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ORIGINAL ARTICLE

Hemodynamic adjustments during breath-holding in trained divers

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Abstract
Purpose Voluntary breath-holding (BH) elicits several hemodynamic changes, but little is known about maximal static immersed-body BH. We hypothesized that the diving reflex would be strengthened with body immersion and would spare more oxygen than maximal dry static BH, resulting in a longer BH duration.
Methods Eleven trained breath-hold divers (BHDs) performed a maximal dry-body BH and a maximal immersed-body BH. Cardiac output (CO), stroke volume (SV), heart rate (HR), left ventricular end-diastolic volume (LVEDV), contractility index (CTI), and ventricular ejection time (VET) were continuously recorded by bio-impedancemetry (PhysioFlow PF-05). Arterial oxygen saturation (SaO_2) was assessed with a finger probe oximeter.
Results In both conditions, BHDs presented a bi-phasic kinetic for CO and a tri-phasic kinetic for SV and HR. In the first phase of immersed-body BH and dry-body BH, results (mean ± SD) expressed as percentage changes from starting values showed decreased CO (55.9 ± 10.4 vs. 39.3 ± 16.8 %, respectively; $p < 0.01$ between conditions), due to drops in both SV (24.9 ± 16.2 vs. 9.0 ± 8.5 %, respectively; $p < 0.05$ between conditions) and HR (39.7 ± 16.7 vs. 33.6 ± 17.0 %, respectively; $p < 0.01$ between conditions). The second phase was marked by an overall stabilization of hemodynamic variables. In the third one, CO kept stabilizing due to increased SV (17.0 ± 20.2 vs. 10.9 ± 13.8 %, respectively; $p < 0.05$ between conditions) associated with a second HR drop (14.0 ± 10.0 vs. 12.7 ± 8.9 %, respectively; $p < 0.01$ between conditions).
Conclusion This study highlights similar time-course patterns for cardiodynamic variables during dry-body and immersed-body BH, although the phenomenon was more pronounced in the latter condition.

Keywords Diving reflex · Water immersion · Kinetics · Hemodynamic · Breath-holding

Introduction

The human physiological response to breath-holding (BH) is called the "diving reflex" and the main effects are bradycardia, decreased cardiac output, and increased arterial blood pressure (Foster and Sheel 2005). Bradycardia is induced by increased vagal activity, whereas the peripheral vasoconstriction of selected vascular beds is linked to increased sympathetic discharge (Fagius and Sundlöf 1986). Recent studies have included splenic contraction, which occurs at the early stage of BH, as part of the diving reflex (Palada et al. 2007a). In the human species, the diving reflex is triggered by BH and accentuated by face immersion (Andersson et al. 2000). These mechanisms slow the depletion of lung oxygen stores through an oxygen-conserving effect, thereby reducing overall O_2 uptake (Andersson et al. 2008). Although most studies on human diving physiology deal with the cardiovascular results of BH performed in laboratory conditions, i.e., dry static BH or simulated dynamic BH with face immersion (Palada et al. 2007b; Heusser et al. 2009; Tocco et al. 2012a), a few recent studies have confirmed these phenomena in real

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conditions. These studies have shown a significant decrease in cardiac output (CO) due to reductions in both heart rate (HR) and stroke volume (SV) during deep static BH (Marabotti et al. 2009; Tocco et al. 2012b). However, these investigations did not focus on the time courses of the cardiodynamic variables during BH and the hemodynamic data were recorded only over short periods (respectively, 1 and 3 min), which does not reflect the real hemodynamic changes during a maximal static BH performed in competition.

The purpose of this investigation was to describe the hemodynamic and arterial oxygen saturation (SaO_2) changes during maximal static BH both in air and in immersion. We hypothesized that the magnitude of the diving reflex (hemodynamic parameters) would be more pronounced in immersion than in air. Therefore, a more pronounced diving response during static BH with body immersion (head-in) was expected to spare more oxygen than during dry BH, explaining a longer duration of BH.

Materials and methods

Subjects

The study was performed on 11 healthy active breath-hold divers (BHDs; 10 males and 1 female). The presence of a single female subject was fortuitous, and a recent article concluded that males and females have similar diving responses (Tocco et al. 2012a). All participants were informed about the objectives and procedures of the study, and all gave written consent prior to the start of the experiment. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local research ethics committee.

Experimental design

Each subject performed two maximal voluntary static BH, as they would do while training, in two experimental conditions. The first BH was carried out in air and the second was performed at water surface in a fully-immersed condition (head-in). Dry-body BH and immersed-body BH were conducted in the same session with at least 4 h between each one and were preceded by a 15 min rest period. During the first rest period (in air), the subjects relaxed in supine position on a mattress and data such as systolic and diastolic blood pressure, calibration of hemodynamic parameters and baseline values were recorded. After dry-body BH and before entering the water, the subjects put on wet suits, diving masks and snorkels. Before starting immersed-body BH, the subjects assumed the prone position and breathed through the snorkel with their heads submersed in water while baseline data were recorded underwater. Prior to starting BH, BHDs were asked for their expected BH duration. In both conditions, BHDs followed a countdown process similar to that of BH competitions, i.e., countdown signals were given with the following timing (seconds): 120, 90, 30 then every second for the last 10 s prior to starting. After 2.5 min into BH, the subjects were notified of the elapsed time every 30 s. The subjects were instructed to keep their chest relaxed and to perform BH without prior hyperventilation. Ambient air and pool water temperature were, respectively, 26 and 27 °C.

Arterial oxygen saturation and hemodynamic measurements

SaO_2 was assessed by fingertip pulse oximetry (PalmSat 2500, Nonin Medical, Inc., USA). Contractility index (CTI), ventricular ejection time (VET), left ventricular end-diastolic volume (LVEDV), CO, SV and HR were estimated by bio-impedancemetry (PhysioFlow PF-05, Manatec Biomedical, France), a non-invasive method commonly used nowadays to determine cardiodynamic parameters at rest (Charloux et al. 2000; Tonelli et al. 2011) and during exercise (Tordi et al. 2004; Welsman et al. 2005). The PhysioFlow methodology has been described in detail elsewhere (Richard et al. 2001). Briefly, the bio-impedance method for determining CO uses transthoracic impedance changes (dZ) in response to an electrical current administered during cardiac ejection to calculate SV. After shaving and applying a mildly abrasive gel (NuPrep) to the skin, two sets of electrodes are applied above the supraclavicular fossa (left side) and along the xiphoid process of each subject. Another pair of electrodes is used to measure a single electrocardiogram signal (ECG). After a calibration over 30 heart beats, CO is then continuously calculated (beat-to-beat) by multiplying the stroke volume index (SVi) with the body surface area (BSA) and HR, which is obtained from the R–R interval determined on the ECG first derivative:

$$\mathrm{CO}\ \left(\mathrm{l\ min^{-1}}\right) = \mathrm{HR}\ \left(\mathrm{beats\ min^{-1}}\right) \times \mathrm{SVi}\ \left(\mathrm{ml\ m^{-2}}\right) \times \mathrm{BSA}\ \left(\mathrm{m^2}\right)$$

The mean systolic ejection rate (SV/VET ratio, MSER) was calculated, a parameter considered as an index of myocardial performance (Concu and Marcello 1993).

Particular precautions were taken while recording the hemodynamic data underwater to preserve the impedance signal, i.e., the electrode contact with the skin was protected from water by adhesive films (Tegaderm™) and the wetsuits worn by the subjects. Upon completion of the recording, the ICG and impedance waveform (the "drums and saxophone" criteria) were verified according to the designers' recommendations for this impedance-based

device (Bour and Kellett 2008). Before starting the whole experiment, the quality of the impedance signal was checked on a subject in true experimental condition and no sign of moistened electrodes was noted. The same experimenter used this device and performed the hemodynamic recording throughout the study.

Data analysis

The baseline values were calculated for each variable as the average value over a 5 min period in both rest conditions. Throughout BH, the average values of CO, SV and HR were calculated over 3–5 heart beats for every 20 s. SaO_2 was also averaged every 20 s. The mean value over 3–5 heartbeats at the beginning of BH was used as starting value for each variable as well as reference value for each hemodynamic change during BH (represented by the 100 % time points values on Fig. 1). Because of both hemodynamic variations induced by body immersion and inter-individual variance, we chose to normalize the data using the percentage change from the starting value for each BH as follows:

$$\text{relative hemodynamic change } (\%) = \frac{\text{absolute hemodynamic value}}{\text{absolute hemodynamic starting value}} \times 100$$

All samples were first tested for normality distribution with the Shapiro–Wilk test. Because of the latter test failure, Friedman's non-parametric test was used to compare the time courses of CO, SV, HR and SaO_2 for each condition. Either Student's paired *t* test or Wilcoxon's tests were used to compare each value with the next, depending on the hemodynamic data distribution of each time point. In a similar manner, Student's paired *t* test or Wilcoxon's test were used to do a point-to-point comparison between dry-body and immersed-body results. Spearman or Pearson correlations were performed to assess the relationships between BH times, years of BH practice and the hemodynamic data, depending on the data distribution. For the sake of clarity, the results shown in the figure are expressed as the mean value ±SE, whereas the results in tables and the text are expressed as the mean value ±SD. Statistical analysis and graphs were performed using Sigma Plot software version 12.3 (SPSS, Chicago, USA) and a *p*-value <0.05 was considered statistically significant.

Results

The cumulative years of BH practice, time spent in BH training per week, personal best performance in static BH, and anthropometric data of BHDs are shown in Table 1. The BH duration was longer in immersed-body BH than in dry-body BH (267 ± 21.9 vs. 229 ± 12.3 s, $p < 0.05$). Baseline and BH starting values for both conditions are presented in Table 2. The Friedman analysis for the time course of hemodynamic changes showed a decrease in CO, SV HR and SaO_2 in both immersed-body and dry-body BH from their respective BH starting values (Fig. 1). More specifically, CO, SV and HR showed a drop in the early stage (1 min) for both BH conditions (Fig. 1a–c). Furthermore, HR again decreased when BH was 55–60 % completed (time-wise) and kept sliding until completion of BH (Fig. 1c). SV was the only parameter that increased in the last phase of BH whether in immersed-body BH (17.0 ± 20.2 %, $p < 0.05$ vs. at 140 s time point) or dry-body BH (10.9 ± 13.8 %, $p < 0.05$ vs. at 140 s time point; Fig. 1b). CO in immersed-body BH reached percentage lower than in dry-body BH (55.9 ± 10.4 vs. 39.3 ± 16.8 %, respectively; $p < 0.01$ between conditions) and HR (first drop 39.7 ± 16.7 vs. 33.6 ± 17.0 %, respectively $p < 0.01$ between conditions; second drop: 14.0 ± 10.0 vs. 12.7 ± 8.9 %, respectively, $p < 0.01$ between conditions; Fig. 1a, c). In both immersed-body BH and dry-body BH, mean SaO_2 decreased (7.9 ± 2.3 vs. 5.0 ± 1.4 %, respectively; $p < 0.01$ between conditions), although no difference in SaO_2 was found between conditions regardless of the BH time point (except at the end of BH, Fig. 1d). From the beginning to 55–60 % of BH duration, LVEDV decreased in immersed-body BH (6.8 ± 10.3 %, $p < 0.05$ vs. starting values) and dry-body BH (7.2 ± 8.7 %, $p < 0.05$ vs. starting values) and then increased from 55–60 % to the end of BH in immersed-body BH (8.9 ± 12.3 %, $p < 0.05$ vs. at 140 s time point) and dry-body BH (11.0 ± 9.0 %, $p < 0.05$ vs. at 140 s time point). No difference was found in VET. CTI and MSER decreased only in immersed-body BH (45.3 ± 49.4 %; $p < 0.05$ and 17.5 ± 7.4 %, respectively; $p < 0.01$ vs. baseline values). No correlation was found between BH times, years of apnea practice and hemodynamic data.

Discussion

The main finding of the present study was that BHDs showed a bi-phasic time course of CO associated with a tri-phasic time course of SV and HR while BH. The first phase presented a fast and pronounced decrease in all hemodynamic parameters followed by a second one whose main feature was an overall hemodynamic stabilization. The third kinetic phase (55–60 % of BH time) showed a second bradycardia associated with an increase in SV, which resulted in the continuation of the current CO stabilization. This kinetic was very similar in the two conditions and

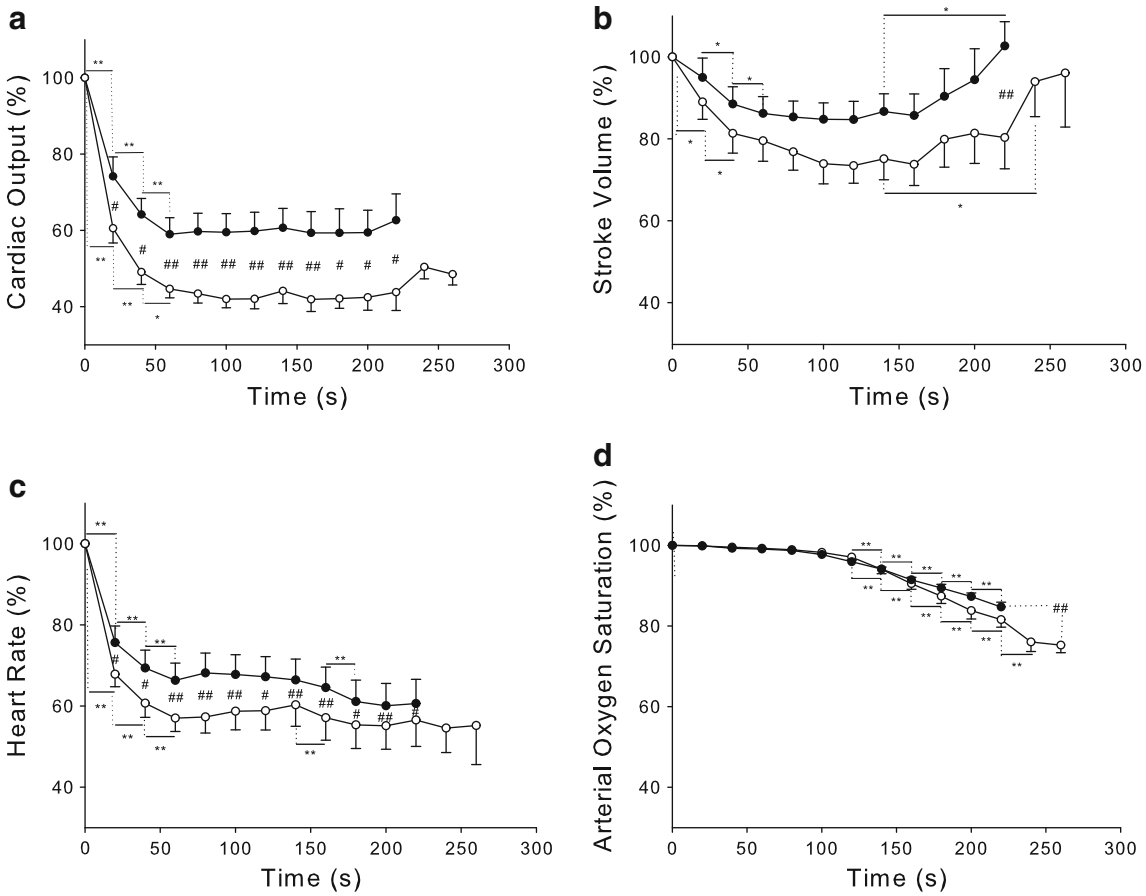

Fig. 1 **a–d** Cardiac output, stroke volume, heart rate and arterial oxygen saturation time course in dry-body breath-holding and immersed-body breath-holding. Values are mean ± SE percentage from the starting values of breath-holding. $*p < 0.05$; $**p < 0.01$ versus precedent time point; $^{\#}p < 0.05$; $^{\#\#}p < 0.01$ versus corresponding time point of the other condition. *Filled circles* dry-body BH, *empty circles* immersed-body BH

Table 1 Anthropometric data of breath-hold divers (BHDs)

	$n = 11$
Age (years)	36.0 ± 9.6
Height (cm)	175.7 ± 4.2
Body mass (kg)	71.2 ± 8.9
Body fat mass (%)	19.9 ± 6.0
YAP	7.3 ± 5.6
BH training (h wk^{-1})	3.8 ± 1.13
PB (s)	316 ± 40

Values are mean ± SD

BH breath-holding, *YAP* years of apnea practice, *PB* personal best static BH performance

occurred at almost the same absolute time of BH although it was stronger in underwater BH. Similar bi-phasic and tri-phasic time courses have been reported in elite BHDs, but most of the previous studies focused mainly on HR and/or arterial blood pressure (Lemaître et al. 2008; Perini et al. 2008; Perini et al. 2010).

In this investigation, the hemodynamic results revealed a fast drop in CO during the initial phase of BH in both conditions as a result of decreased SV and HR. Despite the bradycardic response is variable among individuals, both time courses and magnitudes of the first bradycardia are within the range reported in other investigations (Caspers et al. 2011; Alboni et al. 2011). In studies with similar immersion conditions, Tocco et al. (2012a) also highlight a drop in CO, whereas Marabotti et al. (2009) did not. From a physiological standpoint, the greatest effect of BH on cardiac kinetics seems to be the increased (positive) intrathoracic pressure (ITP) (Ferrigno et al. 1986; Ley et al. 2006). Indeed, Ferrigno et al. found a (24 %) decrease in the cardiac index as a consequence of increased ITP during BH when performed at large lung volume. In healthy humans, the increased ITP displaces the central blood volume into the peripheral capillary beds, i.e., a decrease in venous return, resulting in reduced transmural end-diastolic pressures, LVEDV and therefore SV (Tyberg et al. 2000). In our study, we indirectly confirmed this phenomenon in

Table 2 Baseline and starting BH hemodynamic data in both conditions (absolute values)

conditions	HR (bpm)	SV (ml)	CO (l min^{-1})	SaO_2 (%)	VET (ms)	CTI	MSER (ml s^{-1})	MBP (mmHg)	SVR (dyne s cm^{-5})
DA									
Baseline values	64 ± 7	95 ± 14	6.0 ± 0.7	99 ± 1	282.0 ± 17.9	215.2 ± 74.2	339.2 ± 58.2	91.6 ± 7.3	1229.0 ± 148.5
Starting BH values	96 ± 8*	97 ± 14	9.5 ± 1.8*	99 ± 0.5					
WI									
Baseline values	71 ± 10	86 ± 12	6.1 ± 0.9	99 ± 1	252.8 ± 36.6	212.9 ± 85.4	348.0 ± 89.1		
Starting BH values	98 ± 12*	96 ± 14	9.4 ± 1.4*	99 ± 0.5					

Values are mean ± SD

DA dry ambient, *WI* water immersion, *HR* heart rate, *SV* stroke volume, *CO* cardiac output, *SaO_2* arterial oxygen saturation, *VET* ventricular ejection time, *CTI* contractility index, *MSER (SV/VET ratio)* mean systolic ejection rate, *MBP* mean blood pressure, *SVR* systemic vascular resistance

* $p < 0.001$ versus corresponding baseline value

both conditions by showing a decrease in LVEDV (from 0 to 55–60 % BH time), a parameter which provides a good estimate for preload and thus SV, according to the Frank–Starling law.

In the last stage of BH, the hemodynamic kinetics revealed that CO kept stabilizing due to a second decrease in HR and an increased SV. The SV increase was confirmed in our study with an increase in LVEDV from 55–60 to 100 % of BH time. The hemodynamic kinetic showed that the onset at which the second decrease in HR and the concurrent rise in SV occurred is similar to the time reported for changes in oxygen and carbon dioxide concentration, i.e., the physiological break point, thus eliciting chemoreflex activation (Lin et al. 1974; Breskovic et al. 2012). Although diaphragmatic contractions were not recorded, we hypothesized that the increased SV corresponded to the "struggle phase", thus involving progressive involuntary breathing movements (IBMs) (Hentsch and Ulmer 1984). Our results are in agreement with a recent study which reported a rise in SV up to 90 % of its starting value at the end of BH, to carry on cerebral perfusion (Dujic et al. 2009). The authors reported that IBMs are involved in the decreased (negative) ITP, thereby leading to an increase in venous return, and thus SV, which normalizes CO.

The present study also demonstrated that immersed-body BH elicited a more powerful diving response compared with dry-body BH. This is in agreement with earlier studies (Schagatay et al. 2007; de Bruijn et al. 2009), but these investigations were performed in simulated conditions and none of them reported the time courses on SV and CO kinetics. In addition, our impedance-based data showing a decrease in both MSER and CTI (only in immersed-body BH) further highlight this phenomenon, implying reduced myocardial work. It should be noted that the short-term body immersion in moderately cold water haven't elicited any cardiovascular changes in both baseline and starting values. According to our data, the diving reflex magnitude is mainly related to the HR drop, as no difference was found in SV between respective time points in the two conditions (except at the end of BH time, Fig. 1b). The amplification of this "diving bradycardia" results from the synergistic effect of BH-induced respiratory arrest and face immersion, an effect that is greater than the sum of each individual response (Marsh et al. 1995). It is now accepted that the diving reflex is strengthened by face immersion (forehead, periorbital and nasal region) via the trigeminus nerve (Schaller et al. 2009), since strong links remain between the trigemino-cardiac reflex and the diving reflex (Cornelius et al. 2010). Our study reported significant hemodynamic changes in BHDs between dry-body BH and immersed-body BH at a water temperature above 20 °C. It should be noted that this additional effect of face immersion at a fairly similar water temperature has not been reported in previous studies in non-divers (Jay and White 2006; Jay et al. 2007). However, SaO_2 showed no difference between the two conditions when each relative time point was compared (except at the end of BH due to a longer immersed-BH duration). These results were unexpected and in disagreement with our initial hypothesis and the findings of a study performed at rest in a simulated condition (Andersson et al. 2008). Indeed, our results showed a longer immersed-body BH time and a more pronounced diving reflex throughout an immersed-BH without any change in SaO_2, while it has been shown than the oxygen-sparing effect appears to be proportional to the degree of the bradycardia (Lindholm and Lundgren 2009).

A number of limitations have to be considered in this study. The main one is the lack of continuous arterial blood pressure recording and subsequent lack of information on baroreflex activity and systemic peripheral resistance while in BH. One could also question the impedance-based method we used to track changes in the hemodynamic parameters. Early impedance-based devices lacked validity as they were based on basal thoracic impedance, a measure of chest resistance that varies with thoracic morphology, the homogeneity of thorax perfusion, electrode placement, perspiration, subcutaneous adiposity and electrical contact (Charloux et al. 2000). Nowadays, more current impedance devices such as the PhysioFlow are baseline-independent, which facilitates underwater recording. To our knowledge, this is the first study to report underwater hemodynamic time courses while BH with a new-generation impedance-based device. A study using the same device reported an overestimation of CO in patients with chronic obstructive pulmonary disease compared with the direct Fick method (Bougault et al. 2005). The authors hypothesized that changes in ITP, pulmonary vascular flows and dynamic hyperinflation may have modified the first mathematical derivative of the impedance signal. Thus, because of the physiological similitudes induced by BH, only relative hemodynamic changes were considered, to avoid possible bias.

In conclusion, the results of this study highlight similar time-course patterns for cardiodynamic variables in maximal dry-body BH and immersed-body BH. This analogous diving reflex kinetic was nevertheless more pronounced when BH was performed in the fully-immersed condition. Whole body immersion, and mainly face immersion, seems to have an important role in the magnitude of the diving reflex, but not in the kinetics.

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Conflict of interest The authors declare that they have no conflict of interest.

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