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ORIGINAL ARTICLE

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Effects of lung volume and involuntary breathing movements on the human diving response

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Abstract The effects of lung volume and involuntary breathing movements on the human diving response were studied in 17 breath-hold divers. Each subject performed maximal effort apnoeas and simulated dives by apnoea and cold water face immersion, at lung volumes of 60%, 85%, and 100% of prone vital capacity (VC). Time of apnoea, blood pressure, heart rate, skin capillary blood flow, and fractions of end-expiratory CO_2 and O_2 were measured. The length of the simulated dives was the shortest at 60% of VC, probably because at this level the build up of alveolar CO_2 was fastest. Apnoeas with face immersion at 100% of VC gave a marked drop in arterial pressure during the initial 20 s, probably due to high intrathoracic pressure mechanically reducing venous return. The diving response was most pronounced at 60% of VC. We concluded that at the two larger lung volumes both mechanical factors and input from pulmonary stretch receptors influenced the bradycardia and vasoconstriction, resulting in a non-linear relationship between the breath-hold lung volume and magnitude of the diving response in the near-VC range. Furthermore, the involuntary breathing movements that appeared during the *struggle phase* of the apnoeas were too small to affect the diving response.

Key words Apnoea · Face immersion · Bradycardia · Intrathoracic pressure · Pulmonary stretch receptors

Introduction

The human diving response is initiated by breath-holding (apnoea) and potentiated by face immersion in cold water. It has been found that cold receptors, especially on the forehead, initiate this potentiation (Schuitema and Holm 1988). The human diving response involves selective vasoconstriction and heart rate reduction (bradycardia). It has been shown by others that the reduction in cardiac output is not matched with the vasoconstriction, and, as a consequence, a progressive hypertension develops (Elsner and Gooden 1983; Kawakami et al. 1967; Lin 1982; Paulev et al. 1988). The response ensures that the brain and the heart, the most hypoxia-sensitive organs, receive an adequate amount of oxygen at the expense of less sensitive organs. There have been different conclusions regarding the oxygen conserving effects of the response (Elsner et al. 1971; Hong et al. 1971; Lin 1982; Moore et al. 1973), although the lowering of the heart rate in itself limits oxygen usage (Lin 1982).

Two distinct phases of a breath-hold have been identified, separated by the *physiological breaking point*, at which the elevated arterial carbon dioxide tension leads to excitation of the respiratory muscles (Lin 1982). The two phases have been called the "easy-going phase" and the "struggle phase" (Dejours 1965; Hentsch and Ulmer 1984), as during the latter the subject feels an urge to breathe and exhibits progressive involuntary breathing movements. No previous studies have addressed the question as to whether or not the diving response is altered or modified when the subject enters the struggle phase.

The amount of air inspired before breath-holding, i.e. the held lung volume, and the involuntary breathing movements during the struggle phase could possibly affect the diving response by mechanical and receptor-mediated mechanisms. The aim of our study was to investigate what effect different held lung volumes and involuntary breathing movements during the struggle phase had on the duration of the simulated dive and the diving response.

Methods

Subjects

People with experience of breath-hold diving have been shown to be more likely to reach the physiological breaking point than

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inexperienced people (Hentsch and Ulmer 1984). Hence active breath-hold divers or underwater-rugby players were selected as subjects. A group of 17 healthy men of a mean age of 24 (SEM2.5) years, volunteered for the study. Their physical training averaged 2–3 times a week, and all were non-smokers (one was taking snuff).

Measurements

Lung volumes were measured using a spirometer (Spirolite 201, Vise Medical Co, LTD, Chiba, Japan). The end-tidal fractions of CO_2 and O_2 ($F_{ET}CO_2$ and $F_{ET}O_2$) at the end of breath-holds were measured with a dual CO_2 and O_2 analyser (Engström Eliza Duo CO_2/O_2 analyser, Gambro Engström AB, Bromma, Sweden), connected to the mouthpiece on the spirometer. Breathing movements were recorded by pneumatic chest bellows, connected to an amplifier and an analogue recorder. The duration of the easy-going phase was determined by identifying the first involuntary breathing movement. Heart rate and arterial blood pressure were continuously measured using a photoplethysmometer (Ohmeda 2300 Finapres, Ohmeda, Madison, Wis., USA) connected to the middle finger. Skin capillary blood flow in the thumb was continuously measured using a laser Doppler flowmeter (Advanced Laser Flowmeter 21, Advance Company Ltd., Tokyo, Japan). The laser Doppler technique measures the velocity of moving red blood cells. It has been shown that if the cross-sectional areas of the capillaries remain constant, the velocity measured will be proportional to the flow (Holloway and Watkins 1977). Blood pressure, heart rate, and capillary blood flow values were continuously stored in a computer as mean values for each 5-s interval. Breath-holding times were measured with a chronometer.

Experimental procedure

The experiments were conducted in conformity with the principles of the Declaration of Helsinki. When the subject arrived at the laboratory he was given a demonstration of the instruments and thoroughly informed about the procedures. He was told to avoid hyperventilation before breath-holding and neither to swallow nor to exhale during breath-holding. He was also instructed to refrain from unnecessary movements during the tests to facilitate the determination of the physiological breaking point, and to hold his breath for as long a time as possible.

After a recording of vital capacity (VC) while standing, the subject assumed a prone position on a mattress with his head on a pillow placed above a water container and with his forearms on each side of the container. Water temperature was maintained at 9.5–10.5°C, and room temperature at 20.5–24.5°C. The sensors were connected to the subject, after which three measurements of VC in the prone position were made.

Each subject performed three series of apnoeas, differing only in the amount of air inspired: 55%–60%, 80%–85%, and 95%–100% of prone VC. In all series, the subject performed one apnoea in air and three apnoeas with face immersion. Between the apnoea in air and the first apnoea with face immersion within a series, the subject rested for 15 min, and between the apnoeas with face immersion for 2 min. The subject learned to find the appropriate lung volume in the 10-min pause between the series. The series were performed in random order.

The continuous measurements commenced 120 s before the apnoea and continued until 120 s after the last apnoea with face immersion. The subject was told when 60 s remained before each apnoeic episode, 30 s before apnoea a nose clip was attached, and 15 s before the apnoea the pillow was removed by the investigator.

The procedures for performing and measuring lung volume were different in two groups of subjects. One group consisting of 9 subjects expired through the spirometer to their functional residual capacity by just relaxing their thorax muscles, they then inhaled to the predetermined lung volume through the spirometer. The remaining 8 subjects made a maximal expiration to their residual volume and then put in the spirometer mouthpiece and inhaled to the appropriate lung volume. They also made a maximal expiration through the spirometer at the end of apnoea. The first 9 subjects did not expire through the spirometer, and no $F_{ET}CO_2$- or $F_{ET}O_2$-values were measured.

If any of the subjects inhaled an inappropriate amount of air, that particular apnoea was excluded or transferred to another test series if possible. During apnoea in air, the subject held his head just above the water, and during apnoeas with face immersion he immersed his face including the chin and forehead. After each apnoea the investigator replaced the pillow under the subject's head and dried his face with a towel.

Data analysis

The average $F_{ET}CO_2$ and $F_{ET}O_2$, breath-holding times, easy-going phase, and struggle phase duration for the three lung volumes in the apnoeas with face immersion were compared statistically. The absolute values of the cardiovascular parameters (systolic and diastolic blood pressure, capillary blood flow, and heart rate) were compared among the three lung volumes. The following periods were analysed: the initial 20 s of apnoea, the period 30–60 s after the beginning of apnoeas, the 30 s before and after the physiological breaking point, the last 20 s of apnoeas, and the first 30 s after apnoeas. For all statistical evaluations, paired students's *t*-test with Bonferroni correction for multiple comparisons was used. The level used for significance was $P < 0.05$.

Results

Inspiration to the appropriate lung volume was easily learned by the divers, and only 13 of 204 recorded apnoeic episodes were excluded.

The effect of lung volume

Figure 1 shows the average durations of breath-holding time, easy-going phase, and struggle phase for the apnoeas with face immersion using the three different lung volumes. Breath-holding time, easy-going phase, and struggle phase duration at 60% of prone VC were significantly shorter than at the two larger lung volumes. There were no differences in breath-holding time, easy-going phase, and struggle phase duration between 85%

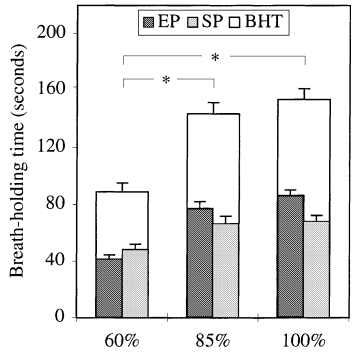

Fig. 1 Effect of lung volume expressed as percentage of vital capacity on the duration of breath-holding (*BHT*), easy-going phase (*EP*), and struggle phase (*SP*). Values are means with SEM for 17 subjects. * $P < 0.05$

and 100% of prone VC. Although breath-holding times for the larger lung volumes were longer, the $F_{ET}CO_2$ and $F_{ET}O_2$ (0.07% and 0.11%, respectively) after the apnoeas with face immersion did not differ among the different lung volumes (Table 1).

Figure 2 shows the continuous record of arterial blood pressure at the different lung volumes from 90 s before to 60 s after the start of the apnoeas with face immersion, and over the breaking point of the apnoeas. During the first 20 s of the apnoeas, there was a relative hypertension at 60% of prone VC, a brief hypotension followed by an increase in pressure at 85% of prone VC, and a marked hypotension at 100% of prone VC. The hypotension at 100% of prone VC was significantly different from the responses at the other two lung volumes. These initial differences were followed by similar changes in blood pressure at all lung volumes. At the breaking point of the apnoeas, the blood pressures at 85% and 100% of prone VC were higher than that at 60% of prone VC.

The capillary blood flow during apnoeas with face immersion at 100% of prone VC was significantly higher than at 60% of prone VC (Fig. 3). The pattern of the capillary blood flow response, however, was similar at all lung volumes. After a transient reduction followed by a rise, there was a continuous reduction of flow throughout the apnoeas.

Heart rate during apnoeas with face immersion at 60% of prone VC was significantly lower than at 85% of prone VC (Fig. 4). During recovery after breath-holding at 60% of prone VC, the heart rate was significantly lower than during recovery after breath-holding at 85% and 100% of prone VC. The pattern of the heart rate response, however, was similar at all lung volumes.

The diving response during the struggle phase

This part of the study was based on the results using 85% and 100% of prone VC, where the easy-going phase was sufficiently long for reliable analysis without influence of the adjustments at the onset of the diving response. No differences in the development of the cardiovascular parameters during the struggle phase compared to the easy-going phase were found in the pooled data from 85% and 100% of prone VC. Both during the easy-going phase and the struggle phase, there was a linear increase in blood pressure, a constant capillary blood flow level, and a stable diving bradycardia (Fig. 5).

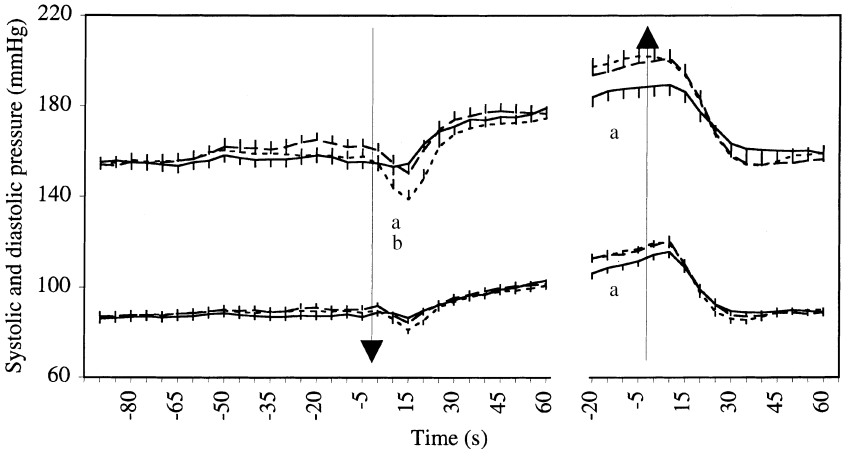

Fig. 2 Effect of lung volume expressed as percentage of vital capacity (VC) on systolic and diastolic blood pressures during apnoeas with face immersion. Values are means with SEM for 17 subjects. *Arrows* indicate the start and the end of apnoeas. [a] $P < 0.05$ between 60% and 100%, and [b] $P < 0.05$ between 85% and 100%. *Solid line* 60% VC, *dashed line* 85% VC, *dotted line* 100% VC

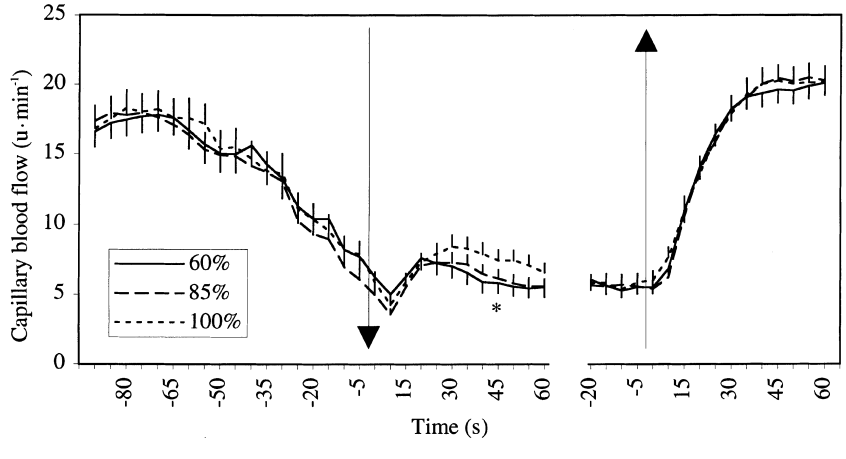

Fig. 3 Effect of lung volume expressed as percentage of vital capacity on skin capillary blood flow during apnoeas with face immersion. Values are means with SEM for 17 subjects. *Arrows* indicate the start and the end of apnoeas. * $P < 0.05$ between 60% and 100%

Table 1 Composition of expired air after breath-holds with 60%, 85% and 100% of vital capacity (*VC*). n = 8, $F_{ET}CO_2$, $F_{ET}O_2$ End tidal fractions of CO_2 and O_2

	60% of VC		85% of VC		100% of VC	
	mean	SEM	mean	SEM	mean	SEM
$F_{ET}CO_2$	0.074	0.001	0.074	0.003	0.072	0.003
$F_{ET}O_2$	0.113	0.005	0.108	0.005	0.111	0.005

Discussion

The effect of lung volume

This study showed that there was an effect of lung volume on both the duration of the simulated dive and diving response. The shorter breath-holding time and easy-going phase at 60% of prone VC may be explained by a faster build-up of CO_2 in the alveoli at the small lung volume, leading to a more rapidly rising arterial CO_2 tension, triggering breathing movements at an earlier point. This is supported by the finding of similar levels of $F_{ET}CO_2$ at the breaking point of breath-holding at the different lung volumes, in spite of different breath-holding times. This was also in accordance with several other studies (Hong et al. 1970, 1971; Sterba and Lundgren 1985, 1988), which would indicate that the end of an apnoea occurs at a given tension of CO_2 in the arterial blood (Sterba and Lundgren 1985, 1988) and/or that the arterial-alveolar CO_2 exchange decreases as the breath-hold proceeds (Hong et al. 1970, 1971). It has been suggested that a maximal inspiration leads to the longest breath-holding time (Lin 1982). However, the breath-holding time did not differ between 85% and 100% of prone VC in the breath-hold divers in this study.

During the first 20 s of apnoea there was a transient arterial hypotension depending on held lung volume. This was probably due to the elevation of intrathoracic pressure at apnoea with a large lung volume, leading to a decreased venous return. This would have resulted in a smaller stroke volume and a decreased cardiac output, and as a consequence, a hypotension would have developed (see Ferrigno et al. 1986; Paulev and Wetterqvist 1968; Paulev et al. 1988). Since it has been found that blood pressure, apparently due to the progressive vasoconstriction, is a function of breath-holding time (Hong et al. 1971; Lin 1982), the blood pressures at the end of apnoeas with face immersion at 85% and 100% of prone VC were more elevated than those at 60% of prone VC.

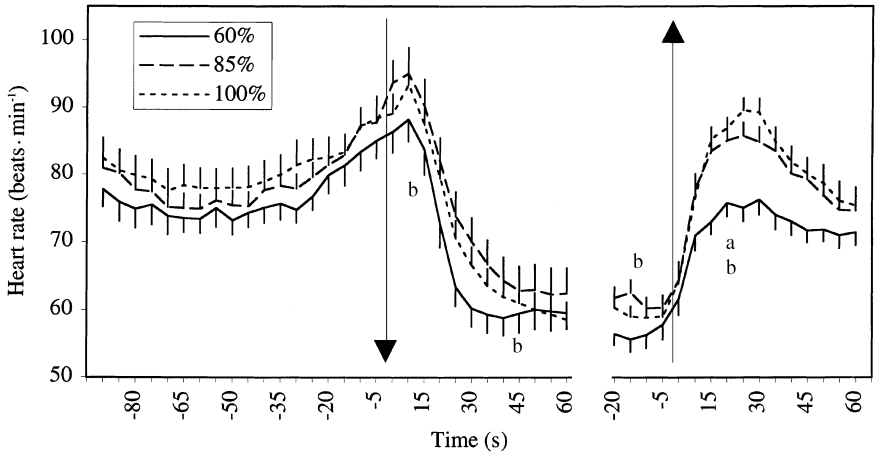

Fig. 4 Effect of lung volume expressed as percentage of vital capacity on heart rate during apnoeas with face immersion. Values are means with SEM for 17 subjects. *Arrows* indicate the start and the end of apnoeas. [a] $P < 0.05$ between 60% and 100% and [b] $P < 0.05$ between 60% and 85%

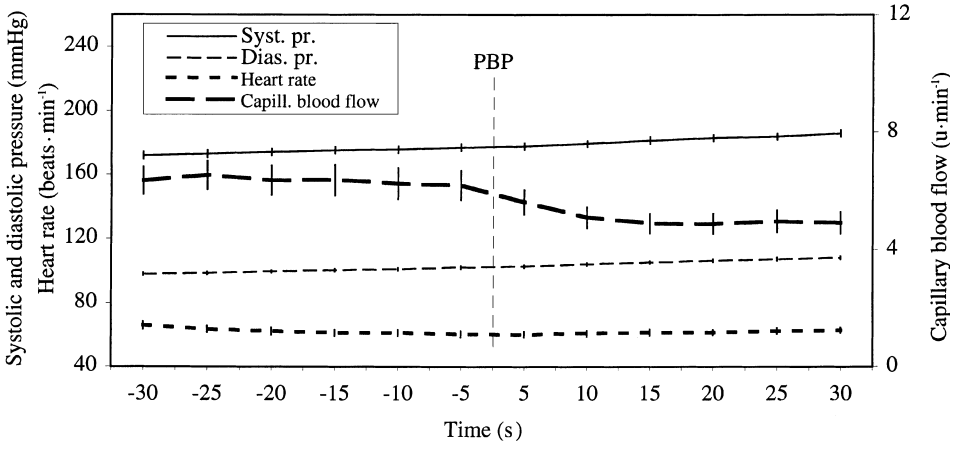

Fig. 5 Development of the diving response during the easy-going phase and the struggle phase. Values are means with SEM for 17 subjects. The *vertical dashed line* indicates the physiological breaking point (*PBP*)

It has been reported that after a small inspiration, the diving response will be completely developed (Angell-James and De B. Daly 1972), while breath-holding at large lung volumes seems to influence the cardiovascular system by both a haemodynamic mechanism related to an increased intrathoracic pressure and by a non-vascular pulmonary stretch receptor mechanism. Based on previous studies, it may be assumed that:

1. When the breath-holds are performed at 60% of prone VC neither the slowly adapting nor the J-receptors are stimulated
2. At 85% of prone VC, the slowly adapting receptors are stimulated
3. At 100% of prone VC, the J-receptors are stimulated (Ferrigno et al. 1986; Kaufman et al. 1982; Shepherd 1981),

which could explain the results obtained in this study.

It has been found that at both 85% and 100% of prone VC, a reduced stroke volume is caused by a high intrathoracic pressure (Ferrigno et al. 1986; Linér 1994; Paulev et al. 1988). This will be partly compensated for by a relative increase in heart rate. It has been reported that reflex cardioacceleration and vasoconstriction are induced by the slowly adapting receptors (Shepherd 1981). At 85% of prone VC, these adjustments result in a bradycardia of lesser magnitude that at 60% of prone VC, together with a capillary blood flow of the same magnitude as at 60% of prone VC. Reflex cardiac slowing and vasodilatation are induced by the J-receptors (Shepherd 1981). At 100% of prone VC, this will oppose the effect of the reduced venous return on heart rate and result in a high capillary blood flow.

After the apnoeas with face immersion, the differences in heart rate among the lung volumes may have been caused by a larger expiration and more drastic changes in intrathoracic pressure at the end of apnoeas at larger lung volumes. However, the differences among the lung volumes persisted for more than 30 s, and a pressure effect would have been expected to be very brief. Alternatively, we speculate that the difference could have been caused by larger asphyxic stimuli after the longer apnoeas at 85% and 100% of prone VC. If certain tissues or parts of organs were isolated from the circulation during apnoea, the produced asphyxic stimuli, for example lactate, would not have been accessible to the arterial chemoreceptors until the recovery phase after the end of apnoeas. Also, buffered CO_2 in the blood could increase arterial partial pressure of CO_2 as haemoglobin is resaturated during breathing.

The effect of involuntary breathing movements

In this study, no differences were found between the easy-going phase and the initial 30 s of the struggle phase in the changes in blood pressure, heart rate, and capillary blood flow. The effect of the involuntary breathing movements on the intrathoracic pressure, and hence venous return, seems to have been too small to have had an influence on the cardiovascular system. This is in contrast to the effects of voluntarily performed Valsalva and Mueller's manoeuvres found by Paulev and Wetterqvist (1968) and Paulev et al. (1988), that had marked effects on the magnitude of the diving response.

In conclusion, this study would indicate that the human diving response is influenced by lung volume, by both mechanical and pulmonary stretch receptor effects, but that the involuntary breathing movements are too small to influence the diving response. A high intrathoracic pressure, reducing venous return, seems to prevent the diving bradycardia from being completely developed. Thus, the human diving response evoked by the conditions present in this study, would be most pronounced after a small inspiration. This would suggest that, in actual breath-hold diving, the influence of the initial lung volume on the diving response may be modified by compression of the lungs at depth. Input from pulmonary stretch receptors (J-receptors, leading to cardiac slowing) at 100% of prone VC would seem to result in a non-linear relationship between held lung volume and diving response in the near-VC range.

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