post-apnea HR decreases remained unchanged throughout the series for UHP and CTL, with a greater HR decrease only in A4 for UHP ($p<0.05$) (Figure 1). The post-apnea HR

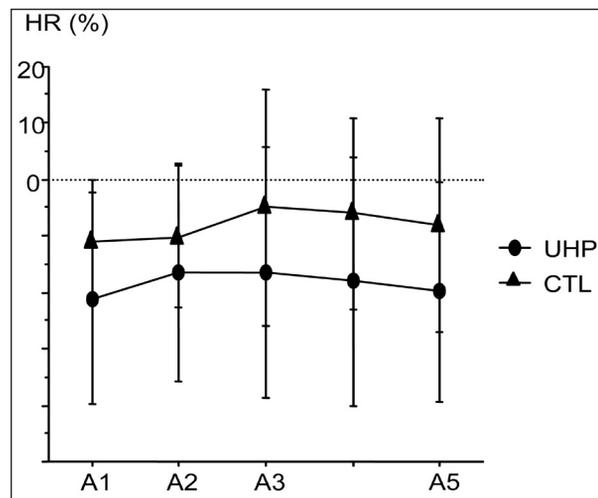


Fig. 1. Heart rate decreases (HR %) in underwater hockey players (UHP) and controls (CTL) recorded during each apnea (5 apneas with exercise: A1 to A5) *: $p<0.05$ for differences between UHP and CTL.

averaged over the five apneas was lower in UHP than CTL ($p<0.01$) (Table 2). Before the apneas, the averaged SDP, DBP and MAP were always higher in UHP than in CTL ($p<0.001$, respectively) (Table 2). After the apneas, only SBP and MAP remained higher in UHP than CTL ($p<0.05$ and $p<0.01$, respectively) (Table 2). The SaO_2 values were higher in UHP than in CTL after A4 and A5 ($p<0.05$) (Figure 2). After the apneas, the averaged SaO_2 values were higher in UHP than in CTL. No difference was found between UHP and CTL for hematocrit or hemoglobin kinetics. Lactate concentrations were lower in UHP than in CTL at 5 and 10 minutes post-apnea (A5) (Figure 3). In UHP

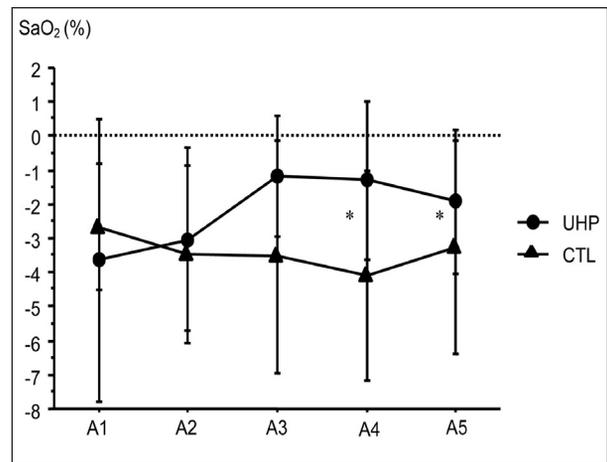


Fig. 2. Arterial oxygen saturation decreases (SaO_2 %) recorded after each dynamic apnea (A1 to A5) in underwater hockey players (UHP) and controls (CTL) *: $p<0.05$ for differences between UHP and CTL.

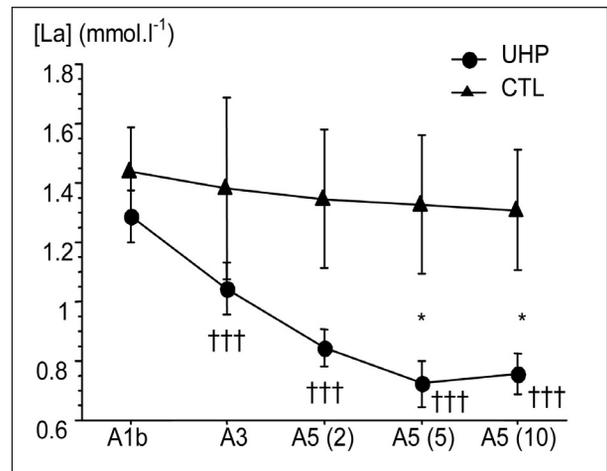


Fig. 3. Lactate (La) in underwater hockey players (UHP) and controls (CTL) recorded before the first apnea (A1b), after the third apnea (A3), and 2, 5 and 10 minutes after the fifth apnea (A5) *: $p<0.05$ for differences between UHP and CTL. †††: $p<0.001$ for the differences between A1b and A3, 2, 5 and 10 minutes after A5 for UHP only.

only, stepwise regression analyses showed that the main factor contributing to the reduction in SaO_2 was HR (for A1 to A5; $r=0.810, 0.676, 0.777, 0.815$ and 0.608 , respectively; $p<0.05$).

DISCUSSION

The main finding of this study is that the underwater hockey players display a specific

cardiorespiratory pattern characterized mainly by low ventilation before the apneas, as well as a more pronounced diving response during the apneas, than control CTL subjects, who showed signs of slight anticipatory hyperventilation.

Respiratory parameters and breathing pattern

Although they had had specific underwater hockey training, the UHP have the same level of fitness than CTL, as reflected by their similar maximal power output. Indeed, brief, intense exercise apnea training may induce performance adaptations comparable to those of traditional endurance training (14). Their lower $[La]_{peak}$ during the incremental test may indicate that they produce less lactic acid than the untrained subjects during heavy exercise, as suggested in a previous study (15). Increased ventilatory function has been described in Ama divers (16) and trained breath-hold divers as compared with a standard population (17, 18). However, we found no significant difference between our UHP and CTL, in agreement with the findings of Davis et al. (4). These results may be explained by the specific training pattern of UHP, i.e., frequent, brief and intense dynamic apneas, and by their short inspirations with uncompleted lung inflation compared with free divers.

In our study, $\dot{V}E$, $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ were lower before each apnea in UHP than in CTL, indicating hypoventilation. These results may also indicate a possible change in the ventilatory response to CO_2 . The UHPs' intermittent exposure to hypercapnia may have displaced the CO_2 sensitivity threshold and reduced central chemoreceptor sensitivity, thereby reducing the drive to breathe. This effect has been described in diving groups such as synchronized swimmers, Amas, elite divers and underwater hockey players (4, 19, 20) and indicates that these players have a higher tolerance to carbon dioxide than non-

divers (4). Moreover, it has been found that re-breathing the air in the snorkel will cause an increase in inspired CO_2 that changes the ventilation and the work of breathing (21). Furthermore, because CTL started the apneas with more O_2 and less CO_2 in their lungs and blood, they showed higher O_2 and lower CO_2 at the end of the apneas. Because the CTL were not trained at apnea, they indeed showed signs of slight anticipatory hyperventilation. Moreover, the decrease in $P_{ET}O_2$ does not seem low enough to have affected O_2 delivery, while the increase in CO_2 had effects on ventilation. These results agree with previous studies (22, 23) and indicate that hockey training allows UHP to sustain higher $P_{ET}CO_2$ and lower $P_{ET}O_2$ than controls and thus to perform intense dynamic apneas. Such differences might be attributed to a need to buffer lactic acidosis with increased ventilation at lower exercise intensities in untrained subjects and/or to an attenuated chemoresponse to CO_2 . Moreover, apneas may be associated with large shifts in tissue O_2 and CO_2 stores and much of these shifts can be explained by primary circulatory events.

Cardio-circulatory changes

Our observed changes in cardiovascular parameters are consistent with the response patterns noted in previous studies (24, 25). The exercise stimulus to increase HR seemed less able to overcome the diving stimulus to decrease HR in UHP than in CTL, in agreement with previous results in trained breath-hold divers (7, 26). However, this reduced tachycardia found in UHP may indicate either a higher metabolic demand was placed on the cardiac and skeletal muscles of CTL than UHP or the exercise-induced decrease in cardiac vagal tone overrode the trigeminal input from the face, which primarily increases cardiac vagal tone, in the untrained subjects (27). The apnea series increased SBP and decreased DBP

for both groups but to a lesser magnitude for CTL (+17% vs. +16% and -16.5% vs. -13%, for UHP and CTL, respectively). In fact, the blood pressure elevations could be the result of an increased total peripheral resistance to muscular contraction and to successive maximal apneas (9) and thus may reflect a greater peripheral vasoconstriction in UHP than CTL that modulates the exercise hyperemia. Indeed, although vasomotor tone was inferred and not measured during our repeated apneas, a gradual diminution in muscle vasodilatation and tachycardia was observed in both UHP and CTL, while muscle vasoconstriction became more prominent in UHP than in CTL. Moreover, the lactic acid concentrations in our UHP were reduced markedly more after the apnea series than in controls, in agreement with previous studies (6). Several hypotheses can be advanced to explain these results. First, the observation that PO_2 values dropped more in UHP than in CTL after breath-hold exercise is an indication that the need for an anaerobic energy supply is low for UHP. Second, the low lactate accumulation observed in the UHP of this study is similar to the findings of other studies employing intermittent training (15). Last, our results may indicate reduced production (not consistent with lower aerobic metabolism) and greater uptake and/or slower washout in UHP.

The Hct and Hb kinetics did not differ between the two groups across time (15.8 ± 1.2 g/dl vs. 15.7 ± 1.1 g/dl, NS, and 48.7 ± 3.3 % vs. 48.05 ± 3.2 %, NS, respectively, between UHP and CTL), but the desaturation was lower in UHP than in CTL after the repeated apneas (A2-A5) from the fourth to the fifth apnea (Figure 2). Similar results were found in trained breath-hold divers suggesting greater oxygen conservation in both static and dynamic conditions (17) due to splenic contractions (10). Although not directly measured in our investigation, our lower desaturation may implicate these contractions. The lack of a statistically significant difference

in the Hct and Hb kinetics may have hidden a physiological response which, however slight, could have significant effects on apneas. Stepwise regression analyses showed that the main factor contributing to the reduction in SaO_2 was HR (A1-A5) ($p < 0.05$) only in UHP. This finding also suggests that more pronounced bradycardia could lead to less oxygen desaturation during repeated apneas in UHP.

CONCLUSION

In conclusion, the UHP did not show any detraining of the diving response. They showed a specific ventilatory pattern after repeated apneas, with lower minute ventilation, higher vasoconstriction and lower lactate concentration than CTL during recovery. These findings require further investigation to determine whether continuous intermittent apneas during maximal exercise have different effects than sustained apneas at rest or low metabolic levels.

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