Involuntary breathing movements improve cerebral oxygenation during apnea struggle phase in elite divers

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Dujic Z, Uglesic L, Breskovic T, Valic Z, Heusser K, Marinovic J, Ljubkovic M, Palada I. Involuntary breathing movements improve cerebral oxygenation during apnea struggle phase in elite divers. J Appl Physiol 107: 1840–1846, 2009; doi:10.1152/japplphysiol.00334.2009.—We investigated whether the involuntary breathing movements (IBM) during the struggle phase of breath holding, together with peripheral vasoconstriction and progressive hypercapnia, have a positive effect in maintaining cerebral blood volume. The central hemodynamics, arterial oxygen saturation, brain regional oxyhemoglobin (bHbO2), deoxyhemoglobin, and total hemoglobin changes and IBM were monitored during maximal dry breath holds in eight elite divers. The frequency of IBM increased (by ~100%), and their duration decreased (~30%), toward the end of the struggle phase, whereas the amplitude was unchanged (compared with the beginning of the struggle phase). In all subjects, a consistent increase in brain regional deoxyhemoglobin and total hemoglobin was also found during struggle phase, whereas bHbO2 changed biphasically: it initially increased until the middle of the struggle phase, with the subsequent relative decline at the end of the breath hold. Mean arterial pressure was elevated during the struggle phase, although there was no further rise in the peripheral resistance, suggesting unchanged peripheral vasoconstriction and implying the beneficial influence of the IBM on the cardiac output recovery (primarily by restoration of the stroke volume). The IBM-induced short-lasting, sudden increases in mean arterial pressure were followed by similar oscillations in bHbO2. These results suggest that an increase in the cerebral blood volume observed during the struggle phase of dry apnea is most likely caused by the IBM at the time of the hypercapnia-induced cerebral vasodilatation and peripheral vasoconstriction.

breath hold; cerebral autoregulation; near-infrared spectroscopy; diaphragmatic movements

ELITE BREATH-HOLD DIVERS ARE extreme athletes trained to maintain the prolonged periods of hypoxia/hypercapnia, lasting up to 10 min (current world record is 10 min 12 s). Breath-hold diving is associated with a diving response comprised of bradycardia, decreased cardiac output (CO), peripheral vasoconstriction, and increased arterial blood pressure (AP) (15, 16). These cardiovascular adaptations are caused by the simultaneous activation of sympathetic and parasympathetic nervous systems (8, 10), with consequent reduction of oxygen in peripheral tissues and oxygen supply of the vital organs (brain and heart). Although the arterial oxygen saturation (SaO2) is reduced with the long breath holds, the compensatory increase in cerebral blood flow (CBF) could potentially offset the reduced SaO2 and maintain the cerebral tissue oxygenation. Near-infrared spectroscopy (NIRS) is the method that continuously and noninvasively monitors the cerebral oxygenation and cerebral blood volume (CBV). Regional oxygen delivery and consumption in the brain can be assessed by measuring the changes in oxyhemoglobin (bHbO2) and deoxyhemoglobin (bdHb), whereas the relative changes in CBV can be assessed by measuring the total hemoglobin (bTHb) in the absence of major changes in hematocrit (2, 3, 7, 23). Our laboratory has shown previously, using the transcranial Doppler (TCD) and NIRS techniques, that, despite the large increases in cerebral perfusion in the later phase of the breath hold, the regional cerebral desaturation may become a factor limiting the maximal breath-hold duration (18). Therefore, the cerebral oxygenation is reduced only slightly until the end of the breath hold at the expense of reduced peripheral blood flow and oxygenation.

Breath-hold diving research can be performed in the field (wet) head-out immersion and dry laboratory conditions, with and without face immersion in cold water. Under all of these conditions, diving response can be elicited, although the magnitude of observed cardiovascular changes may differ. Since the methodological approach used in this study was not compatible with the field conditions, we investigated the physiological mechanisms of interest in the model of dry apnea in the laboratory.

Maximal voluntary apnea is divided into two phases: the initial or easy-going phase that lasts until the physiological breaking point when the accumulated carbon dioxide (CO2) stimulates the respiratory drive (15), and the struggle phase, during which the subject feels a growing urge to breathe and shows progressive involuntary breathing movements (IBM) (5). Recently, our laboratory reported that the IBM are involved in restoration of the venous return by improving the inferior vena cava blood flow, leading to augmentation of the stroke volume (SV) and normalization of the CO (17). The influence of the IBM on cerebral oxygenation and CBV is presently unknown.

Therefore, in the present study, we tested the hypothesis that IBM, together with peripheral vasoconstriction and progressive hypercapnic cerebral vasodilatation, contribute to centralization of the blood volume, thus maintaining the cerebral oxygenation during the extended breath holds. To investigate this hypothesis, the CO, cerebral, muscle, and arterial oxygenation, as well as blood pressure and IBM, were measured during the maximal apnea in trained apnea divers.
METHODS

Subjects. Experimental group consisted of eight breath-hold divers (6 men and 2 women). At the time of the study, they were all apparently healthy. The demographic and lung function data of the study subjects are shown in Table 1.

Experimental procedures. All experimental procedures were performed in accordance with the Declaration of Helsinki on the treatment of human subjects and were approved by the Ethical Committee of the University of Split School of Medicine. Informed, written consent was obtained from each subject. All experiments were carried out in a climatized room in the morning hours. The participants arrived at the laboratory 30–45 min before the start of the experiments for instrumentation and explanation of the procedures. They had abstained from caffeine for at least 12 h and from food for at least 4 h. In each subject, the dynamic spirometry (Quark PFT, Cosmed, Rome, Italy) was evaluated in the upright posture. Measurement of body fat index, height, and weight was performed. After emptying their bladders, the subjects rested in the supine position for 30 min to ensure stabilization of the cardiovascular parameters. During testing, each participant performed one maximal breath hold. The subjects were instructed not to hyperventilate before the apnea.

AP and heart rate (HR) were assessed using a pneumatic cuff placed around the middle finger of the nondominant hand (Finimeter, Finapress Medical Systems, Arnhem, the Netherlands). SaO2 was measured continuously by pulse oximetry (Poet II, Criticare Systems, Waukesha, WI), with the probe placed on the middle finger of the dominant hand.

From the continuous blood pressure measurement, the arterial pulse wave was analyzed, and changes in left ventricular SV were computed from the pulsatile systolic area. We employed the improved method of Wesseling using the Modelflow program (model-based measurement method based on a nonlinear, three-element model of the input impedance of the aorta) (13). This methodology detects rapid changes in SV accurately (compared with inert-gas rebreathing) during leg crossing, with and without muscle tension (24). CO was computed as SV times HR, and total peripheral resistance (TPR) was calculated as the quotient of mean AP (MAP) and CO.

A pneumatic respiratory belt, located around the chest at the level of the xiphoid process, was coupled to a differential pressure transducer (Prignitz Mikrosystemtechnik, Wittenberge, Germany) and was used to monitor the IBM. An additional reservoir was connected to the pneumatic system, which avoided any constriction of the thoracic cage during chest expansion. This method for IBM assessment was superior to the approach utilized in our laboratory’s previous study (17), and it enabled more detailed analysis of the IBM properties.

Local tissue oxygenation levels were recorded from the muscle and brain using a NIRS unit NIRO-200 (Hamamatsu Photonics KK, Tokyo, Japan). The first pair of emitting and detecting optodes of the NIRS unit was placed to the left side of the forehead, while the second pair was attached over the participant’s left calf muscle. An interoptode spacing of 4 cm was used to separate the emitting from the detecting sensor. The optodes were held within a black rubber housing that maintained constant optode spacing. The housing was affixed with tape. This minimized the loss of the near-infrared light from the recording field, as well as the intrusion of light from the environment. The full assembly was further secured with an elastic bandage that encircled the head and lower leg.

Levels of oxygenated hemoglobin, deoxygenated hemoglobin, and total hemoglobin were continuously sampled by the NIRS unit at a rate of 6 Hz. These parameters are expressed in micromoles as a change from zero.

Data acquisition and analysis. Analog signals were sampled at 1,000 Hz and stored on a personal computer using a PowerLab 16S data acquisition system (ADInstruments, Castle Hill, Australia). The time points of interest were the periods between 30 and 60 s before the onset of apnea and last 15 s of an easy-going phase of the breath hold. During the apnea struggle phase, we averaged three IBM at the start, in the middle, and at the end. In addition, data were collected during 60 s of postapnea recovery period.

Throughout the struggle phase, duration, amplitude, and frequency of IBM were analyzed. Duration was calculated as the base width of the IBM curve (in seconds). The amplitude was derived from the height of the IBM curve and was expressed as arbitrary units. Average frequency of the IBM was calculated from the total duration of three consecutive IBM for the observed time period and extrapolated as numbers of IBM per minute.

Latencies between the onset of the IBM and the peaks in MAP and change (Δ) in bTHb tracings were determined at the beginning, middle, and the end of the struggle phase. Time delays were measured for three consecutive IBM for each part of the struggle phase and averaged. The beginning of the IBM was defined as the onset of deflection in the respiratory belt recording.

Statistics. Using Statistica 7.0 software (Statsoft, Tulsa, OK), comparisons between changes of variables from the control value were first tested with nonparametric Friedman analysis of variance. In case of a significant difference, the Wilcoxon signed rank test was applied for the particular comparison. Differences between time delays from onset of IBM until the rise in MAP vs. onset in IBM and increase in ΔbTHb were compared using Mann-Whitney U-test. The level of significance was set at P < 0.05.

RESULTS

Apnea performed by the eight participants lasted, on average, 240 ± 51 s. The struggle phase, during which the IBM can be observed, occupied 47.0 ± 14.1% of total apnea duration. During this phase, on average, 27.1 ± 8.1 IBM per diver were detected.

Analysis of the IBM properties is shown in Table 2. During the struggle phase, the IBM duration significantly decreased, while their frequency progressively increased. The amplitude of IBM remained unchanged throughout the struggle phase.

Analysis of the individual responses during apnea (Fig. 1) revealed that the fluctuations caused by the movements of diaphragm muscle during IBM precede fluctuations of MAP, HR, and SV. Moreover, fluctuations in brain hemoglobin levels also had the same pattern as the fluctuations of IBM, but that was not observed in hemoglobin measured in the calf muscle. Analysis of time latency between the onset of individual IBM and fluctuations in MAP and brain hemoglobin levels revealed that IBM occurs first and increase in MAP is second and is followed by the change in the level of brain hemoglobin (Fig. 2, Table 3, individual recordings shown in data supplement; the

Table 1. Demographic and baseline lung function in eight breath-hold divers

<table>
<thead>
<tr>
<th>Metric</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>28 ± 4</td>
<td>22–33</td>
</tr>
<tr>
<td>Height, cm</td>
<td>184 ± 7</td>
<td>170–190</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25 ± 2</td>
<td>22–28</td>
</tr>
<tr>
<td>Body fat index, %</td>
<td>18 ± 11</td>
<td>5–36</td>
</tr>
<tr>
<td>FEV₁, %predicted</td>
<td>115 ± 13</td>
<td>92–131</td>
</tr>
<tr>
<td>FVC, %predicted</td>
<td>131 ± 15</td>
<td>112–154</td>
</tr>
<tr>
<td>FEV₁/FVC ratio, %</td>
<td>90 ± 9</td>
<td>74–100</td>
</tr>
</tbody>
</table>

BMI, body mass index; FEV₁, forced expiratory volume in first second; FVC, forced vital capacity. Spirometric values were estimated by Cosmed software (Quark PFT, Cosmed, Rome, Italy). Body fat index was calculated by Jackson and Pollock three-site method measurement.
There were no significant changes in the time delays between different parts of the struggle phase of the apnea. The magnitudes of responses of hemodynamic parameters and blood SaO₂ are summarized in Table 4. During the breath hold, the arterial blood saturation was reduced to 81.5/12.7%. MAP increased continuously throughout apnea, with the progressive rise occurring during the struggle phase. The SV fell down to 64% of its baseline value at the end of the easy-going phase, but started to normalize during the struggle phase, and reached 90% of its initial value at the end of apnea. The HR remained unchanged until the middle of the struggle phase, when it reduced discretely toward the end of apnea. These almost opposite trends of changes in the HR and SV caused the CO, after the initial drop, to remain constant throughout the apnea and stabilized 70% of the baseline value. The TPR increased rapidly after the onset of apnea and remained almost doubled throughout the breath hold.

The levels of oxygenated hemoglobin in the brain showed an increase in concentration during the major part of apnea (Fig. 1 and Table 5). Decline in bHbO₂ concentration can be seen in the last part of the struggle phase, while the bdHb concentration increased continuously throughout apnea. bTHb concentration also rose during the breath hold, especially in the late struggle phase, when >60% of the entire bTHb increase occurred, indicating an increase in CBV. Concentration of oxygenated hemoglobin in the calf muscle decreased progressively during apnea, in contrast to the similar magnitude of increase in deoxygenated hemoglobin concentration, resulting in an unchanged concentration of the total hemoglobin in calf muscle throughout the apnea (Table 5).

**DISCUSSION**

This study is the first to report that IBM during the struggle phase of the dry breath hold are followed by the simultaneous phasic fluctuations of the cerebral oxygenated hemoglobin. The brain oxygenation fluctuations were also paralleled with the fluctuations of MAP, HR, and SV, indicating that IBM may influence the central hemodynamic by maintaining the CO. This suggests that IBM, together with peripheral vasoconstriction-mediated centralization of the blood volume and progressive hypercapnia-induced cerebral vasodilatation, likely act to

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**Table 2. IBM characteristics during the struggle phase of apnea**

<table>
<thead>
<tr>
<th></th>
<th>Beginning</th>
<th>Middle</th>
<th>End</th>
</tr>
</thead>
<tbody>
<tr>
<td>IBM duration, s</td>
<td>3.9 ± 1.8</td>
<td>3.6 ± 1.0</td>
<td>2.6 ± 0.8*</td>
</tr>
<tr>
<td>IBM amplitude, AU</td>
<td>101.9 ± 8.9</td>
<td>100.0 ± 4.1</td>
<td>99.7 ± 5.7</td>
</tr>
<tr>
<td>IBM frequency, min⁻¹</td>
<td>10.7 ± 3.7</td>
<td>13.7 ± 4.6*</td>
<td>19.9 ± 7.1*</td>
</tr>
</tbody>
</table>

Values are means ± SD for all subjects completing the study. IBM, involuntary breathing movements; AU, arbitrary units. *P < 0.05 vs. the preceding stage of the struggle phase.
maintain the cerebral oxygenation throughout the struggle phase, thus prolonging the maximal apneic time.

The impact of IBM on cerebral oxygenation and blood volume. In this report, we investigated the influence of IBM on cerebral oxygenation during the struggle phase of apnea. We used NIRS to monitor the cerebral tissue oxygenation, which depends on changes in blood flow, SaO₂, cerebral metabolism, and arterial/venous vascular beds partitioning. In our laboratory’s previous study, we found that, during the apnea period, skeletal muscle oxygenation decreased earlier than the cerebral oxygenation (18). This observation was also confirmed in the present study, supporting the hypothesis of centralization of blood volume toward the brain and heart under such conditions. Dynamic cerebral autoregulation is a mechanism that maintains CBF relatively constant, despite the changes in MAP in the range of 60 – 150 mmHg (21). However, the nature of this mechanism has recently been challenged when the spontaneous fluctuations of CBF that corresponded to simultaneous changes in MAP were found (1, 19, 27). In the present study, our data imply that the IBM-induced sudden oscillations in MAP were paralleled by the oscillations in brain oxygenation and blood volume, suggesting incomplete cerebral autoregulation under these conditions. Our analysis of time relationship between these parameters revealed that IBM occurs first and is followed by the increase in MAP and, last, by the change in brain hemoglobin levels (Fig. 2, Table 3), suggesting a causal relationship between the three parameters.

The most distinct increase in bTHb occurred during the struggle phase (~60% of the total increase detected during apnea), which is not proportional to the average duration of this phase (~45% of total apnea time). Since the IBM are the hallmark of the struggle phase, it can be inferred that they are responsible for the additional increase in CBV observed during this period. In addition, a significant increase in bHbO₂ level was observed until the middle of the struggle phase, and it may help to prolong the total apnea time. Therefore, the IBM impose as the most likely mechanism that, in addition to redistribution of the blood to the central nervous system caused by the massive sympathetic activation (12) and CO₂-induced cerebral vasodilation, works to raise the CBV and to maintain the cerebral oxygenation during the struggle phase of apnea. Afterwards, close to the end of the breath hold, an evident drop in bHbO₂ occurs, possibly due to the prevailing regional cerebral desaturation, despite the compensatory mechanisms. The cerebral oxygen desaturation, together with intense hypercapnia, finally determines the end of the breath hold.

Table 3. Time delay between onset of IBM and peaks in MAP and ΔbTHb for three parts of the struggle phase

<table>
<thead>
<tr>
<th>Time to MAP peak, s</th>
<th>Time to ΔbTHb peak, s</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beginning</td>
<td>2.3 (1.6–3.0)</td>
<td>4.1 (3.4–4.8)</td>
</tr>
<tr>
<td>Middle</td>
<td>2.8 (1.7–3.8)</td>
<td>5.3 (3.9–6.7)</td>
</tr>
<tr>
<td>End</td>
<td>2.3 (1.6–3.0)</td>
<td>4.3 (3.4–5.2)</td>
</tr>
</tbody>
</table>

Values are means with 95% confidence intervals in parentheses. MAP, mean arterial pressure; ΔbTHb, change in concentration of total hemoglobin in the brain. P values present the differences between time delays from onset of IBM until the rise in MAP vs. onset of IBM and increase in ΔbTHb.
Table 4. Hemodynamic parameters and arterial blood oxygen saturation before, during, and following apnea attempts

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>End of Easy-Going Phase</th>
<th>Beginning of the Struggle Phase</th>
<th>Middle of the Struggle Phase</th>
<th>End of Apnea</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>THb, change in concentration of total hemoglobin</td>
<td>7.4±2.4*</td>
<td>8.6±2.5*</td>
<td>14.8±8.0*</td>
<td>20.4±10.5*</td>
<td>33.7±7.0*</td>
<td></td>
</tr>
<tr>
<td>HbO2, change in concentration of oxygenated hemoglobin</td>
<td>0</td>
<td>3.7±3.3*</td>
<td>4.4±4.2*</td>
<td>7.2±5.0*</td>
<td>3.9±6.3</td>
<td>11.2±5.8*</td>
</tr>
<tr>
<td>dHb, change in</td>
<td>0</td>
<td>3.7±2.3*</td>
<td>4.2±2.2*†</td>
<td>7.6±4.0*†</td>
<td>16.5±12.2*</td>
<td>2.8±1.1*</td>
</tr>
<tr>
<td>SV, ml</td>
<td>97.4±22.1</td>
<td>62.7±22.9*</td>
<td>71.9±23.4*</td>
<td>80.1±21.4*</td>
<td>87.3±32.7†</td>
<td>111.7±23.3*</td>
</tr>
<tr>
<td>CO, 1 × min⁻¹</td>
<td>7.3±1.9</td>
<td>5.0±2.0*</td>
<td>5.4±2.0*</td>
<td>5.7±1.9*</td>
<td>5.6±2.0*</td>
<td>8.8±2.1*</td>
</tr>
<tr>
<td>TPR, AU</td>
<td>14.9±6.0</td>
<td>29.4±24.0*</td>
<td>28.6±23.7*</td>
<td>29.3±25.0*</td>
<td>31.4±26.1*</td>
<td>13.0±5.9*</td>
</tr>
</tbody>
</table>

Values are means ± SD for all subjects completing the study. SaO₂, arterial oxygen saturation; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance. *Differences are reported between baseline values vs. intra-apneic changes and recovery (P < 0.05). †Differences between the end of an easy-going phase and various parts of struggle phase (P < 0.05).

The respiratory contractions were also analyzed previously in untrained or moderately trained subjects during breath holding that lasted only ∼1 min (26). The authors reported that diaphragmatic electromyography showed little or no muscle activity during the initial easy-going phase of the breath hold, but IBM became more frequent and intense in the latter, struggle phase until the breath hold breaking point (26). In the present study, like prior studies, large interindividual differences in achieving the long breath holds. 

Different proportions of struggle phase in total apnea time may be explained by an unknown mechanism, which acts to increase the amplitude of the IBM (26). In the present study, large interindividual differences in duration of the struggle phase were noted (ranged from 28 to 68% of the total breath-hold duration), but this did not affect the maximal duration of the breath hold. Different proportions of struggle phase in total apnea time may be explained by an interindividual variability in achieving the long breath holds.

**IBM-induced cardiovascular changes.** As anticipated, the cardiovascular changes observed in the present study were typical for dry breath holds (Fig. 1). Initially, AP dropped due to an increased intrathoracic pressure with subsequent reduction in venous return, SV, and CO, as shown previously by others (9, 20). Measurements of the muscle sympathetic neural activity during the breath holding in our laboratory’s recent study (12) revealed a fivefold increase in muscle sympathetic neural activity during maximal dry apnea in elite divers. However, in the later stage of the breath holding, this increase in sympathetic activity was not associated with a concomitant increase in TPR. Indeed, in the present study, we found no further increase in TPR during the struggle phase. It is very likely that the sympathetically mediated vasconstriction has been opposed by the direct vasodilator effect of hypoxia (14) and/or hypercapnia (22). Therefore, the gradual rise in the AP, which was, after the initial drop, observed throughout the apnea, is probably due to the partial restoration of the CO possibly via IBM-induced venous return (17) that is backed by the sympathetically maintained peripheral resistance. This mechanism is further supported by the observation that fluctuations in the AP appeared soon after the onset of IBM and stopped after the end of apnea. The HR can be either unchanged during dry breath holds or reduced as a part of the diving response. Here, we observed a reduction in the HR, which also contributed to diminished CO. This suggests that either parasympathetic input to the heart prevails over the sympathetic input under this condition, or that sympathetic efferent nerve activity can be differently expressed in different tissues.

**Methodological considerations.** Introduction of an additional group of divers with respiratory muscle paralysis would further confirm the causal relationship between the IBM and improved brain perfusion. However, due to ethical issues and technical difficulties, we were unable to perform such a procedure. Additionally, apnea attained under those circumstances would likely elicit different physiological responses than the “normal” apnea.

Table 5. Changes of tissue hemoglobin levels measured by near-infrared spectroscopy

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>End of Easy-Going Phase</th>
<th>Beginning of the Struggle Phase</th>
<th>Middle of the Struggle Phase</th>
<th>End of Apnea</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔHbO₂, change in concentration of oxyhemoglobin</td>
<td>0</td>
<td>3.7±4.4*</td>
<td>4.4±4.2*</td>
<td>7.2±5.0*</td>
<td>3.9±6.3</td>
<td>11.2±5.8*</td>
</tr>
<tr>
<td>ΔdHb, change in concentration of deoxyhemoglobin</td>
<td>0</td>
<td>3.7±2.3*</td>
<td>4.2±2.2*†</td>
<td>7.6±4.0*†</td>
<td>16.5±12.2*</td>
<td>2.8±1.1*</td>
</tr>
<tr>
<td>ΔTHb, change in concentration of total hemoglobin</td>
<td>0</td>
<td>7.4±4.7*</td>
<td>8.6±5.2*</td>
<td>14.8±8.0*†</td>
<td>20.4±10.5*</td>
<td>33.7±7.0*</td>
</tr>
</tbody>
</table>

Values are means ± SD in μM for all subjects completing the study. ΔHbO₂, change in concentration of oxygenated hemoglobin; ΔdHb, change in concentration of deoxygenated hemoglobin; ΔTHb, change in concentration of total hemoglobin. *Differences are reported between baseline values vs. intra-apneic changes and recovery (P < 0.05). †Differences between the end of an easy-going phase and various parts of struggle phase (P < 0.05).

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CEREBRAL OXYGENATION DURING APNEA STRUGGLE PHASE IN HUMANS

One limitation that most techniques that monitor the cardiovascular parameters, such as the CO, have in common is that they do not provide the continuous assessment of cardiovascular changes in a beat-to-beat manner (for example, dye or thermodilution). The continuous monitoring of the SV can be performed noninvasively by impedance cardiography, ultrasound, and by pulse-wave analysis. However, with pulmonary hyperinflation occurring after maximal inspiration, the use of ultrasound is complicated by locating the appropriate recording window, while the motion artifacts remain a problem in impedance cardiology. Therefore, we used the Modelflow method that is based on computation of an aortic flow waveform from finger by simulating the nonlinear three-element model of the aortic input impedance (13). However, even this method is somewhat limited, since the model assumes a normal aortic valve and unaffected transmural aortic pressure that may change by the maximal pulmonary hyperinflation. During maximal apneas, pulmonary volume is slightly below the total lung capacity, but it remains nearly unchanged throughout the breath hold. Nevertheless, van Lieshout et al. (25) have shown very reproducible continuous measurement of SV by the noninvasive Modelflow method and ultrasound in patients with instantaneous fluctuations in SV during arrhythmias. Our laboratory has used this method for assessment of the relative SV changes in several recent studies (6, 17, 18).

In our previous report investigating IBM, we have used the respiratory belt that enabled a reliable detection of the respiratory movements, but a detailed analysis of individual IBM in terms of duration, amplitude, and area under curve was not possible (17). This was significantly improved in the present study with the use of the pneumatic respiratory belt that allowed for such analyses.

Two methods that allow for the continuous monitoring of the CBF are the TCD and NIRS. Previously, we have described changes in TCD (determined by the middle cerebral artery blood velocity) and NIRS during the breath hold (18). In this study, we chose to measure relative changes in the CBV by NIRS, since it has been demonstrated that changes in cerebral oxygenation assessed by NIRS correlate well with the changes in CBF (4, 7, 23).

In conclusion, the present study is the first to report that IBM may increase cerebral oxygenation during the struggle phase of the dry breath hold. It is a result of the beneficial impact of IBM on central hemodynamics via partial restoration of the SV and CO. This effect of IBM on cerebral oxygenation may act, concomitantly with the peripheral vasoconstriction-mediated centralization of the blood volume and progressive hypercapnia-induced cerebral vasodilation, as a cofactor in prolonging maximal apnea time in these individuals exposed to extreme hypoxia/hypercapnia. Further studies are required to differentiate the relative contribution of each of these factors.

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GRANTS

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DISCLOSURES

No conflicts of interest are declared by the author(s).

REFERENCES


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