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**CARDIO-VASCULAR CHANGES INDUCED BY COLD WATER IMMERSION  
DURING HYPERBARIC HYPEROXIC EXPOSURE**

**Running Head** : Hyperbaric hyperoxia and cold water immersion

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**Abstract:**

The present study was designed to assess the cardiac changes induced by cold water immersion compared with dry conditions during a prolonged hyperbaric and hyperoxic exposure (ambient pressure between 1.6 to 3 ATA and  $PiO_2$  between 1.2 to 2.8 ATA).

Ten healthy volunteers were studied during a 6 hours compression in a hyperbaric chamber with immersion up to the neck in cold water while wearing wet suits. Results were compared with measurements obtained in dry conditions. Echocardiography and Doppler examinations were performed after 15 minutes and 5 hours. Stroke volume, left atrial and left ventricular (LV) diameters remained unchanged during immersion whereas they significantly fell during the dry session. As an index of LV contractility, Percent Fractional shortening remained unchanged, in contrast with a decrease during dry experiment. Heart rate significantly decreased after 5 hours, although it had not changed during the dry session. The changes in the total arterial compliance were similar during the immersed and dry sessions, with a significant decrease after 5 hours. In immersed and dry conditions, cardiac output was unchanged after 15 minutes but decreased by almost 20% after 5 hours. This decrease was related to a decrease in heart rate during immersion and to a decrease in stroke volume in dry conditions.

The hydrostatic pressure exerted by water immersion on the systemic vessels could explain these differences. Indeed, the redistribution of blood volume toward the compliant thoracic bed may conceal a part of hypovolemia that developed in the course of the session.

*248 words*

**Keys words :** Diving, hyperbaric hyperoxia, cardiac function, cold exposure, healthy volunteers, echocardiography-Doppler

## INTRODUCTION

The diving-induced changes in hemodynamics have been mostly studied during experiments conducted in hyperbaric chambers. When the ambient pressure is increased, the elevated oxygen partial pressure (hyperoxia) leads to bradycardia, decreases cardiac output and triggers an increase in systemic vascular resistance (Whalen *et al.*, 1965; Hahnloser *et al.*, 1966; Shida & Lin 1981; Pelaia *et al.*, 1992; Lund *et al.*, 1999; Rossi & Boussuges, 2005). A negative inotropic effect of hyperoxia has been reported during exposure to normobaric conditions (Berk *et al.*, 1983; Lodato, 1989), but was not found during exposure to hyperbaric hyperoxia as the increased ambient pressure causes an elevation in cardiac contractility (Savitt *et al.*, 1994; Risberg *et al.*, 1995). We previously reported our observations on hemodynamic changes in healthy subjects during prolonged hyperbaric hyperoxic exposure while breathing through SCUBA, fasting and with intermittent exercise (Molenat *et al.*, 2004). During open sea diving, in addition to hyperoxia and increased ambient pressure, the subjects face environmental constraints such as immersion and cold exposure. These environmental features are also known to be responsible for hemodynamic changes (Lin, 1984). We hypothesised that the supplemental constraints induced by cold water immersion modify the hemodynamic profile usually observed during experiments conducted in hyperbaric chambers. Therefore, the present study was designed to assess the circulatory and cardiac changes induced by cold water immersion compared with dry conditions during a prolonged hyperbaric and hyperoxic exposure (HH). Ten subjects underwent two similar 6 HH exposures performing a dry session (Dry HH), and after 4 weeks, a wet session with immersion up to the neck (sternoclavicular notch) in cold water (immersed HH) while wearing wet suits. The hemodynamic modifications during immersion were studied using trans-thoracic pulsed Doppler echocardiography with the probe in a wet suit muff. Results were compared with measurements obtained in dry conditions.

## MATERIAL AND METHODS

### **Experimental protocol**

The experimental procedure was designed to insure the conditions of a real dive as near as possible, combining HH, SCUBA breathing and exercise, without any opportunity to drink or eat during the session. The hyperbaric chamber consisted of a large cylindrical chamber (diameter 2m, length 4m), where the humidity was maintained between 50 and 80 % and the temperature between 20 and 24 °C.

**Ten trained military divers aged 32 +/- 5 years (26 - 40 years), weight (82 +/- 11 kgs (61 – 104 kgs) and height 181 +/- 9 cm (168 – 193 cm)** took part in the experiment. All subjects had a negative history of hypertension and cardiovascular or kidney diseases, and none was taking any medication at the time of the study. The local Ethics Committee approved the protocol, and written informed consent was obtained after careful oral and written explanation of the experiment. Criteria for the experiment to be stopped were the request of the subject or the appearance of convulsions or any preliminary sign of seizure.

All subjects underwent two similar 6-h exposures with 1-month intervals, the first one in dry conditions and the second immersed up to the neck, in cold water. During the dry experiments, divers wore cotton short and tee-shirt. During the immersed session, the average water temperature was 14.4°C (min 13.6° C – max : 15.9°C). Divers wore a polar fiber underwear, a 2mm neoprene farmer wet suit and a 7mm Neoprene farmer and jacket wet suit (total of 16 mm neoprene on the thorax), individually fitted by a local manufacturer (Squale – Toulon France).

The volunteers spent the two experimental sessions (dry and immersed) in the same sitting position. The subjects sat in a tank containing a submersible mechanical

cycle ergometer designed for the study. Rectal temperature was continuously monitored. Subjects were also required to report any cold sensation or shivering.

In both conditions, subjects were breathing using a closed-circuit SCUBA (Oxymixgers 97, Aqualung, Carros, France) positioned on their backs. The breathing gas was 100 % oxygen or an inspired gas enriched with oxygen (60% oxygen/40% nitrogen).

**The French Navy has previously measured the supplemental work and the inspiratory and expiratory pressures induced at 10-meter depth by the breathing apparatus, positioned on the back with subjects in a sitting position (Figure 1). A supplemental work amounting at 0.5 Joule per liter of ventilation has been measured and the pressure was found negative (-4 cm H<sub>2</sub>O) at the inspiratory time, and positive (+5 cm H<sub>2</sub>O) at the expiratory time.**

The continuous monitoring of the ambient pressure and of the FiO<sub>2</sub> in the inspiratory circuit allowed a continuous monitoring of the PiO<sub>2</sub>.

All subjects were exposed for 6 hours to the same compression profile, with pressure varying between 1.6 to 2 ATA (1 ATA = 101.325 kPa) with some short descent to 2.8 ATA. Divers were exposed to a PiO<sub>2</sub> of 1.2 ATA during 180 minutes (min), a PiO<sub>2</sub> between 1.2 and 1.6 ATA during 150 min, a PiO<sub>2</sub> above 1.6 ATA with some peak up to 2 ATA, during 20min and a PiO<sub>2</sub> under **1 ATA** during 10min. Each experimental exposure comprised the same pattern of alternating periods of rest and exercise (two cycling periods for a total duration of 165 minutes).

Before and after each exposure, venous blood samples from an antecubital vein, electrocardiographic (ECG) data, and measurements of SAP and DAP were taken after 20 min of rest in a quiet room at 24°C. Moreover, the subjects were weighed on a precision scale (Ohaus, Pine Brook, NJ, USA)

## **Echographic and Doppler study**

### *Technical considerations*

The echocardiography-Doppler examinations were performed using a Dasonics **Vingmed** CFM 750 type ultrasonography machine (GE Ultrasound, Millwaukee, USA).

The same measurements were performed on a control day at normobaric pressure and during the two experimental sessions, the first in dry condition and the second during head out water immersion. The ultrasonography unit was located outside the hyperbaric chamber and connected to a combined 3.25 MHz imaging/2.5 MHz Doppler probe inside the chamber via a feed-through into the hull. This technique did not expose the ultrasonographic unit to pressure changes. The explorations were carried out by an investigator (AB or AG) compressed to the pressure of the subjects within the chamber. The probe was brought up to the thorax through a wet suit muff.

Subjects were in a sitting position with a slight left lateral shift, on a seat located below a porthole. The inside investigator attempted echocardiography Doppler examination with visual control on the outside monitor through the porthole. Also, a second investigator (FM, AG) stayed outside the hyperbaric chamber and positioned the monitor in such a way that the inside investigator could see it. The two investigators communicated via a microphone. The investigator outside the chamber processed the recording of the echocardiogram using the Dasonics Vingmed computer system, and also recorded the examinations on VHS videotapes, to be reviewed and analysed later. The mean duration for each test was about 30 minutes.

### Schedule of the echographic examination:

A reference examination, at sea level, was performed 4 to 5 days before the first experimental session. At this time, any cardiac abnormalities led to the exclusion of the

subject from the study. At the time of the compression, the schedule of the experiment permitted 2 echographic examinations as follows: an early examination after 15 minutes of compression and a late examination after 5 hours of compression, before the last exercise period. In both cases, the ambient pressure and  $PiO_2$  were identical, i.e. respectively 2 ATA and 1.2 ATA. 2D echocardiographic images were obtained from a transthoracic approach. Examinations were made using two dimensional and M mode echocardiography associated with pulsed and continuous wave Doppler. Each view was recorded for 30 seconds on videotape. Doppler recordings were performed at the end of a normal expiration to eliminate the effects of the respiration on the parameters studied. Measurements were averaged from at least three different beats. Tape recordings were obtained at a speed of 100mm/s with simultaneous tracing of the electrocardiogram.

Sphygmomanometric blood pressure measurements on the right arm were obtained before each examination. Mean arterial blood pressure (MAP) was calculated as :  $MAP = DAP + 1/3 (SAP - DAP)$ , where SAP and DAP were respectively the systolic and diastolic arterial blood pressure. Pulse pressure was defined as systolic minus diastolic blood pressure :  $PP = SAP - DAP$

*Echographic parameters:* We combined M-mode and 2D echocardiography with continuous and pulsed Doppler.

LV systolic function : Left atrial diameter (LA), and left ventricle end systolic and end diastolic diameters (LVEDD, LVESD) were measured by M-mode echocardiography from the left parasternal short and long axis views. The standard index of global LV systolic performance was LV percent fractional shortening (%FS). %FS was taken as the ratio  $(LVEDD - LVESD / LVEDD)$  Heart rate (HR) was recorded by echocardiogram and the rate was averaged over 60 s. Cardiac output (CO) was derived from the aortic flow. Aortic cross section area (ACSA) was first measured by 2D echocardiography from the left parasternal long axis view at the level of the aortic root. The aortic systolic



flow velocity time integral (AoVTI) was measured by computer assisted determination from the pulsed-wave Doppler profile of the aortic blood flow from the apical four chamber view, allowing the calculation of stroke volume ( $SV = AoVTI \times ACSA$ ) and CO ( $CO = SV \times HR$ ).

LV Afterload : LV meridional wall stress (LVmWS) was used as a LV afterload index, independent of the LV preload. LVmWS is a function of the inner pressure of the LV at the end of the systole (considered as equal to the systolic blood pressure (SBP) in the absence of any aortic shrinking or mitral insufficiency) and of the myocardial contractility. It was calculated as follows:  $LVmWS = (0.33 \text{ SAP} \times LVESD) / LVESPW (1 + LVESPW / LVESD)$ .

The total arterial compliance was calculated as  $SV/PP$  (Chemla et al.,1998), where PP was the pulse pressure.

Left Ventricular filling : Transmitral blood flow velocities were obtained from the apical four chamber view, positioning the sample volume at the mitral valve leaflet tips. Doppler velocity curves were recorded at 100mm/s. Peak velocity and velocity time integral (VTI) of the initial flow (E wave), representing the early filling phase, and of the late flow (A wave), representing the atrial contraction, were measured. The peak velocity ratio (E/A) and the ratio of the A wave VTI to the total VTI (relative contribution of atrial contraction to the total LV filling) were calculated. In addition, two other variables were measured: the deceleration pressure half time of early diastolic transmitral flow (PHT) and the isovolumetric relaxation time (IVRT). The IVRT was the interval from the aortic valve closure signal to the mitral valve opening signal.

### **Statistical Analysis**

Data are expressed as mean +/- SEM. Statistical tests were run on the Sigma Stat software. The cohorts for comparison consisted of the ten subjects at 3 time points:

reference, after 15 minutes of exposure to immersed HH and after 5 hours of exposure to immersed HH. The data distribution was analysed with a Kolmogorov –Smirnov test. Comparison between cohorts of continuous variables having a normal distribution was carried out with analysis of variance with repeated measures ANOVA; the post-hoc comparison of dichotomous variables was performed with a Tukey’s test. In the case of variable cohorts not having a normal distribution, comparisons were performed with non-parametric univariate analysis (Friedmann’s test) and the post-hoc dichotomous comparisons with a Dunn’s test. Differences between groups were considered significant at  $p < 0.05$ .

We also compared the values obtained at 15 min and 5 hours of immersed HH groups in the present study to values obtained at 15min and 5 hours during dry HH exposure in the same subjects with the same measurements. When the data were normally distributed, a Student’s test for paired data was used. If the distribution was not normal, the Wilcoxon’s paired signed rank test was used.

## RESULTS

Similar weight loss was induced by the two sessions (Table 1):  $- 2.2 \pm 0.4$  kg after dry HH and  $- 2.4 \pm 0.6$  kg after immersed HH. Furthermore, no significant changes in SAP and DAP were observed after the two exposures.

### Immersed HH

Divers performed immersed HH exposure without any incident. All the divers complained from cold at the end of the two rest periods. Shivering occurred after 100min of exposure in 8 divers, and ceased at the time of the first exercise period in all subjects. Four of them shivered again after 5 hours of exposure before the second exercise period. **Figure 2** displays the mean rectal temperature (Tre) recorded during the immersed sessions. A significant decrease of Tre was observed after 70 min. Tre had significantly increased 60 minutes after the beginning of the exercise, and remained increased until 40 minutes after the end of exercise. A second significant decrease of Tre occurred after 5 and half hours of exposure and disappeared after the second bout of exercise.

### **Hemodynamic and echocardiographic measurements during immersed HH (Table 2 and 3)**

Systolic arterial pressure (SAP), Mean AP and Diastolic AP remained unchanged during the whole exposure. Pulse pressure increased significantly after 5 hours of immersed HH. HR had not changed significantly after 15 minutes but was significantly decreased

after 5 hours. Stroke volume remained unchanged. In total, CO was unchanged after 15 minutes but was decreased by almost 20% after 5 hours ( $p = 0.02$ ).

LA diameter, LVEDD and LVESD did not show any change. The % FS and the LVmWS remained unchanged. The total arterial compliance was unchanged after 15 minutes but decreased significantly after 5 hours ( $p = 0.017$ ). There was no significant modification of the transmitral flow profiles and of the IVRT after 15 minutes and 5 hours of exposure of immersed HH (table 4).

### **Hemodynamic and Echocardiographic studies: Comparisons with the dry session (table 2 and 3)**

LA diameters were significantly higher during immersed HH than during dry HH. LVEDD was significantly higher during immersed than dry HH after 15 minutes but was not significantly different after 5 hours.

As LV systolic indices, %FS was significantly greater after 15 minutes exposure in the immersed HH than during dry HH but was similar in the two sessions after 5 hours exposure. Stroke volume was not different after 15 minutes in the immersed and dry sessions. After 5 hours there was a trend for a higher stroke volume when immersed than in the dry condition ( $91 \pm 14$  versus  $78 \pm 14$  ml –  $p=0.09$ ).

Heart rate was not different after 15 minutes in immersed and dry HH but was significantly lower after 5 hours immersion.

Cardiac output was similar in immersed and dry conditions, both after 15 minutes and after 5 hours.

The LV afterload indicator, LV meridional wall stress (LVmWS) was unchanged during the whole immersed HH exposure at variance with a significant increase during dry exposure.

Pulse pressure after 5 hours was significantly higher during immersed than dry HH. Systolic, mean and diastolic arterial pressures were not different in the immersed and dry sessions.

The changes in the total arterial compliance were similar during the immersed and dry sessions, with values not different from the reference measurement after 15 minutes, but significantly lower after 5 hours.

## DISCUSSION

This work is the first with measurement of hemodynamic parameters during prolonged exposure to both cold water immersion and hyperbaric hyperoxia. We performed an Echocardiography, and Doppler study with a method previously used during experiments in hypobaric or hyperbaric conditions with satisfactory results, (Lafay et *al.*, 1997; Boussuges et *al.*, 2000). In the present study, we report cardiovascular changes during 6 hours of immersed HH. In addition, these results were compared with the observations made in the same subjects during a similar profile of hyperbaric hyperoxia in a dry condition.

### *15 minutes*

In contrast with the dry experiment, after 15 minutes of hyperbaric exposure in cold water immersion LA diameter, **LV dimensions** and stroke volume were unchanged as compared with the reference determination. This evidenced a lack of decrease in LV preload in immersed condition. This may be caused by the water hydrostatic pressure (Lin, 1984). Indeed, the restriction of the systemic vascular capacity triggers a redistribution of some peripheral blood volume into the intrathoracic vascular network, and favours venous return to the heart. Furthermore, the increased hydrostatic pressure applied on the skin triggers an increase in plasma volume via a shift of interstitial fluid through the capillary walls in the portion of the body submerged in water. Transcapillary fluid shift occurs during the first minutes of exposure and leads to an increase in plasma volume and some hemodilution (Greenleaf et *al.*, 1981; Miki et *al.*, 1989; Johanssen et *al.*; 1998). Finally, all studies observe an increase in central blood volume associated with an increase in cardiac diameters and stroke volume during head out water

immersion (Arborelius et al., 1972; Lin, 1984; Christie et al., 1990; Sramek et al., 2000).

In our study, divers were breathing through a SCUBA, leading to increased intrathoracic and intrapleural pressures. In dry conditions, these mechanical breathing strains caused a decrease in LV preload (Molenat et al., 2004). In the present report, the unchanged cardiac dimensions and stroke volume could result from two opposing effects: water immersion which increased central blood volume and ventilation strains of the SCUBA which acted to decrease cardiac preload.

The %FS was not changed during immersed HH exposure, suggesting that the LV contractility did not vary either. Conversely, in dry conditions, an almost 10% reduction of %FS was found. This echocardiographic index is dependent on contractility and both preload and afterload. Since Starling's work, it has been well known that increasing the initial length of the muscle fiber by increasing the heart volume enhances the cardiac contractility. Consequently, in dry condition, the decrease in LV preload led to a reduction in %FS, whereas during water immersion, LV preload was unchanged leading to the stability in %FS.

The systolic, mean and diastolic blood pressure, total arterial compliance and LVmWS were unchanged after 15 minutes of hyperbaric exposure in water immersion. In contrast, under dry conditions, LVmWS increased suggesting an elevation in LV afterload that we attributed to a decrease in cardiac contractility and/or to an arterial vaso-constriction induced by hyperoxia. In thermoneutral water immersion past studies have shown a decrease in systemic vascular resistance and an increase in arterial compliance (Arborelius et al., 1972; Farhi & Linnarsson, 1977; Park et al., 1999; Boussuges, 2006). Consequently, modifications of the systemic arterial circulation induced by immersion could explain that the afterload indices did not increase after 15 min of immersed HH exposure before any adverse effect of cold exposition. Indeed, the

wet suits worn by the subjects are designed for real diving operation and within 15 min rest, their insulating capacity had not been overcome.

At least, echocardiography-Doppler permitted, in our subjects, the assessment of LV filling. In normal young adults, LV elastic recoil is vigorous and myocardial relaxation is swift, so that most LV filling is completed during the early diastole with only a small contribution of filling during atrial contraction. This results in an elevated transmitral E velocity peak, a smaller transmitral A velocity peak and a E/A ratio greater than 1 (Oh et al., 1997), as we observed in our subjects at the sea level point during baseline measures. After 15 minutes, LV filling pattern seemed unchanged. However, this should be analysed in the light of the above results. Indeed, the major determinant of the transmitral flow is the pressure gradient between LA and LV (Oh et al., 1997), depending on LV preload, relaxation and contractility. In our subjects, the decrease in LV preload due to the breathing apparatus should have led to a decreased peak E velocity, an increased peak A velocity and a decreased E/A ratio. However, water immersion, by increasing the LA pressure and LA/LV gradient might have increased the early LV filling and the E/A ratio (Appleton & Hatle, 1992). As a result of those two opposing effects, the LV filling pattern appeared unchanged.

### *5 hours*

During the hyperbaric exposure, divers did not drink or eat. They performed two periods of exercise. In dry conditions, hypovolemia after 5 hours was evidenced by the decrease in stroke volume and in cardiac diameters decrease (Molenat et al., 2004). In contrast with the dry experiment, left ventricular diameters and stroke volume were not reduced after 5 hours of hyperbaric exposure with water immersion. Again, the hydrostatic pressure exerted by immersion provides a likely explanation to this paradox.



Indeed, during immersion, the redistribution of blood volume toward the compliant thoracic bed may conceal a part of hypovolemia (Christie et al., 1990; Norsk et al., 1990; Sramek et al., 2000). Furthermore, as immersion persists, up to 12 hours, the shift of fluid from the interstitial and cellular compartment to the plasma persists (Johansen et al., 1992), and explains the lower reduction in plasma volume during the immersed HH in comparison with dry condition. For this reason, the left ventricular filling profile was also unchanged.

Bradycardia was observed after 5 hours of hyperbaric exposure. Bradycardia could be explained by two factors. In contrast with dry session, hyperoxia-induced bradycardia was not masked by a hypovolemia-induced tachycardia. In addition, the divers progressively experienced the sensation of cold during the resting periods and some went to shivering. Thermal monitoring evidenced a continuous decrease of rectal temperature during these periods. Consequently, at the time of our second echocardiographic examination, cold exposure might have contributed to the heart rate lowering (Sramek et al., 2000). As the result of the unchanged stroke volume and of the lowered heart rate, cardiac output decreased to a similar degree as in dry condition.

An increase in pulse pressure and a decrease in total arterial compliance were observed after 5 hours of immersed HH exposure, suggesting arterial vasoconstriction. These modifications could be attributed both to hyperoxia and to a reaction to peripheral cooling. A similar reduction in total arterial compliance was observed after 5 hours of dry HH exposure but the mechanisms might have been different. **In the dry condition**, vasoconstriction might have been triggered by both hyperoxia and hypovolemia.

In contrast with the **decrease** in total arterial compliance, LVmWS was unchanged after 5 hours of immersed HH exposure. This index is a function of the systolic blood pressure and of the LV dimensions, both of them being unchanged during hyperbaric exposure with water immersion.

In conclusion, hemodynamic changes induced by exposure to the same profile of hyperbaric hyperoxia were quite different in cold water immersion and in dry condition. The relative hypervolemia caused by water immersion concealed the loss of plasma volume, which occurred during the six hours of cold water immersion with SCUBA breathing and intermittent exercise. **The stroke volume assessed non-invasively with a thoracic impedance device by another team during the same experiment, demonstrated a greater decrease post immersion HH compared to the SV changes after the dry condition (Mourot et al., 2004). This modification was supported by the changes in plasma volume observed at the same time after immersion.** While the unchanged indices of cardiac function, and arterial pressure appeared to evidence a maintained efficient plasmatic volume during immersion, the loss of fluids may be as great as or greater, than in **a dry environment**. Finally, the adverse circulatory effects of a prolonged stay in water are unveiled only after emersion.

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Table 1 : Weight and arterial pressure recorded before and after dry and immersed hyperbaric hyperoxia exposures

|             | Dry Hyperbaric Hyperoxia |              | Immersed Hyperbaric Hyperoxia |               |
|-------------|--------------------------|--------------|-------------------------------|---------------|
|             | Before                   | After        | Before                        | After         |
| Weight (kg) | 81.7 +/-11               | 79.4 +/-11 * | 80.6 +/-12                    | 78.2 +/- 11 * |
| SAP (mmHg)  | 120 +/- 8                | 126 +/- 12   | 117 +/- 7                     | 122 +/-8      |
| DAP (mmHg)  | 75 +/- 11                | 70 +/- 10    | 71 +/- 9                      | 73 +/- 9      |



Table 2 : Hemodynamic datas recorded during the two sessions

|                       | Sea level   | Dry Hyperbaric Hyperoxia |                 | Immersed Hyperbaric Hyperoxia |                 |
|-----------------------|-------------|--------------------------|-----------------|-------------------------------|-----------------|
|                       |             | 15 minutes               | 5 hours         | 15 minutes                    | 5 hours         |
| HR (beat per min)     | 62 +/-7     | 69 +/-12                 | 63 +/-11        | 66 +/-9                       | 54 +/-9 * § £   |
| CO (l/min)            | 6.1 +/- 1.1 | 6.3 +/- 0.9              | 4.9 +/- 0.7 * § | 6.3 +/- 0.9                   | 4.8 +/- 0.6 * § |
| SV (ml)               | 98 +/- 15   | 93 +/- 20 *              | 78 +/- 14 * §   | 98 +/- 15                     | 91 +/- 14       |
| Mean BP (mmHg)        | 91 +/- 7    | 93 +/- 13                | 93 +/- 9        | 92 +/- 11                     | 91 +/- 10       |
| Systolic BP (mmHg)    | 124 +/- 9   | 131 +/-15                | 130 +/-9        | 128 +/-18                     | 132 +/-9        |
| Diastolic BP (mmHg)   | 74 +/- 8    | 74 +/- 15                | 75 +/- 10       | 74 +/- 10                     | 71 +/- 13       |
| Pulse Pressure (mmHg) | 50 +/- 10   | 58 +/- 19                | 55 +/- 7        | 55 +/- 15                     | 60 +/- 11 *§ £  |

\* p<0.05 versus reference, § p<0.05 versus 15 min of the same environment, £ p<0.05 versus dry at the same time point

Table 3 : Echocardiographic variables recorded during the two sessions

|  | Sea level     | Dry Hyperbaric Hyperoxia |                 | Immersed Hyperbaric Hyperoxia |                         |
|--|---------------|--------------------------|-----------------|-------------------------------|-------------------------|
|  |               | 15 minutes               | 5 hours         | 15 minutes                    | 5 hours                 |
| <b>Cardiac diameters</b>                           |               |                          |                 |                               |                         |
| LA (mm)  | 36.3 +/- 3.8  | 29.5 +/- 3.0 *           | 28.6 +/- 4.2 *  | 34.6 +/- 2.5 <sup>£</sup>     | 34 +/- 2.4 <sup>£</sup> |
| LVEDD (mm)   | 53.5 +/- 3.5  | 50.9 +/- 3.5 *           | 50.6 +/- 3.7 *  | 52.9 +/- 2.5 <sup>£</sup>     | 52.4 +/- 2              |
| LVESD (mm)   | 34.2 +/- 2.6  | 34.3 +/- 3.6             | 34.6 +/- 2.6    | 34.3 +/- 2.9                  | 34.9 +/- 2              |
| %FS (%)  | 36 +/- 3      | 32 +/- 5 *               | 31 +/- 3 *      | 35 +/- 4 <sup>£</sup>         | 33 +/- 3                |
| <b>LV afterload indices</b>                        |               |                          |                 |                               |                         |
| Total arterial compliance (ml/mmHg)                | 1.97 +/- 0.38 | 1.74 +/- 0.57            | 1.49 +/- 0.30 * | 1.81 +/- 0.52                 | 1.52 +/- 0.34 *         |
| LV meridional wall stress (dynes/cm <sup>2</sup> ) | 69 +/- 11     | 81 +/- 13 *              | 84 +/- 16 *     | 69 +/- 16 <sup>£</sup>        | 69 +/- 7 <sup>£</sup>   |

\* p<0.05 versus reference, § p<0.05 versus 15 min of the same environment, <sup>£</sup> p<0.05 versus dry at the same time point

Table 4 : Left ventricular filling profile recorded during immersed session

|   | <b>Sea level</b> | <b>Immersed</b>   | <b>Hyperbaric Hyperoxia</b> |
|---|------------------|-------------------|-----------------------------|
|   |                  | <b>15 minutes</b> | <b>5 hours</b>              |
| E max velocity (cm/s)                                   | 73 +/- 11        | 69 +/- 6          | 65 +/- 13                   |
| A max velocity (cm/s)                                   | 44 +/- 11        | 38.0 +/- 8        | 41 +/- 11                   |
| E/A   | 1.75 +/- 0.50    | 1.66 +/- 0.44     | 1.51 +/- 0.35               |
| Contribution of atrial contraction<br>to LV filling (%) | 23 +/- 6         | 19 +/- 5          | 21 +/- 5                    |
| PHT (cm/s)  | 57.7 +/- 9.9     | 70 +/- 15         | 75 +/- 28                   |
| IVRT (ms)   | 90. +/- 7        | 93 +/- 10         | 100 +/- 10                  |

Figure 1 : Peak inspiratory and expiratory pressures recorded at different depths in diver using the closed-circuit SCUBA Oxy mixgers 97

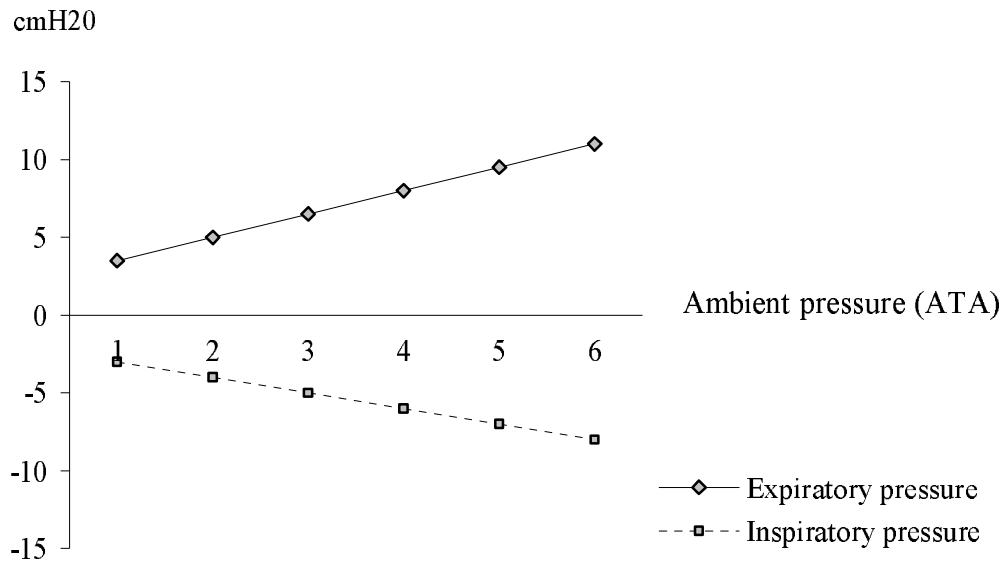
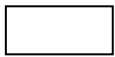


Figure 2 : Evolution of central temperature in the ten subjects, during the immersed hyperbaric hyperoxic exposure

\* significant (compared with sea level - reference)



Echocardiography



Exercise

°C

