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HIGHLIGHTED TOPIC | *The Physiology and Pathophysiology of the Hyperbaric and Diving Environments*

Cardiac changes induced by immersion and breath-hold diving in humans

Claudio Marabotti,[1,2] Alessandro Scalzini,[1] Danilo Cialoni,[3] Mirko Passera,[2] Antonio L'Abbate,[2,4] and Remo Bedini[2]

[1]*Unità Operativa Cardiovascolare-Utic Ospedale di Cecina (Livorno), Padua;* [2]*Consiglio Nazionale delle Ricerca Institute of Clinical Physiology, Pisa;* [3]*Apnea Academy Research, Padua; and* [4]*Scuola Superiore Sant'Anna, Pisa, Italy*

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Marabotti C, Scalzini A, Cialoni D, Passera M, L'Abbate A, Bedini R. Cardiac changes induced by immersion and breath-hold diving in humans. *J Appl Physiol* 106: 293–297, 2009. First published May 8, 2008; doi:10.1152/japplphysiol.00126.2008.—To evaluate the separate cardiovascular response to body immersion and increased environmental pressure during diving, 12 healthy male subjects (mean age 35.2 ± 6.5 yr) underwent two-dimensional Doppler echocardiography in five different conditions: out of water (basal); head-out immersion while breathing (*condition A*); fully immersed at the surface while breathing (*condition B*) and breath holding (*condition C*); and breath-hold diving at 5-m depth (*condition D*). Heart rate, left ventricular volumes, stroke volume, and cardiac output were obtained by underwater echocardiography. Early (E) and late (A) transmitral flow velocities, their ratio (E/A), and deceleration time of E (DTE) were also obtained from pulsed-wave Doppler, as left ventricular diastolic function indexes. The experimental protocol induced significant reductions in left ventricular volumes, left ventricular stroke volume ($P < 0.05$), cardiac output ($P < 0.001$), and heart rate ($P < 0.05$). A significant increase in E peak ($P < 0.01$) and E/A ($P < 0.01$) and a significant reduction of DTE ($P < 0.01$) were also observed. Changes occurring during diving (*condition D*) accounted for most of the changes observed in the experimental series. In particular, cardiac output at *condition D* was significantly lower compared with each of the other experimental conditions, E/A was significantly higher during *condition D* than in *conditions A* and *C*. Finally, DTE was significantly shorter at *condition D* than in basal and *condition C*. This study confirms a reduction of cardiac output in diving humans. Since most of the changes were observed during diving, the increased environmental pressure seems responsible for this hemodynamic rearrangement. Left ventricular diastolic function changes suggest a constrictive effect on the heart, possibly accounting for cardiac output reduction.

diving response; hemodynamics

PREVIOUS STUDIES ON NATURAL divers (mainly marine mammals) showed that breath-hold diving is associated with energy-saving cardiovascular changes, i.e., a marked reduction in cardiac output [due to the reduction of both stroke volume and heart rate (HR)] and the redistribution of blood flow away from skin and myoglobin-rich muscles in favor of brain and heart (7, 28, 30). For years, however, technical difficulties have prevented a comprehensive assessment of cardiovascular changes during breath-hold diving in humans. Most of the knowledge on human diving physiology has been obtained from the study of head-out immersed subjects (9, 27), or extrapolated from the results obtained in breath-holding subjects, either with or without face immersion (2, 8, 10). The recent wide diffusion of recreational and competitive breath-hold diving (with a progressive increase of attained depths) highlighted the presence of serious diving-related pathologies, like syncope (ascent blackout), decompression illness, hemoptysis, and pulmonary edema, whose pathophysiology is not yet completely understood (13, 31). A deeper knowledge of breath-hold diving physiology in humans seems thus needed.

Using a submersible echocardiographic machine, we recently showed, in humans, during short breath-hold dives up to 10-m depth, a hemodynamic pattern qualitatively similar to that described in marine mammals (22), with a significant decrease in HR, stroke volume, and, hence, cardiac output.

Breath-hold diving involves progressive decrease of O_2 and increase of CO_2 blood content, changes in temperature and thermal conductivity, evocation of neural reflexes (induced by face immersion), change in environmental pressure (linearly increasing with depth), which, in turn, modulates the venous return to the heart (21). The relative role of these factors in determining the cardiovascular response to diving is still to be elucidated.

The aim of the present study was to separately evaluate, in humans, the cardiovascular response to body immersion, with or without face immersion, breath holding, and diving.

MATERIALS AND METHODS

Subjects. A group of 12 healthy male subjects (age 35.2 ± 6.5 yr; range 24–51 yr; height 180.2 ± 6.8 cm; weight 77.4 ± 10.2 kg; body mass index 23.8 ± 2.3 kg/m^2) was studied. The absence of female subjects was casual and not due to a selection criterion. All subjects were experienced, active breath-hold divers (practicing breath-hold diving from 5.8 ± 3.5 yr), undergoing at least 2 h/wk of breath-hold diving training; no subject was engaged in regular physical activity besides underwater training. Each diver had the ability to reach a depth of at least 30 m under constant weight (i.e., with no ballast aid for descent); their maximum static breath-hold time at surface was 4.5 ± 0.8 min (range 4–6 min). No subject had historical, clinical, or instrumental (resting ECG, Doppler echocardiography) evidence of

Address for reprint requests and other correspondence: C. Marabotti, UO Cardiovascolare-UTIC, Ospedale di Cecina, Via Montanara, 57023 Cecina (LI), Italy (e-mail: maralbian@alice.it).

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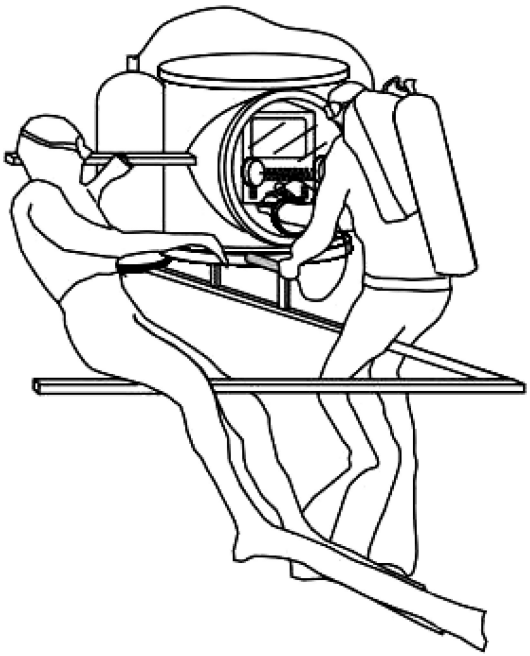

Fig. 1. Schematic representation of the underwater echocardiographic equipment.

arterial hypertension or cardiac or pulmonary diseases. All subjects were nonsmokers and had been fasting from at least 2 h before the study.

The study protocol was approved by the Scientific Committee of the Consiglio Nazionale delle Ricerca Institute of Clinical Physiology. All participants were informed about the aims and procedures of underwater ultrasound examination and gave their written consent.

Underwater echocardiographic equipment. Doppler-echocardiographic examination was performed by a commercially available instrument (MyLab 30, Esaote SPA, Florence, Italy) as part of a submersible echograph previously described elsewhere (5). Briefly, a special, patented, water-tight container, made by two steel cylinders (60-cm diameter) intersecting each other (Fig. 1) contained the echocardiograph (6). A rubber glove sealed to the front Plexiglas panel allowed the user to access the instrument's control keys. A pressure regulator connected to a standard 200-ATA compressed air cylinder (normally used for scuba diving) maintained the pressure inside the container of the echocardiographic instrument at the same level of external pressure, reducing the risk of water leakage and preventing rubber glove inflation. Ultrasound reflection in water was prevented by the rubber and silicon backing of the probe, ensuring the extinction of backward ultrasound radiation. The absence of ultrasound interferences in free water was confirmed by tests performed by the manufacturing company.

Experimental protocol. The study was performed, between 10 AM and 2 PM, in a 10-m-deep pool (water temperature 29°C; air temperature 27°C). Subjects were studied, by two-dimensional Doppler echocardiography, in five different conditions: out of water (basal), head-out immersion during normal breathing (*condition A*), fully immersed (head-in) at the surface while breathing by a snorkel (*condition B*), fully immersed (head-in) at the surface while breath holding (*condition C*), and, finally, breath-hold diving at 5-m depth (*condition D*). Subjects were simply wearing a bathing suit and, during full immersion (*conditions B*, *C*, and *D*), a diving mask. Echocardiogram was recorded, in each condition, with the subject lying on his left side during normal breathing (basal, *conditions A* and *B*) or after a maximal inspiration (*conditions C* and *D*). Each dive was preceded by 2–4 min of surface floating preparation, during which subjects were breathing normally (no preliminary hyperventilation was done). Descents were done by using 10-kg ballast, thus reducing the cardiovascular effects of muscular work. Subjects equilibrated the pressure in the medium ear by Frenzel technique (in most cases) or by a mild Valsalva maneuver. As soon as the diver reached the echo station, he positioned himself on a metallic bracket, lying on his left side (Fig. 1). Cardiac imaging started during the first minute of apnea and lasted <90 s in all subjects, so that echo-Doppler data were acquired in a cardiovascular steady state (not influenced by the possible, if any, effects of Valsalva maneuver, nor by those of hypoxia).

The time necessary for handling and positioning the underwater echocardiograph in the different positions prevented the possibility of performing, in each subject, the entire series of tests consecutively. The sequence of echocardiographic studies was the following: basal for all subjects; after the launch of the echocardiograph, the three studies at surface (*conditions A*, *B*, *C*) were performed consecutively in each subject. Finally, after echocardiograph was positioned at depth, ultrasonic study during diving (*condition D*) was made in all subjects. During the periods of instrumentation handling, subjects stayed out of the water, at rest.

Doppler-echocardiographic parameters. To minimize the duration of the echocardiographic study during diving, we recorded only an apical four-chamber view loop (4-s duration) and a pulsed-wave Doppler tracing of transmitral blood flow. Analysis was made offline, according to the American Society of Echocardiography recommendations (19), by an expert in Doppler-echocardiography, unaware of the identity of the subjects and of the condition of recording. From the four-chamber view, the following parameters were obtained: systolic and diastolic left ventricular volumes (calculated by area-length method) (14, 19), and right ventricular internal dimension (maximal diastolic distance from right-side interventricular septum to right ventricular free wall). From the same view, maximal transversal (from interatrial septum to the opposite atrial wall) and supero-inferior (from the mitral valve plane to the opposite wall) dimensions were calculated for 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; E-to-A ratio (E/A) was then calculated. Such indexes allowed the characterization of left ventricular diastolic function, as different filling patterns have been described in case of delayed ventricular

Table 1. *Doppler-echocardiographic data in basal (dry) and in the four different immersion conditions*

Conditions	LV EDV, ml	LV ESV, ml	LV SV, ml	CO, l/min	HR, beats/min	RVD, mm	E Peak, cm/s	A Peak, cm/s	E/A	DTE, msec
Basal	169.9±27.6	75.8±17.3	94.1±19.5	6.3±1.5	66.7±8.5	38.4±7.1	69.8±12.8	51.8±11.0	1.39±0.3	214.2±46.5
A	186.8±29.9	86.9±22.8	99.9±21.6	6.8±2.0	68.3±14.0	36.1±3.1	75.9±16.6	53.2±11.9	1.39±0.3	193.8±33.4
B	173.2±27.1	83.2±12.2	90.0±23.2	6.1±1.9	67.7±11.0	42.8±1.8	76.8±13.6	48.9±8.7	1.6±0.29	183.5±38.9
C	152.7±21.2	60.6±11.5	93.1±18.3	5.2±1.5	57.3±16.0	40.0±2.1	77.7±26.9	45.6±15.7	1.89±1.06	191.7±34.5
D	138.4±27.0	67.2±15.2	71.1±16.2	3.8±1.2	55.6±22.1	39.5±2.7	99.2±25.9	46.4±19.8	2.49±1.29	143.7±19.3

Values are means ± SD. See MATERIALS AND METHODS for definition of basal and *conditions A*, *B*, *C*, and *D*. LV, left ventricle; EDV, end-diastolic volume; ESV, end-systolic volume; SV, stroke volume; CO, cardiac output; HR, heart rate; RVD, right ventricular dimension; E peak, early transmitral flow velocity; A peak, late transmitral flow velocity; E/A, ratio of E to A; DTE, deceleration time of E peak.

Table 2. *P values for comparisons between the different experimental conditions*

	LV EDV	LV ESV	LV SV	CO	HR	RVD	E Peak	A Peak	E/A	DTE
Basal vs. A	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Basal vs. B	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Basal vs. C	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Basal vs. D	NS	NS	NS	0.004	NS	NS	NS	NS	NS	0.009
A vs. B	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
A vs. C	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
A vs. D	0.012	NS	0,03	0.001	NS	NS	NS	NS	0.001	NS
B vs. C	NS	0.028	NS	NS	NS	NS	NS	NS	NS	NS
B vs. D	NS	0.003	NS	0.006	NS	NS	NS	NS	NS	NS
C vs. D	NS	NS	NS	0.045	NS	NS	NS	NS	0.033	0.037

NS, nonsignificant.

relaxation (as in early hypertensive heart disease or during aging), as well as in situations of increased wall stiffness (as in advanced hypertensive heart disease or in constrictive/restrictive heart diseases) (24). Duration of cardiac cycle (R-R interval) was measured as the time interval between two consecutive mitral A peaks; HR was then calculated (60/R-R interval expressed in seconds); the mean value of 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 HR.

Statistical analysis. Data are reported as means ± SD. Normal distribution of the parameters was evaluated preliminarily by the nonparametric Kolmogorov-Smirnov test. All parameters were normally distributed. Analysis of variance for repeated measures was used to evaluate the global effect of the experimental protocol. A post hoc analysis, according to Bonferroni's method, was then implemented, to evaluate the differences between each experimental condition. A probability <5% was assumed as threshold to reject the null hypothesis.

RESULTS

Mean values of Echo-Doppler cardiac parameters observed in the different experimental conditions are reported in Table 1.

The analysis of variance showed, along the series of immersion and diving experiments, significant reductions in left ventricular volumes, both diastolic and systolic ($P < 0.01$ for both), left ventricular stroke volume ($P < 0.05$), cardiac output ($P < 0.001$), and HR ($P < 0.05$). As concerns Doppler indexes of left ventricular diastolic function, an increase in E peak ($P < 0.01$) and E/A ($P < 0.01$) and a reduction in DTE ($P < 0.01$) were observed.

The results of post hoc analysis (Table 2) showed that changes occurring during 5-m breath-hold diving (*condition D* step) accounted for most of variance observed in the experimental series. In particular, cardiac output in *condition D* was lower than at any other experimental conditions (Fig. 2); left ventricular diastolic volume and stroke volume were lower in *condition D* than in *A* (head-out immersion), whereas left ventricular systolic volume results were lower in *condition D* than in *B* (surface submersion while breathing). As concerns diastolic function indexes, E/A was higher in *condition D* than in *condition A* (head-out immersion) and *C* (full body immersion at the surface while breath-holding). Finally, DTE was significantly shorter in *condition D* than in basal and in *condition C* (Fig. 3).

No significant changes were observed in the dimensions of right ventricle at end diastole and of left atrium.

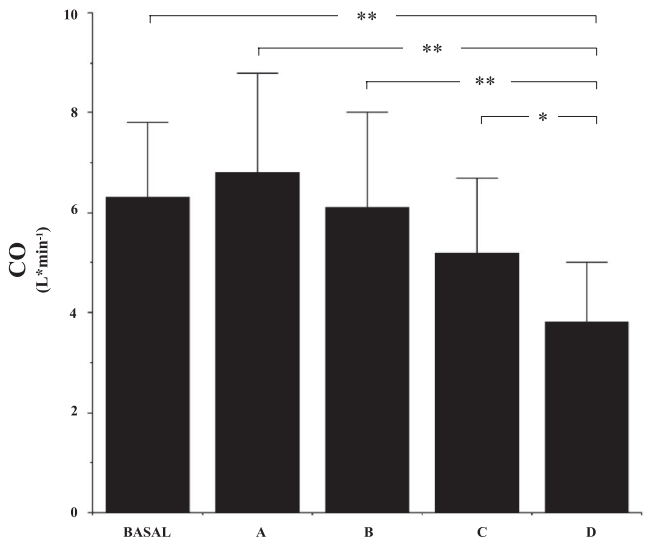

Fig. 2. Cardiac output (CO) in basal (dry) conditions and in the different immersion states. Values are means ± SD. See MATERIALS AND METHODS for definition of *conditions A*, *B*, *C*, and *D*. *$P < 0.05$; **$P < 0.01$.

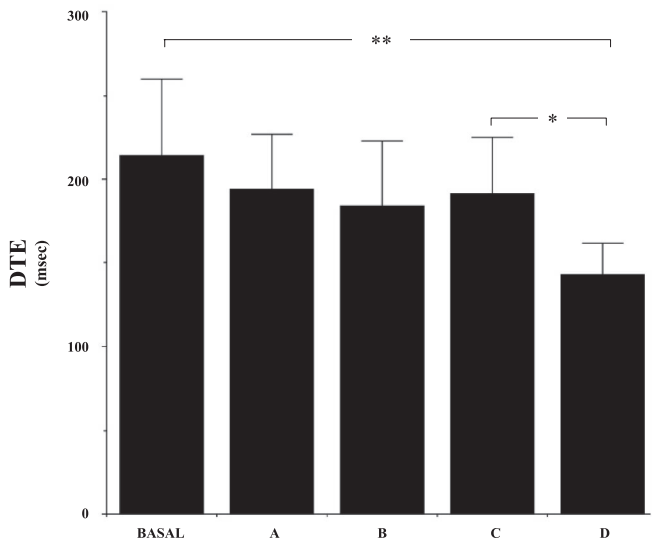

Fig. 3. Deceleration time of E peak (DTE) in basal (dry) conditions and in the different immersion states. Values are means ± SD. *$P < 0.05$; **$P < 0.01$.

DISCUSSION

The present study confirms and extends previous observations of a clearly appreciable diving response that leads to a reduction in cardiac output during breath-hold diving in humans (22). The sequence of immersions actually induced a significant reduction in cardiac output (due to a decrease in both HR and stroke volume) and in left ventricular diastolic and systolic volumes. Such a hemodynamic pattern is consistent with a preload reduction (since both increased afterload and/or reduced myocardial contractility would have implied increased left ventricular volumes).

The design of the study aimed to discriminate among the possible determinants of the diving cardiovascular response, as breath-hold diving exposes the organism to a series of stimuli (body immersion, breath holding, diving reflex elicitation, environmental pressure effects), overlapping each other. Most significant cardiac changes were observed during diving at depth, while surface immersion (irrespective of head in or out, breathing or breath holding) had, in our series, trivial effects on cardiac function. These data may be explained by several reasons. The progressive application of immersion stimuli (from head-out to diving at depth) may have attenuated cardiovascular changes at each step. Moreover, the reduced stimulation of facial receptor during submersion (subjects were wearing a diving mask), the small difference between air and water temperatures, and the relatively comfortable water temperature, not far from thermoneutrality, may also contribute to explaining this observation, since both diving-induced bradycardia and peripheral vasoconstriction are marked in cold water (1, 23, 26). On the other hand, full body immersion in colder water can elicit sympathetic activation, potentially affecting cardiovascular response to immersion and diving (18). Thus body immersion, breath holding, and elicitation of diving reflex seem to have, per se, a relatively minor role in humans compared with the effect of diving at depth. Therefore, the increase in hydrostatic pressure seems to be essential in inducing cardiovascular changes during breath-hold diving. A previous study in humans, evaluating immersion, submersion, and simulated diving at depth in a pressure chamber (12), obtained different results, with cardiac output during diving significantly higher compared with both dry measurement at 1 ATA and surface breath holding. Methodological differences in cardiac output measurement and the possible influences of a sympathetic activation due to the unfamiliar experience represented by compression in a confined space might explain this discrepancy (25).

An increase in E/A with reduction of deceleration time of early filling peak was observed, during diving at depth, at Doppler evaluation of transmitral blood flow. This change, already observed in breath-hold diving athletes (22), is, in the clinical setting, typical of a restrictive/constrictive left ventricular diastolic dysfunction (24). It may be hypothesized that the reduction of chest volume (due to the increased environmental pressure), combined with an increase in intrathoracic blood content (3, 16), may exert a constraint on the heart, able to induce an impairment of left ventricular filling and, in turn, a relative reduction in preload (transmural filling pressure) and cardiac output (22). It may be speculated that these changes might contribute to the pathophysiology of diving-induced acute pulmonary edema. During deep and prolonged dives, pulmonary vascular bed congestion (due to both an increased venous return and an impaired left ventricular filling), combined with an uneven hypoxic pulmonary vasoconstriction caused by hypoxia (4, 15), could lead to a pulmonary capillary stress failure (33) and pulmonary edema (34). The unfavorable consequences of a large intrathoracic blood redistribution, combined with thoracic squeeze during diving, are supported by two observations. On one hand, a recent report by Lindholm et al. (20) showed that breath-hold divers may have hemoptysis and instrumental signs of lower airway edema after shallow diving (6 m) performed at residual volume. On the other hand, animals highly adapted to diving (like pinnipeds) have specialized anatomical structures devoted to reduce the intrathoracic venous return during immersion (caval sphincter, hepatic sinus) (11, 29).

It is noteworthy to mention that changes in right ventricular diastolic dimension were not observed at any stage of the protocol, while intrathoracic blood displacement induced by immersion (3, 16) should, theoretically, be associated with right ventricular volume overload (17). Our negative finding might be explained by circulatory adjustments that rapidly occur in the period preceding cardiac imaging during the different experimental conditions. Alternatively, it might reflect the intrinsic inefficiency of echocardiography in accurately detecting small changes in right ventricle dimensions, owing to its complex three-dimensional anatomy (32).

In conclusion, our study documents, in humans performing breath-hold diving at shallow depth (5 m), a cardiovascular response qualitatively similar to marine mammals. Body immersion at surface, diving reflex elicitation, and breath holding all seem to contribute only marginally to cardiac changes observed at depth, where the hydrostatic pressure on the chest becomes sufficiently high to constrict the heart, hampering its diastolic filling and reducing stroke work.

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