Left ventricle changes early after breath-holding in deep water in elite apnea divers

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ABSTRACT

Purpose: To study by ultrasounds cardiac morphology and function early after breath-hold diving in deep water in elite athletes.

Methods: Fifteen healthy male divers (age 28±3 years) were studied using doppler-echocardiography, immediately before (basal condition, BC) and two minutes after breath-hold diving (40 meters, acute post-apnea condition, APAC). Each subject performed a series of three consecutive breath-hold dives (20 – 30 and 40 m depth).

Results: End-diastolic left ventricular (LV) diameter (EDD) and end-diastolic LV volume (EDV) increased significantly (p<0.01). Stroke volume (SV), cardiac index (CI), septal and posterior systolic wall-thickening (SWT) also significantly increased after diving (p<0.01). No wall motion abnormalities were detected, and wall motion score index was unchanged between BC and APAC. Doppler mitral E wave increased significantly (p<0.01), whereas the A wave was unchanged. Systemic vascular resistance (SVR) decreased significantly after diving (p<0.05). In the factor analysis, filtering out the absolute values smaller than 0.7 in the loading matrix, it resulted that factor I consists of EDV, posterior SWT, SV and CI, factor II of diastolic blood pressure, waves A and E and factor III of heart rate and SVR.

Conclusions: Systo-diastolic functions were improved in the early period after deep breath-hold diving due to favorable changes in loading conditions relative to pre-diving, namely the recruitment of left ventricular preload reserve and the reduction in afterload.

INTRODUCTION

The human cardiovascular response to breath-hold diving has been documented in different experimental models, including deep or shallow diving, simulated diving in a hyperbaric chamber, with and without face immersion (2,3,9,25). The response is similar but less intense than in aquatic mammals and consists of bradycardia, decreased cardiac output and increased peripheral vascular resistance (4,26). It is the result of complex and integrated reactions, acting synergically or in contrast, evoked by the activation of the sympato-vagal system and by hypoxemia and hypercapnia (7,12,5,17). Studies of cardiac output during breath-hold diving in humans offer conflicting results, likely depending on the different experimental conditions, i.e., hyperbaric chamber, sea diving, head-out immersion or prolonged in-air apnea, diving depth, environmental temperature as well as enrollment of elite athletes.

Head-out immersed subjects without breath-holding showed an increased cardiac output according to increased stroke volume and practically unchanged heart rate (24,27). On the
contrary, studies on breath-hold diving performed in a wet pressure chamber did not show any significant change in cardiac output, with the exception of a study of three elite apnea divers, during simulated deep breath-hold diving in cool water at a depth of 40-55 meters, which showed a bradycardia-mediated decrease in cardiac output (8,11).

During breath-hold diving at 3 and 10 meters’ depth the direct assessment of cardiac function by means of submersible Doppler-2D-echocardiography showed significant reduction of cardiac output, due to a concomitant reduction in heart rate and left ventricular stroke volume (19). Ten to fifteen minutes following emersion from a deep diving, Frassi et al., have shown the complete reversion of left ventricular morphology and systo-diastolic function to pre-diving conditions but the presence of water accumulation in the lungs (14).

At present, the time course and modalities of cardiovascular readjustments early after emersion are still unknown. Therefore, in a natural scenario, we studied the cardiovascular changes in elite athletes as early as possible after breath-hold diving (40 meters) in deep water. To accomplish this plan, we assessed volumes as well as regional and global function of LV by 2D Doppler echocardiogram performed on the boat supporting the diving athletes.

METHODS
Subjects
Fifteen healthy male divers (age 28±3 years) were studied using Doppler echocardiography, immediately before (basal condition, BC) and after a regular training session of breath-hold diving (acute post-apnea condition, APAC). The absence of female subjects was by chance and not due to preliminary sample selection.

All subjects were experienced active breath-hold divers (affiliated with the Apnea Academy, Italian School for Education and Research of Free-diving), undergoing at least two hours per week of breath-hold diving training. Each diver had the ability to reach a depth of at least 40m under constant weight. No subject had a history or clinical evidence of arterial hypertension, cardiac, pulmonary or metabolic diseases. Resting electrocardiogram and Doppler echocardiographic examinations were normal. All subjects were non-smokers and had been fasting for at least two hours before the study. The Scientific Committee of the Institute of Clinical Physiology, National Council of Research, approved the protocol of this observational study. All participants were informed about the aims and procedures of the study protocol and gave their written consent.

Breath-hold dives
Dive sessions were performed at Sharm El Sheikh sea between 10 a.m. and 1 p.m.; water temperature was 29°C. Each subject performed a series of three consecutive breath-hold dives (20, 30 and 40 meters’ depth, respectively). Descents were made by the use of variable weight (10 kg ballast), thus reducing the cardiovascular effects of muscular work.

The first dive was preceded by three to five minutes of surface-floating preparation; and a surface recovery of 10 minutes separated the following dives so that the whole protocol lasted 30 minutes. In order to minimize the latency between emersion and ultrasound assessment, the post-diving scan was performed on the boat supporting divers during the contest (latency less than two minutes after the emersion). Figure 1 (facing page) represents the time sequence of the echocardiographic analysis and reports some field images.

Doppler-echocardiographic study
All ultrasound scans were performed with the cardiac probe (2.5-3.5 MHz) from Sonosite Sono Heart Elite Model (Bothell, WA 98021-3904 USA). All images were digitized, transmitted to a PC and stored on DVD for post processing. To minimize variability, a single independent observer, board-certified by the European Association of Echocardiography, read the digitized images and was blinded to both subject identity and status (pre- or post-dive). End-diastolic and end-systolic LV diameters (EDD, ESD, mm, respectively), end-diastolic and end-systolic wall thickness of the interventricular sep
DOPPLER ECHOCARDIOGRAPHY IN BASAL CONDITION

THREE SUBSEQUENT VARIABLE WEIGHT DIVES:

-20 m
10 minutes of latency
-30 m
10 minutes of latency
-40 m

DOPPLER ECHOCARDIOGRAPHY EARLY AFTER 40 m DIVE
tum and of the posterior wall were measured from the M-mode trace obtained by parasternal long-axis view. Percent regional systolic wall-thickening (PSWT, %) was calculated at the level of the interventricular septum and posterior wall in parasternal long axis view according to the following formula:

$$PSWT\, (\%) = \frac{\text{end-systolic thickness} - \text{end-diastolic thickness}}{\text{end-systolic thickness}} \times 100$$

Left ventricular volumes (end-diastolic and end-systolic volume, EDV and ESV, in ml, respectively) were obtained by Teicholz's formula. LV indexes of global LV function were: stroke volume (SV, in ml) derived as EDV-ESV, cardiac output (CO, SV x heart rate, in ml and bpm, respectively); cardiac index (CI, CO normalized with respect to the body surface index, in ml/(min*m²)).

Diastolic function was determined from the pattern of mitral flow velocity by pulsed Doppler echocardiography: early (E, in cm/sec) and late (A, in cm/sec) peak transmitral diastolic flow velocities. More accurate diastolic measurements (i.e., tissue Doppler or pulmonary venous flow velocity) were not available due to measurement difficulties related to logistic reasons.

All measurements were performed following the recommendations of the American Society of Echocardiography (15). Arterial blood pressure was measured by a conventional cuff sphygmomanometer. The hemodynamic variables were: heart rate (HR, bpm), systolic, diastolic and mean blood pressure (SBP, DBP, MBP, in mmHg) and systemic vascular resistance (SVR, in mmHg and min/ml) calculated as the MBP/CO ratio.

Statistical analysis
Paired T-tests have been performed to test the hypothesis of no difference between the acute post-apnea and the basal measurements. Significance level for the hypothesis rejection was fixed at 99%, and the Bonferroni correction for multiple comparisons was applied. To avoid spurious effects due to anomalies of distribution, Wilcoxon’s signed rank tests were performed, and results overlapped those obtained by T-test.

In addition to the univariate analysis, a factor analysis (maximum likelihood factoring) (23) was used to uncover the latent structure of the set of variables. Namely factor analysis was used to identify groups of inter-related variables, each of them describing a pattern or response of a latent physiological mechanism of adaptation. Factor analysis models each observed variable as a linear combination of the factors, plus an “error” term. In detail, factor analysis permits moving from a large number of variables to a smaller number of factors able to model the variance in the raw data as

$$x = \mu + \Lambda f + \varepsilon$$

where $x$ is a vector of observed variables, $\mu$ is a constant vector of means, $\Lambda$ is a constant matrix named factor loadings, $f$ is a vector of independent, standardized common factors, and $\varepsilon$ is the error term vector.

Absolute high values in the loading matrix express an interrelation among the corresponding variables: an usually stringent high threshold for the absolute values of the loading coefficients is 0.7.

The number of factors in the model was determined by the Kaiser criterion (all components with eigen-values under 1.0 dropped out) and validated comparing the correlation matrix of the raw data (experimental one) with that of original items, which would result on the assumption that the computed factors were the true and only factors (estimated one). For this reason, a likelihood ratio statistic test was used to verify whether the number of factors hinted by the Kaiser criterion could explain the data. The significance of the goodness-of-fit test was fixed to 95%.

To facilitate the factors interpretation on the basis of the loadings, rotation methods have been employed. In particular, varimax rotation was used. Using varimax rotation, each factor tended to have either large or small loadings of any particular variable, and this behavior helps in pointing out the variables related to each factor. In addition, oblique rotation (promax) has been examined to allow the factors to eventually be correlated.
**TABLE 1**

<table>
<thead>
<tr>
<th>variables</th>
<th>Conditions</th>
<th>Paired variations</th>
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<tbody>
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<td>APAC</td>
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<tr>
<td></td>
<td>means</td>
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</tr>
<tr>
<td>SBP</td>
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<tr>
<td>DBP</td>
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<tr>
<td>HR</td>
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<td>EDD</td>
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<tr>
<td>EDV</td>
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<td>ESV</td>
<td>24.7±5</td>
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<td>Septal SWT</td>
<td>27.3±5</td>
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<td>Posterior SWT</td>
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<td>SV</td>
<td>40.7±8</td>
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<tr>
<td>CI</td>
<td>1474.7±343</td>
<td>2073.7±455.1</td>
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<td>SVR</td>
<td>1.7±0.4</td>
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<tr>
<td>E wave</td>
<td>66.2±10</td>
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<tr>
<td>A wave</td>
<td>46.3±8</td>
<td>45.1±7.7</td>
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**RESULTS**

LV morphology and function was evaluated in 15 elite freedivers. All subjects performed the diving protocol without cardiovascular or neurological complications. The mean apnea time duration was 90 ± 18 s (dive at 40 meters). Post-apnea echocardiogram was performed in all athletes within 1.45 ± 0.30 minutes after deep diving. In Table 1 (above), BC and APAC descriptive statistics and T-test comparisons of the obtained variables are summarized.

As compared to pre-dive, EDD and EDV increased significantly (p<0.01), whereas ESD and ESV were unchanged after diving. SV, CI, septal and posterior SWT, also significantly increased after diving. No wall motion abnormalities were detected, and WMSI was unchanged between BC and APAC. Regarding diastolic function, only mitral E wave increased significantly (p<0.01), whereas the A wave was unchanged in APAC compared to BC. Out of the hemodynamic variables, statistical analysis yielded a slight significant reduction only for SVR after diving (p<0.05). Factor analysis seems to unveil a latent structure of the data since three latent factors were sufficient to model the dataset of the paired variations (p<0.05).

Irrespective of the rotation used (varimax or promax), the analysis of the loading matrix,
TABLE 2

<table>
<thead>
<tr>
<th>variables</th>
<th>Factors</th>
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<tr>
<td>SBP</td>
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<td>HR</td>
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<td>A wave</td>
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Table 2 shows the factors composition and the sign with which each variable contributes to the factor.

**TABLE 2.** Factorial analysis identified 3 sets (factors) of linked variables.

**Factor I:** End-diastolic volume (EDV), posterior systolic wall-thickning (SWT), stroke volume (SV), cardiac index (CI).

**Factor II:** Diastolic blood pressure (DBP) and E and A waves.

**Factor III:** Systolic blood pressure (SBP), heart rate (HR) and systemic vascular resistances (SVR).

Other variables not included in factors are end-systolic volumes (ESV) and septal systolic wall-thickning (SWS). Signs indicate the contribution to the factor of each variable.

expressing the interrelations among variables, showed three sets of linked variables. Each resulting factor was largely independent from the others. Filtering out the absolute values smaller than 0.7 in the loading matrix, it resulted that EDV, posterior SWT, SV and CI constitute Factor I, DBP, waves A and E Factor II, and HR and SVR Factor III. Table 2 (above) shows the factors composition and the sign with which each variable contributes to the factor.

**DISCUSSION**

The findings of our study showed that 40-meter-deep breath-hold diving induced significant changes in LV morphology and function which were still present two minutes following emersion. In particular, relative to pre-dive, end-diastolic left ventricular volume was enlarged, global systolic and diastolic function was enhanced, no alterations were present in regional wall motion.

Regarding hemodynamic measures, 40-meter-deep breath-hold diving evoked only a significant reduction in SVR. The factorial analysis we performed suggests the presence of three latent independent mechanisms, coordinating the cardiovascular changes. Considering each factor in terms...
of involved variables and their ongoing function, it seems that all of them contribute to the modulation of cardiac pre-load and after-load, namely increased pre-load and reduced after-load. Thus the heart improves its systolic and diastolic function through appropriate changes in loading conditions.

Considering Factor I, the Frank-Starling mechanism (increased myocardial contractility secondary to increased EDV) might be responsible for the improved cardiac performance both at a global level, as shown by increased SV and CI, and at the regional one, as shown by the increased posterior SWT (6). The augmented ventricular diastolic filling might be related to central-to-peripheral blood shift during dive-ascent. In fact, in the descending phase of breath-hold diving a peripheral-to-central blood flow redistribution occurs with increased carotid blood flow and concomitant peripheral vasoconstriction (1,20), while in the ascending phase, as well as in early phase after emersion, the reversal occurs — i.e., central-to-peripheral blood flow redistribution.

In a previous study Marabotti et al. (19), using a submersible Doppler 2D echocardiography during breath-hold diving at 10 meters underwater, showed a decrease in cardiac output due to a concomitant decrease in both heart rate and left ventricular stroke volume.

Combining the results of the underwater study of Marabotti (19) with the results of the present study we hypothesized the following scenario:

- during breath-hold diving, blood is compartmentalized into the lungs as consequence of air volume reduction (pulmonary blood shift); consequently, left ventricular filling decreases as well as left ventricular EDV and stroke volume (19);

- subsequently, while ascending, pulmonary gases re-expand because of external pressure lowering, and pulmonary blood is pushed ahead into the systemic circulation. This may favor a counter blood shift from the central circulation to the periphery, thus increasing left ventricular filling and enhancing systolic and diastolic functions.

This second phase is still present few minutes after emersion as documented in the present study and is likely completely exhausted after 10 minutes, as suggested by the study of Frassi et al. (14), where 10-15 minutes following prolonged breath-hold diving LV morphology and systo-diastolic functions were not different from the pre-diving condition.

The above hypothesized scenario — i.e., the peripheral to central blood shift followed by a counter blood shift from central to periphery, respectively during the descent and ascent breath-hold diving phases — is suggested by the recent data showing an increased diffusing capacity for the lungs, measured using single-breath carbon monoxide test, and attributed to expanded pulmonary capillary blood volume, at two minutes after breath-hold diving and progressively decreasing toward baseline at 10 and 25 minutes (22).

In addition, a further mechanism possibly favoring the center to periphery blood shifting early after breath-hold diving is the reduction in intrathoracic pressure when the glottis is reopened.

Factor II suggests that the increased LV ventricular filling is also favored by the improved diastolic function documented by increased rapid filling (mitral echo-Doppler E wave). This finding appears in line with the improved LV compliance documented following physical performances in athletes (16). Factor III further links the improvement of LV function to modifications of cardiac loading — i.e., the reduction in SVR. Furthermore, this factor also suggests the role of baro-receptive reflex in increasing HR secondary to SVR reduction.

Finally, the lack of any hypoxemia-related neurological symptomatology (13,18) together with normal LV regional wall motion observed in the present study supports the view that the reduction of CO observed during diving by Marabotti et al. (19) is not sustained by hypoxemia but rather by hemodynamic changes such as blood volume redistribution associated with constriction of the heart by increased environmental pressure. In a previous
study, prolonged air breath-holding induced LV dilatation, reduction in regional systolic function as assessed by PSWT, but increased LV stroke volume secondary to LV dilatation (21). The hypothesized mechanism was that the increased LV filling was necessary to maintain stroke volume in accordance with Frank-Starling law.

In contrast, in the present study, LV diastolic volume paralleled the increase in PSWT. This difference may depend on the different study conditions of breath-holding, given that the breath-hold model was static and maximal in the previous study, whereas it was dynamic in the present study. Secondly, LV function was assessed during breath-holding in the previous study, whereas in the early recovery phase after the end of breath-holding in the present study.

Some limitations of the present study deserve mention. Echo-Doppler measurements were all performed following the recommendations of the American Society of Echocardiography (15). However, because of the logistic difficulties related to a field character of our study, we did not study the diastolic function according to the state of the art, i.e., tissue Doppler imaging and assessment of pulmonary venous flow velocity. For the same logistic reasons, ultrasound imaging could not be anticipated.

The succession of events hypothesized in the present study is the result of the combination of the results of different studies obtained in different subjects and in different experimental conditions. To confirm this view further studies are required, being left ventricular function evaluated in rapid sequence in the same diver during and after breath-hold diving.

CONCLUSIONS
Our study showed that in healthy athletes LV systo-diastolic functions early after breath-hold diving in deep water was enhanced, compared to prediving, due to the recruitment of left ventricular preload reserve, and to the reduction of afterload, suggesting that the improved systo-diastolic function of the heart is dependent upon the favorable changes in loading conditions.

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References


