Echocardiography in military oxygen divers.

Alain Boussuges, Florence Riera, Pascal Rossi, Olivier Castagna, François Galland, Jean-Eric Blatteau

Research article (ASEM 1996R2)

Abstract : 241 words

Text : Page number : 17, words : 2615, Table number : 1, Figure number : 0

Reference number : 30

ECHOCARDIOGRAPHY IN MILITARY OXYGEN DIVERS

Running head : Heart and oxygen diving

Authors :

Alain BOUSSUGES [1,2CA], MD. PhD, Florence RIERA [1], Pascal ROSSI [2], MD. PhD, Jean-Eric BLATTEAU [3], MD, Olivier CASTAGNA[1], MD, François GALLAND[1], MD

1 – Naval Medical Institute (IMNSSA), Boulevard de Sainte-Anne, Toulon-France

2 - Mediterranean University, E.A. 3280, Laboratoire de Physiopathologie et Action Thérapeutique des Gaz sous Pression, Faculté de Médecine Nord, 13916 Marseille cedex 20, France.

3 – Département de Médecine Hyperbare, HIA Sainte Anne, Toulon Armées

CA : ✉ Dr Boussuges Alain, IMNSSA, BP 610 – 83800 Toulon Armées, France

Phone : (33-4) 94 09 92 67, Fax : (33-4) 94 09 92 51.

Email : alainboussuges@libertysurf.fr , a.boussuges@imnssa.net

ABSTRACT

Background : Oxygen divers undergo environmental stressors such as immersion, ventilation through the SCUBA, cold exposure and increased ambient pressure. All of these stressors may be responsible for acute hemodynamic modifications. We hypothesised that repeated hyperbaric hyperoxia exposure induces long term cardio-vascular modifications.

Methods: A Doppler-echocardiography was conducted on 20 oxygen military divers (average 12 yr diving experience) and compared to 22 controls. Parameters known to be modified by acute hyperoxic exposure such as left ventricular (LV) function (systolic and diastolic) and arterial compliance were analyzed.

Results: Controls and divers were matched appropriately for age and height, although the divers had a higher body mass index and aerobic capacity. Left atrial and left ventricular diameters did not differ between the two groups. On the other hand, left ventricular mass was significantly higher in the elite military divers (209 +/- 43g) in comparison with the control group (172 +/- 48g), even when LV mass was indexed to body surface area. Left ventricular systolic and diastolic function indices, stroke volume, cardiac index, peripheral vascular resistance and systemic compliance were comparable between the two groups.

Conclusion: A greater LV mass was observed in oxygen military divers. The echocardiographic differences between divers and controls could be attributed to the high level physical training undertaken by the military divers. But some stressors such as cold water immersion, repeated hyperoxic exposures, SCUBA breathing and long distance swimming could have participated to the echocardiographic findings in oxygen divers.

Words: 241

Key words: Oxygen toxicity, diving, hyperbaria, hemodyamic status, echocardiography

INTRODUCTION

During exposure to hyperbaric environments such as diving with self contained underwater breathing apparatus (SCUBA) or industrial activities, supplemental oxygen is given to decrease the nitrogen content of the tissues and blood at the time of the decompression, and therefore limit the occurrence of decompression sickness. In the particular case of military divers, the utilisation of breathing gas enriched with oxygen, through a closed-circuit SCUBA, provides longer self-sufficiency (expired gas is re-inhaled, after CO2 has been extracted by lime) and enhanced discretion (absence of bubbles).

During the dive, subjects undergo environmental stressors such as immersion, ventilation through the SCUBA, cold exposure and increased ambient pressure. All of these stressors may be responsible for hemodynamic modifications which have been well studied in healthy volunteers.

Immersion in water induces a cephalad shift of peripheral venous blood that augments central blood volume (12, 8).

Ventilation against resistance induces modifications of intra-thoracic pressure and consequently modifications of cardiac preload and after load (3).

Cerebral and pulmonary oxygen toxicities are well documented and oxygen diving standards have been established to prevent divers from acute oxygen toxicity. On the other hand, several hemodynamic modifications can be induced by acute hyperoxic exposure. Hyperoxia is recognised as a major factor of heart rate decrease observed at high ambient pressure (10, 27,30). Authors have attributed this heart rate decrease to a parasympathetic hyperactivity (14,26). A decrease in cardiac output related to the simultaneous decrease in heart rate and stroke volume is found at high oxygen partial pressure (1,18,20,30). Furthermore, an increase in oxygen partial pressure can induce impairment in cardiac relaxation (14). Finally, systemic vasoconstriction is consistently observed during exposure to hyperoxia (1,10,24,30).

Although hemodynamic modifications during short-term hyperoxic exposure or during open sea diving are well documented, little is known about the long term cardio-vascular consequences of repeated exposures experienced by oxygen divers.

In a previous study, Stuhr et al (28) have shown that professional saturation divers have no morphologic or functional cardiologic changes. However, the hyperoxic exposure of these divers is less than that of oxygen divers.

We hypothesised that repeated hyperbaric hyperoxia exposure induces long term cardio-vascular modifications. A Doppler-echocardiographic study was carried out on a group of elite military oxygen divers and an age-matched control group to determine if oxygen diving has long term consequences on cardio-vascular function.

METHODS

All experimental procedures were conducted in accordance with the Declaration of Helsinki, and were approved by the ethics committee of the University of Marseilles (CCPPRB Marseille 1). Each method and the potential risks were explained to the participants in detail and they gave written informed consent before the experiment. Subjects reported to the laboratory in the early afternoon, 2-3 hours after a light meal. All subjects were non smokers. During the day of the study, they refrained from consuming coffee or alcohol. They have not dived or exercised for at least 48 hours.

Subjects

Twenty trained male military oxygen divers were included in the study. The subjects were all experienced divers with 500-3000 dives (average 12 yr diving experience). None of them had experienced decompression sickness in the past. Their physical activity was assessed by a questionnaire. Body surface area was calculated according to Dubois Formula: $(\text{height in cm})^{0.725} \times (\text{weight in kg})^{0.425} \times 0.00718$. The percentage of body fat and the fat free mass were obtained from skin fold thickness (13).

Twenty two healthy male controls without regular SCUBA diving activity were studied. They were subjected to the same examinations as the military divers.

Measurement of maximal oxygen uptake

In order to assess individual exercise aptitude, each subject performed an incremental treadmill test. Gas exchange was assessed using a breath-by-breath system, which was calibrated before each test. During the exercise test, subjects breathed through a mouthpiece in order to analyze expired gas using breath-by-breath rapid response paramagnetic O_2 and

infrared CO_2 analyzers (Jaeger Oxycon Pro® gas analyser). Exercises were performed as follows: volunteers started running after reaching a steady-state gas exchange condition while standing quietly. After a 6 min warm-up at 8 km/h with an elevation of 2%, speed was increased by 1km/h/min until exhaustion. VO2 Max was defined as the highest value of oxygen uptake despite increased workload.

Echographic and Doppler study

The ultrasonographic examinations were carried out by an experienced investigator (AB) using a commercially available Doppler echocardiograph (Esaote Mylab 30CV,Genova Italy) connected to a 2.5-3.5 MHz transducer array. Investigations were performed in a quiet room with a stable environmental temperature (25°C). Subjects remained at rest for 10 min before the ultrasonographic examination. Heart rate (HR) was recorded by echocardiogram and the rate was averaged over 60 s. Blood pressure was measured by a sphygmomanometer on the right arm after each echographic examination. Pulse pressure (PP) was defined as systolic minus diastolic blood pressure: PP = SAP - SDP.

Examinations were made using two dimensional and M-mode echocardiography associated with pulsed and continuous wave Doppler. Images were obtained via a trans-thoracic approach from the parasternal views (long axis and short axis) and from an apical four chamber view. The subjects were placed in a left lateral position for the parasternal views and in a supine position for the apical four chamber view. Second harmonic imaging was used to improve the image quality. Doppler recordings were performed at the end of normal expiration in order to eliminate the effects of respiration on the parameters studied. Measurements were averaged from at least three consecutive beats. Tape recordings were obtained at a paper-speed of 100mm/s with simultaneous tracing of the electrocardiogram. Examinations were recorded on standard VHS videotape to be reviewed later. Variables known to be modified by acute hyperoxic exposure such as left ventricular (LV) function (systolic and diastolic) and arterial compliance were analyzed.

Left atrial diameter (LA), left ventricle end systolic and end diastolic diameters (LVEDD, LVESD), left ventricle end diastolic interventricular septal thickness (LVEDSep), left ventricle end diastolic posterior wall thickness (LVEDPW), right ventricle end diastolic diameter (RVEDD), and aortic diameter (Ao) were measured by M-mode echocardiography from the left parasternal short and long axis views (25).

Left ventricular mass (LVM) was assessed by the application of Devereux's formula (6):

$LVM = 1.04 \times ((LVEDD + LVEDSEP + LVEDPW)^3 - LVEDD^3) - 13.6$

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 blood flow. Aortic diameter was measured by 2D echocardiography from the left parasternal long axis view at the level of the aortic root.

Aortic cross section area (ACSA) was calculated as $ACSA = 3.14 \times (diameter / 2)^2$. The aortic systolic flow velocity time integral (Ao VTI) was measured by computer-assisted determination using the pulsed wave Doppler profile of aortic blood flow from the apical four chamber view allowing calculation of LV stroke volume (SV = Ao VTI x ACSA) and cardiac output (CO = SV x HR).

Total arterial compliance (Cw) was calculated by dividing Stroke Volume by Pulse Pressure (4) and systemic vascular resistance (SVR) was estimated by dividing Mean Arterial Pressure by cardiac output.

Doppler measurements were averaged over at least three different beats. Transmitral blood 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 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 velocities ratio (E/A) was calculated. The IVRT was the interval from the aortic valve closure signal to the mitral valve opening signal.

RV-RA pressure gradient (RV/RAg) give an estimation of the systolic pulmonary artery pressure (**5**). It was derived from the tricuspid regurgitant flow, identified in continuous wave Doppler from the apical four chamber view. Instantaneous systolic pressure gradient from RV to RA was calculated with the modified Bernouilli equation from the peak velocity of the tricuspid regurgitant signal: $RV/RAg = 4V^2$ (in which V is the maximal regurgitant velocity in m/s).

Statistical analysis

Continuous variables are expressed as mean +/- 1 standard deviation. Differences between divers and controls were analyzed by a Mann-Whitney's U test. The chi-square test was used to determine correlation between the qualitative parameters. Yate's correction was used where small numbers were involved. Differences between groups were considered significant at $p<0.05$. Statistical tests were run on Sigma Stat software.

RESULTS

Military divers and civilian non diver controls were matched appropriately for age (respectively 33 +/- 4 vs. 32 +/- 6 years – p=0.5), height (177 +/-8 vs. 178 +/-5 cm - p=0.3), and body surface area (2 +/- 0.16 vs. 1.9 +/- 0.09 m^2 – p=0.2), but divers weighed more than the control subjects (80 +/- 10 vs. 74 +/- 7 kg – p=0.02).
Moreover, divers had a significantly higher body mass index (25 +/- 2 vs. 23 +/- 2 kg/m^2 - p=0.001) as the result of a non-significant increase in both body fat mass (16 +/- 3 vs. 15 +/-5 % - p=0.9) and fat free mass (66 +/- 6 vs. 64 +/-5 kg - p=0.3).

Military divers performed endurance training and resistance exercise. They engaged in several sport activities such as swimming (about 6 -10 hours per week), running or cycling (about 2-4 hours per week), weigh lifting (about 1-3 hours per week) and combat sports (judo, karate) about 2 to 3 hours per week. Most of the control subjects engaged in physical activity such as distance running (8) cycling (4), swimming (3), tennis (3) or skiing (3). The duration of the sport activities performed by military divers was significantly longer than that of the controls (12 +/- 3 h vs. 4 +/- 2 hours by week – $p<0.001$). There was no significant difference in systemic blood pressure (systolic, diastolic, mean or pulse pressure) and heart rate between the diver and control groups (table I).

Table I here

At peak exercise, maximal values of VO2 (4396 +/- 500 vs. 3651 +/- 600 ml/min p=0.002) and VO2/weight (57 +/- 7 vs. 49 +/- 7 ml/kg/min p=0.007) were significantly higher in military divers compared with controls.

All volunteers (divers and controls) had a normal left ventricular systolic and diastolic function. None of the military divers were found to have a left ventricular internal diameter over 60mm and a maximum wall thickness over 13mm. Left atrial and left ventricular diameters did not differ between the two groups (Table I). Left ventricular mass was significantly higher in the elite military divers, even when LV mass was indexed to BSA. No differences in left ventricular filling parameters were observed between divers and controls. Stroke volume and cardiac index were comparable in divers and controls. Nevertheless, cardiac output was greater in the military diver group without reaching significance. After load indices such as systemic vascular resistances and total arterial compliance were similar in the two groups.

DISCUSSION

Divers and controls were matched appropriately for age and height, but divers had significantly higher body weight and body mass index. This was the result of a non-significant increase in both body fat mass and fat free mass. The specific physical training experienced by the elite military divers could explain the anthropometric differences between the two groups. Indeed, the military divers performed endurance training and muscular strength training, whereas the control subjects performed only endurance type sports. Furthermore, the weekly duration of the physical activity was significantly longer in the military divers than in the controls. This high level physical training resulted in a greater aerobic capacity. Indeed, maximal values of VO2 and VO2/weight were significantly higher in the military divers than in the controls.

The echocardiographic study demonstrated a significant elevation in left ventricular mass in the military divers. This difference was the result of a non-significant increase in both LV diameter and LV wall thickness. These changes in cardiac morphology could be related to the high level of sport endurance training experienced by military divers. Moreover, the long distance swimming experienced by oxygen military divers during their professional activity could participate to the LV modification. However, a variety of cardiovascular diseases could be responsible for an increase in LV mass, and in some highly trained athletes it may be difficult to distinguish between athlete's heart and hypertrophic or dilated cardiomyopathy (16). In our military diver population, systemic hypertension and valvular heart disease were excluded by the medical history, clinical examination and echocardiography. The increase in left ventricular cavity dimension that occurs with training may be regarded as the pathological range over 58mm and thereby resembles dilated cardiomyopathy (22). In the majority of athletes absolute wall thickness is normal or mildly increased (lesser than 12mm). The upper limit of physiological hypertrophy of ventricular walls and internal diameter was reported as 13 mm and 60 mm respectively (9,17,22). In our population, the absence of left ventricular systolic dysfunction is sufficient to exclude dilated cardiomyopathy. Furthermore, none of the military divers were found to have a left ventricular internal diameter over 60mm and a maximum wall thickness above 13mm. Consequently, the LV mass increase observed in military divers could be attributed to their training regimen. The normality of the left ventricular diastolic function was consistent with

this hypothesis (23). In normal young adults, LV elastic recoil is vigorous and myocardial relaxation is swift, so 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 (21), which we observed in all subjects with no differences between controls and military divers. Consequently, although an increase in oxygen partial pressure can induce impairment in cardiac relaxation (15), repeated acute exposures seems to have no negative long-term consequences on diastolic function.

Arterial pressure did not differ between military divers and controls. Systemic vascular resistance and total arterial compliance were comparable between the two groups. We have previously demonstrated that acute hyperoxic exposure leads to an arterial vasoconstriction and a decrease in arterial compliance in healthy subjects (24). Consequently, although the vasoconstrictor effect of acute hyperoxia is well recognized, there are no permanent vasomotor changes induced by repeated hyperoxic exposures. A relationship between arterial stiffness and fitness level has been described (11,29), and endurance trained athletes have greater arterial compliance compared with age-matched sedentary controls. In a recent study, Edwards et al (7) showed that wave reflection and systolic load were lower in a competitive endurance trained group in comparison with recreationally active subjects. In the present study, military divers exhibited a higher level of aerobic performance compared with the controls. Therefore, a greater degree of arterial compliance could be expected in this group. Although the two groups performed aerobic exercise training, the military divers followed a regular resistance training program which is recognized to reduce the proximal aortic compliance (2). This specific training could explain the fact that although the military divers had a greater aerobic capacity than did the controls, there was no difference in arterial compliance between the two groups. But an alternate hypothesis could be considered. Repeated hyperbaric hyperoxia exposures may have counteracted any fitness related increase in arterial compliance in the diver group.

In this study, the echocardiographic differences between divers and controls could be attributed to the high level physical training experienced by the military divers. But some stressors experienced by oxygen divers such as cold water immersion, repeated hyperoxic exposures, SCUBA breathing and long distance swimming could have participate to the echocardiographic findings in oxygen divers. On the other hand, some modification could be masked by the high level of exercise training. Further studies, including a longitudinal long-term study, should be considered to exclude hyperoxia-induced long term cardiovascular

alterations. Furthermore, investigations in a less physical trained group such as recreational oxygen divers would be of interest.

Acknowledgement: The authors gratefully acknowledge the volunteers and the medical and technical board of the CEPHISMER center, Toulon, France. This work was supported by a research DGA grant (PEA n°98 08 09).

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Table I : Hemodynamic data and Echocardiographic results in divers and controls

	Divers	**Controls**	***p* value**
Hemodynamic datas			
SBP (mmHg)	126 +/- 9	122 +/- 8	0.36
MBP (mmHg)	90 +/- 6	89 +/- 7	0.46
DBP (mmHg)	69 +/- 7	71 +/- 8	0.78
PP (mmHg)	55 +/- 10	51 +/- 8	0.18
HR (beats/min)	63 +/- 10	61 +/- 9	0.67
SV (ml)	93 +/- 12	92 +/- 15	0.43
CO (l/min)	6 +/- 1.1	5.6 +/- 1.1	0.12
CI (l/min/m^2)	3 +/-0.5	2.9 +/- 0.5	0.21
SVR (dyne.s.cm^{-5})	1311+/- 271	1308 +/- 279	0.93
Cw (ml/mmHg)	1.9 +/- 0.37	1.8 +/- 0.4	0.31
Left cardiac dimensions			
LA (mm)	35 +/- 4	34 +/- 2	0.18
Ao (mm)	31 +/- 3	30 +/- 4	0.89
LVEDD (mm)	54 +/- 3	53 +/- 2	0.15
LVESD (mm)	34 +/- 2.5	35 +/- 3	0.17
LVEDSep (mm)	9.2 +/- 1.4	8.3 +/- 1.9	0.31
LVEDPW (mm)	9 +/- 1.2	8,1 +/- 1.6	0.19
%FS (%)	35 +/- 3	33 +/- 3	0.11
LVM (g)	209 +/- 43	172 +/- 48	0.01
LVM/BSA (g/m^2)	107 +/- 22	90 +/- 23	0.03
Left ventricular filling profile			
E (cm/s)	80 +/- 13	83 +/- 18	0.65
A (cm/s)	49 +/- 11	46 +/- 9	0.43
E/A ratio	1.7 +/- 0.4	1.8 +/- 0.6	0.41
IVRT (ms)	80 +/- 10	77 +/- 9	0.56
Right heart study			
RVEDD (mm)	23 +/- 3	23 +/- 4	0.39
RV/RAg (mmHg)	20 +/-2	20 +/-3	0.74

SBP : Systolic Blood Pressure, MBP : Mean Blood pressure, DBP : Diastolic Blood Pressure, PP : Pulse Pressure, HR : Heart rate, SV : Stroke volume, CO : Cardiac output, CI : Cardiac index, SVR : Systemic vascular resistance, Cw : Total arterial compliance

LA : Left atrium, Ao : Aortic diameter, LVEDD : Left ventricular end diastolic diameter, LVESD : Left ventricular end systolic diameter, LVEDSep : left ventricle end diastolic interventricular septal thickness, LVEDPW : left ventricle end diastolic posterior wall thickness, %FS : LV percent fractional shortening, LVM : Left ventricular mass, BSA : Body surface area, E : Peak velocity of the E wave, A: Peak velocity of the A wave, IVRT : Isovolumic relaxation time, RVEDD : Right ventricle end diastolic diameter, RV-RAg : Instantaneous systolic pressure gradient from right ventricle to right atrium