Cardiac function during breath-hold diving in humans: An echocardiographic study.

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1C.N.R Institute of Clinical Physiology, Pisa; 2Scuola Superiore S. Anna, Pisa; 3U.O. Cardiovascolare – UTIC Ospedale di Cecina (Livorno); 4Apnea Academy Research – ITALY

Marabotti C, Belardinelli A, L’Abbate A, Scalzini A, Chiesa F, Cialoni D, Passera M, Bedini R. Cardiac function during breath-hold diving in humans. An echocardiographic study. Undersea Hyperb Med 2008; 35(2):83-90. Breath-hold diving induces, in marine mammals, a reduction of cardiac output due to a decrease of both heart rate and stroke volume. Cardiovascular changes in humans during breath-hold diving are only partially known due to the technical difficulty of studying fully immersed subjects. Recently, a submersible echocardiograph has been developed, allowing a feasible assessment of cardiac anatomy and function of subjects during diving. Aim of the study was to evaluate, by Doppler-echocardiography, the cardiovascular changes induced by breath-hold diving in humans. Ten male subjects were studied by Doppler echocardiography in dry conditions and during breath-hold diving at 3m depth. In addition 14 male subjects were studied, using the same protocol, before and during breath-hold diving at 10m depth. At 3m depth significant reductions in heart rate (-17%), stroke volume (-17%), cardiac output (-29%), left atrial dimensions, and deceleration time of early diastolic transmitral flow (DTE) were observed. At 10m depth similar but more pronounced changes occurred. In particular, increase in early transmitral flow velocity became significant (+33%), while DTE decreased by 34%. At both depths dimensions of right cardiac chambers remained unchanged. Breath-hold diving at shallow depth induced, in humans, cardiovascular changes qualitatively similar to those observed in natural divers such as seals. The reduced dimensions of left atrium associated to a left ventricular diastolic pattern resembling that of restrictive/constrictive heart disease, suggest that the hemodynamic effects of diving could be explained, at least in part, by a constriction exerted on the heart by the reduced chest volume and the increased blood content of the lungs. Finally, the absence of dimensional changes in the right chambers suggests that most of the pulmonary blood shift occurred before cardiac imaging.

INTRODUCTION

Breath-hold diving is associated with complex cardiovascular changes as described in free diving instrumented marine mammals (1,2). The study of cardiovascular response to diving in humans has been hampered by the lack of technology suitable for measuring cardiac anatomical and functional parameters of fully immersed subjects. Thus most of the knowledge on human diving physiology derives either from the study of head-out immersed subjects (3,4) or extrapolated from the results obtained in subjects during breath-hold either with or without face immersion (5,6). Doppler-echocardiography, allowing a real-time assessment of cardiac anatomy and function (both systolic and diastolic), may represent a feasible way of studying cardiac response to diving in humans. Therefore, a submersible Doppler-2D-echocardiographic machine has been recently developed at the CNR Institute of Clinical Physiology in Pisa.
by some of the Authors, making possible to investigate cardiac anatomic and functional changes during free diving.

The aim of the study was to evaluate by Doppler-2D-echocardiography the cardiovascular response to breath-hold diving in humans at two different depths (at 3 and 10 meters).

MATERIALS AND METHODS

We observed by Doppler-2D-echocardiography two groups of subjects during their usual training activity of breath-hold diving. The protocol of the observational study was approved by the Scientific Committee of the CNR Institute of Clinical Physiology. All participants were informed about the aims and procedures of underwater ultrasound examination and gave their written consent.

Subjects
Two groups of healthy subjects were studied during breath-hold diving at 3 and 10 meters depth respectively. The first group (3 meters) consisted of 10 male subjects (age 32±8, range 22-47 years); the second group (10 meters) of 14 male subjects (age 37±5, range 28-47 years). The absence of female subjects was casual and not due to sample selection. All subjects were experienced active breath-hold divers (affiliated to Apnea Academy, Italian School for Education and Research of Freediving), undergoing at least 2 hours/week of breath-hold diving training; each diver had the ability of reaching a depth of at least 30m under constant weight. No subject had history or clinical evidence of arterial hypertension, cardiac or pulmonary diseases. Resting electrocardiogram and Doppler-echocardiographic examination were normal. All subjects were non-smokers and had been fasting from at least 2 hours before the study.

Underwater echocardiographic equipment
Doppler-echocardiographic examination was performed by a commercially available instrument (Caris, Esaote SPA, Florence – Italy) as part of a submersible echograph equipped with a special patented “diving-suit” (7) made by two steel cylinders (60 cm of diameter) intersecting each other (Figure 1). A rubber glove sealed to the front Plexiglas panel allowed to access the instrument’s control keys. A pressure regulator connected to a standard 200 ATM compressed air cylinder (normally used for SCUBA diving) maintained the pressure inside the diving-suit of echocardiographic machine at the same level of external pressure, reducing the risk of water leakage and preventing rubber glove inflation.

Breath-hold dives
Dives at the shallowest depth (3 meters) were performed in a pool (water temperature 29 °C) while 10 meters dives were performed at sea (water temperature 27°C). Both sessions were recorded between 10 AM and 1 PM. Each dive was preceded by 2-4 minutes of surface floating preparation. Descents were done by the use of variable weight (10 Kg ballast), thus reducing the cardiovascular effects of muscular work. As soon as the diver reached the echo station, he positioned himself on a metallic bracket, lying on his left side (Figure 1). Cardiac imaging started during the first minute of apnea and was completed in less than 90 seconds in all subjects.

Doppler-echocardiographic parameters
Subjects were evaluated, by Doppler-echocardiography, in basal conditions (before entering water, during normal breathing, on left lateral decubitus) and during a breath-hold dive. To minimize the duration of the echocardiographic study during diving, we...
recorded only an apical four-chamber view loop (4 seconds duration) and a pulsed-wave Doppler tracing of transmitral blood flow. Measurements were made off-line by a physician expert in Doppler-echocardiography that was unaware of the identity of the subjects and of the condition of recording. From 2D four-chamber view, the following parameters were obtained: systolic and diastolic left ventricular volumes (calculated by area-length method) (8), right ventricular internal dimension (maximal diastolic distance from right-side interventricular septum to right ventricular free wall). Maximal transversal (from interatrial septum to the opposite atrial wall) and supero-inferior (from the plane of atrio-ventricular valve annulus to the opposite wall) dimensions were calculated for both right and left atrium during ventricular systole. Early (E) and late (A) peak transmitral diastolic flow velocities as well as deceleration time of E velocity (DTE) were obtained from pulsed-wave Doppler tracings, by sampling blood velocities at the level of mitral valve tips. These indices allows the characterization of left ventricular diastolic function, as different filling patterns have been described in case of delayed ventricular relaxation (as in early hypertensive heart disease or during aging) and in situations of increased wall stiffness (as in advanced hypertensive heart disease or in constrictive/restrictive heart diseases) (9). Heart rate was obtained from the time interval between two consecutive mitral A peaks (60/A-A interval expressed in seconds); the average calculated on three consecutive cardiac cycles was considered. Left ventricular stroke volume was calculated as the difference between diastolic and systolic left ventricular volumes. Cardiac output was obtained as the product of stroke volume and heart rate.
Statistical analysis
Differences between diving and basal conditions were assessed by Student’s “t” test for paired data. Differences between cardiac changes induced by diving at the two different depths were evaluated by Student’s “t” test for unpaired data. A probability lower than 5% was assumed as threshold to reject the null hypothesis.

RESULTS

Cardiovascular changes during 3 m depth diving
Breath-hold diving at 3 m depth induced, as compared to surface, dry measures, a reduction in heart rate (74±10 vs 60±12 bpm). A reduction of both left ventricular stroke volume and calculated cardiac output was observed during diving (see Table 1) while left ventricular volumes did not show any significant change. Transversal left atrial dimension also showed a significant reduction during diving (36.6±4.2 vs 32.4±4.9 mm). As left ventricular filling concerned, we observed a non-significant increase in E peak velocity yet a significant decrease in DTE (227.0±23.0 vs. 180.9±17.8 ms). No significant changes were recorded either in right ventricular or right atrial dimensions.

Cardiovascular changes during 10m depth diving
A qualitatively similar pattern was observed during breath-hold dive at the greatest depth. Percent changes occurring at the two different depths (as compared to dry values) did not result significantly different (Figure 2).

Fig. 2. Mean percent changes of anatomical and functional cardiac parameters during breath-hold diving at different depths (comparisons between dry and diving conditions).

As reported in Table 1, heart rate decreased during diving (71±11 vs 57±18 bpm); as did left ventricular stroke volume and cardiac output; supero-inferior left atrial dimension resulted significantly reduced during diving (46.7±4.3 vs 42.6±6.4 mm), while left ventricular volumes remained unchanged. Finally, a significant increase in left ventricular early diastolic peak

| Table 1: Anatomical and functional cardiac parameters before and during breath-hold diving at 3 and 10 m depth |
|-------------|--------|----------------|--------|----------------|
|             | 3m     | 10m            |
|             | Basal  | Dive           | Basal  | Dive           |
| Heart rate [bpm] | 73.8 ± 9.6 | 60.5 ± 12.4*  | 71.0 ± 10.7 | 56.7 ± 17.8*  |
| LA trasversal dimension [mm] | 36.6 ± 4.2 | 32.4 ± 4.90‡ | 37.1 ± 4.1 | 37.1 ± 6.4 |
| LA supero-inferior dimension [mm] | 44.3 ± 2.4 | 43.6 ± 2.0 | 46.7 ± 4.3 | 42.6 ± 6.4* |
| Diastolic LV Volume [ml] | 136.3 ± 27.4 | 127.7 ± 11.5 | 158.6 ± 32.6 | 142.4 ± 36.8 |
| Systolic LV Volume [ml] | 69.1 ± 24.5 | 68.7 ± 4.4 | 75.9 ± 23.1 | 73.4 ± 22.2 |
| LV Stroke Volume [ml] | 67.5 ± 18.9 | 58.7 ± 10.2* | 82.7 ± 25.2 | 69.0 ± 23.6* |
| Cardiac Output [l/min] | 5.2 ± 1.85 | 3.7 ± 1.23* | 5.8 ± 2.17 | 3.7 ± 1.10† |
| E peak [cm/s] | 68.2 ± 15.3 | 79.4 ± 30.1 | 70.9 ± 12.8 | 87.1 ± 8.1† |
| Deceleration Time of E [ms] | 227.0 ± 23.0 | 180.9 ± 17.8† | 240.6 ± 43.6 | 159.2 ± 30.0‡ |

Data are average ± SD; LV = left ventricular; LA = left atrial. *P < 0.05; †P < 0.01; ‡P < 0.001 as compared to pre-dive.
filling velocity $E$ (70.9±12.8 vs 87.1±8.1 cm/s) and a significant decrease of DTE (240.6±43.6 vs. 159.2±30.0 ms) were observed. Even at 10m depth diving no significant changes were observed in the dimensions of both right cardiac chambers.

Figure 3 shows, as an example, images obtained on one free diver, in basal conditions and at 3m depth.

**DISCUSSION**

The present study is, at the best of our knowledge, the first dynamic cardiac imaging study in humans during breath-hold diving. The most relevant finding during free-diving was the reduction in cardiac output secondary to the reduction both in heart rate and left ventricular stroke volume. This response has been previously reported as the typical cardiovascular adaptation to diving in natural divers (mostly marine mammals) (1,10) as well as in instrumented dogs during voluntary head immersion (11). Similar effects have been also reported during evocation of the diving reflex by breath holding and face immersion (12) being the response more pronounced with face immersion into cool water (13). Conversely, studies on head-out immersed subjects (no breath holding) showed opposite results being cardiac output increased (14-16) according to increased stroke volume and practically unchanged heart rate. In this condition, increase in stroke volume has been attributed to enhanced venous return to the heart. Actually, studies simulating breath hold diving in a wet pressure chamber (17,18) did not show any significant change in cardiac output with the exception of a study on three elite apnea divers, during simulated deep breath-hold diving in cool water at 40-55 meters depth, which showed a bradycardia-mediated reduction in cardiac output (19). Thus from these studies intensive training and/or special diving conditions (cool water, deep diving) seem essential to induce a “typical” diving response in humans. By contrast, results from the present study document that a diving response, substantially similar to the one observed in natural divers, may be observed also in moderately trained divers during immersions at shallow depths. An oxygen sparing strategy, possibly triggered by a phylogenetical reflex, may still be present in humans (20-22) as in other terrestrial animals during diving (11).

From a physiological point of view, the reduction of left ventricular stroke volume could be ascribed to a decreased preload, to an increased afterload and/or to a reduced contractility. Previous studies on animals
actually reported the occurrence during breath-hold diving of all these haemodynamic changes (11,23,24). It has been reported in simulated breath-hold diving in humans that levels of lung inflation and intrathoracic pressure influence the circulatory effects of diving. Large lung volumes -typical of breath-hold diving- may hamper venous return, thus contributing to the reduction of cardiac output (17). In our study, the significantly higher E (early diastolic) transmitral peak velocity during diving, associated with a significant decrease in the deceleration time of E velocity and decreased or unchanged left ventricular volume, resembles the typical Doppler pattern of restrictive/constrictive cardiac diseases (9).

On this basis, it can be speculated that the reduction of left ventricular stroke volume, one of the two hemodynamic components of the classical diving response beside bradycardia, could be the result of an impaired left ventricular diastolic filling secondary to an “ab-extrinseco” compression (due to the combined effects of a reduced chest volume and an increased blood content of the lungs), in association with a decreased left venous return due to elevated intrathoracic pressure and pulmonary blood-pooling. This interpretation contrasts the commonly reported explanation of reduced stroke volume due to increased left ventricular afterload and/or reduced contractility. Although these two variables were not measured in the present study, in either case, left cardiac chambers are expected to become enlarged, rather than restrained as observed in our study. It may be also speculated that such an impediment to left ventricular filling could contribute, similarly to restrictive/constrictive cardiomyopathy, to the development of acute pulmonary edema in breath-hold divers.

Unfortunately, the methodology adopted in the present study did not allow the appreciation of the intrathoracic blood volume shift already reported as the consequence of both increased tone and loss of gravitational effect on peripheral venous circulation during immersion (25,26). The absence of any significant enlargement of right heart chambers in our study might suggest that, at the time of echo imaging during breath-hold diving, blood redistribution had already occurred during the pre-diving flotation and/or the descent. The transient increase in right atrial dimensions, previously reported in head-out immersed subjects (27), seems to be in accordance with this hypothesis.

As concerns the possible limitations of this report, we acknowledge that the protocol of the present study, comparing data obtained before the subjects entered water with those collected during diving, does not allow to discriminate between the effects of body immersion and those of increased environmental pressure induced by diving.

Moreover, studying two different samples of divers at the two different depths may introduce some confounding factor, due to interindividual variability in adaptation to dive. On the other hand, a study finalized to describe the cardiovascular response to diving needs a group of well-trained divers, not easy to be found. As previously stated, the present data come by the observations of regular training sessions occurring during periodical stages, attended by different groups of free-diving athletes.

In conclusion, the present study is the first report on dynamic imaging of the heart during breath-hold diving in human beings. It documents at 3 and 10 m depth hemodynamic changes that mimic the diving response reported in marine mammals, at the same time providing new information on cardiac volumes and intracardiac flows that might contribute to the understanding of diving physiology.
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