Original articles

Oxygen-conserving effect of the diving response in the immersed human

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Key words
Breath-hold diving, freediving, diving reflex, immersion, vasoconstriction, oxygen consumption

Abstract
(de Bruijn R, Richardson M, Schagatay E. Oxygen-conserving effect of the diving response in the immersed human. *Diving and Hyperbaric Medicine*. 2009;39(4):193-9.)
Introduction: The human cardiovascular diving response has been shown to have an oxygen-conserving effect during simulated breath-hold diving by apnoea with face immersion. However, it is not known if facial immersion enhances the response to the same extent as that in the diver with the body immersed and if this leads to oxygen conservation.
Methods: Seventeen subjects each completed a total of 12 apnoeas of fixed, near-maximal duration. Four series of three apnoeas were conducted: dry body with apnoea (DA), dry body with face-immersion apnoea (DFIA), immersed body with apnoea (IA), and immersed body with face-immersion apnoea (IFIA). Air and water temperatures were 23°C. Heart rate, skin blood flow, arterial blood pressure, arterial haemoglobin saturation, lung volume and end-tidal fractions of carbon dioxide and oxygen were recorded non-invasively.
Results: Face immersion led to a greater reduction in heart rate during apnoea, regardless of body immersion (DA–DFIA 9.3%, 95% confidence interval (CI) 3.54, 0.1; IF–IFIA 7.9%, 95% CI 4.8, 0.2). Both DFIA and DA resulted in skin vasoconstriction, which was more pronounced during DFIA (16%, 95% CI 8.4, 0.3). During body immersion, skin vasoconstriction was reduced considerably, and neither IA nor IFIA reduced blood flow further. Mean arterial pressure increased more in the immersed condition than on dry land. Arterial saturation remained higher after DFIA (0.4%, 95% CI 0.2, 0.01) and IFIA (0.4%, 95% CI 0.4, 0.01) series, suggesting an oxygen-conserving effect of the more powerful diving response associated with face immersion.
Conclusion: We conclude that the oxygen-conserving effect of the diving response in the immersed diver is the same as that observed in the dry, horizontal, simulated diving model.

Introduction

Breath-hold diving, from here on referred to as diving, leads to a series of cardiovascular adjustments called the 'diving response'. The most pronounced adjustments are bradycardia and selective peripheral vasoconstriction.[1,2] The neural stimuli initiating the diving response are derived both from the apnoea and from stimulation of facial cold-receptors, e.g., by immersion.[3,4] Most studies of human diving abilities and associated reflexes have been based on laboratory studies, allowing a controlled environment and advanced techniques. A model used by many laboratories to simulate diving is apnoea with face immersion.[4,5] Studies with the prone subject performing apnoeas with the face either immersed or in air, i.e., causing a more or less pronounced diving response, have revealed that the diving response has an apnoea-prolonging effect and that the arterial oxygen (O_2) store is conserved during apnoea both during rest and exercise.[6–8] Oxygen conservation results from a reduced blood flow in tissues tolerant to hypoxia and lower myocardial O_2 consumption during bradycardia and leads to a slower O_2 depletion in the lungs.[9–11]

The impact of face immersion depends on the temperature difference between the skin and the water.[5] In a natural diving situation, however, the diver's entire body is often constantly immersed in cool water, in addition to the face immersion occurring during the apnoeas. Comparisons of heart rate responses have been made between horizontal apnoeas in air and warm water at 34°C.[12,13] The freediver in temperate regions will likely be diving in waters of 20–25°C with the use of a wetsuit, and with the face uncovered. A cooling of the body will result in a cold-induced vasoconstriction, which may have a negative effect on the diving response and possibly abolish O_2 conservation. Sterba and Lundgren showed that, compared to breath-holding while sitting in air, simultaneous vertical body and face immersion in cold water (20°C) reduced breath-holding time considerably but was accompanied by a strong bradycardia, while breath-holding in warm water (35°C) lengthened the breath-holding time, but without bradycardia.[14] They explain these results by an increased metabolic rate and respiratory drive at 20°C due to chilling. Paulev showed that, during continuous body immersion, apnoea without facial immersion resulted in a bradycardia similar to that found while breath-holding with facial immersion with the body in air.[15] However, no direct comparison was allowed between the two stimuli in that study, as the constant body immersion was done in 22°C water, and facial immersion in 15°C water, a situation likely to induce a more powerful response.[5]

An interesting observation during apnoea and facial immersion in 10°C water, in combination with immersion of the forearm in 10°C water, was that the bradycardia from the diving response had priority over the tachycardic response induced from the arm chilling.[16] This speaks for a maintained response with body immersion. However, in an already cool environment with reduced facial temperature, the effects of facial immersion may be reduced, decreasing the response magnitude.[5] Ferrigno and associates observed a diving response during immersion and exposure to increased ambient pressure, but its magnitude was not compared to the responses of the same individuals in air.[17]

Thus, it is not known to what extent O_2 conservation applies to the immersed diver. The present study, therefore, investigated the development of the diving response in the immersed human with and without facial immersion, with specific regard to its effect on O_2 conservation.

Methods

We compared the diving response during apnoeas (A) and face-immersion apnoeas (FIA) of the same duration, in horizontal dry-body (D) and immersed-body (I) conditions, respectively. A difference in arterial haemoglobin saturation after apnoea and face-immersion apnoea was used as an indication of O_2 conservation.

SUBJECTS

Seventeen healthy subjects (3 females and 14 males) volunteered for the study. Mean (SD) age was 28.7 (7.3) years, height 178.4 (7.7) cm, weight 77.4 (10) kg and mean standing vital capacity was 4.96 (0.9) L. Fourteen of the subjects had some prior experience in diving but no ongoing training, and three subjects practised diving regularly but with a maximum of two hours per week. Two subjects smoked occasionally and two were snuff tobacco users.

EXPERIMENTAL PROCEDURE

The study complies with the Helsinki Declaration and with Swedish laws and ethical standards. All subjects signed a consent form after being fully informed of the experimental protocol, which had been approved by the regional human research ethics board at Umeå University. To prevent excessive strain, the apnoeic duration was set for each subject at approximately 15 s less than their individual maximum breath-hold time, based on a single pre-trial maximal apnoea performed without hyperventilation or facial immersion. The short-term training effect observed when performing serial apnoeas, in combination with the time reduction by 15 s, would thus ensure that each subject could perform all apnoeas at the same, predetermined, fixed duration.[18] The average (SD) apnoeic time performed during the experiments was 58 (10) s. The experiments consisted of 12 apnoeas in total, divided in four different series of three successive apnoeas spaced two minutes apart. Body conditions were constant across each series, while facial immersion refers to apnoeic periods. These series were:

- Dry-body apnoea (DA)
- Dry-body face-immersion apnoea (DFIA)
- Immersed-body apnoea (IA)
- Immersed-body face-immersion apnoea (IFIA)

To minimize order effects, the conditions in the series were alternated and the starting situations were weighted between subjects. The choice was made to limit the alternating sequences to four, thus limiting the times a subject needed to change outfit. The four sequences used were as follows:

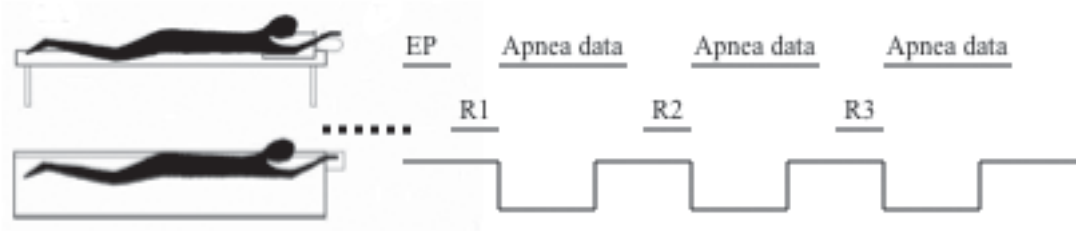
- DA, DFIA, IA, IFIA
- DFIA, DA, IFIA, IA
- IA, IFIA, DA, DFIA
- IFIA, IA, DFIA, DA

For the face and body immersions, mean (SD) water temperature was 23.1(0.4)°C, and the mean air temperature was 23.3(1.2)°C.

Vital capacity (VC) was measured in the standing subject at the start of all experimental sessions, in the prone position prior to the dry-body apnoeas, and in the immersed-body condition prior to the start of the immersed-body apnoeas.

In the dry-body series, subjects were outfitted with pneumatic chest bellows to detect respiratory movements and asked to

Figure 1
Position of the subject during apnoeas; dry body (top), immersed body (bottom); schematic shown of a series of three apnoeas with indications of different periods used for analyses (EP: effects of position; R1 – R3: reference periods for apnoeas 1 – 3); two-minute rest period between apnoeas



lie in a prone position on the bed with the head resting on a pillow on a board covering a small water container (Figure 1). In the immersed-body series, subjects wore 5 mm full wetsuits with the chest bellows placed on top. Once in a prone, floating position, a bar was placed across the tank in the water to support the legs, and a removable foam board supported the head of the subject between apnoeas. Probes for the pulse oximeter, photoplethysmometer and laser-Doppler flow meter were placed on the left hand, which was kept dry.

In all series the subjects relaxed for a minimum of 10 minutes prior to the beginning of each apnoea series. At two minutes before the first apnoea the data recording commenced and the subject was notified of the time remaining. At 30 s before each apnoea a nose clip was placed and with 10 s remaining a countdown began and the spirometer mouthpiece was offered to the subject, who continued to breathe normally and started the apnoea at the end of the countdown after a full exhalation followed by a deep, but not maximal, inhalation. The mouthpiece was removed during the apnoeas. Subjects were notified about the time at the half-apnoea point and by countdown for the last 10 s, and at apnoea termination they exhaled completely through the mouthpiece. Thus, recordings of inspired and expired lung volume, end-tidal fraction of oxygen ($F_{ET}O_2$) and end-tidal fraction of carbon dioxide ($F_{ET}CO_2$) were made. The face was dried immediately after each apnoea and the subject had a two-minute recovery period between apnoeas of a series. Recording continued throughout until two minutes after each third apnoea. At the end of the experiments, general comfort was evaluated for the dry- and the immersed-body conditions using a scale from 1 (very uncomfortable) to 10 (very comfortable) and a thermal comfort evaluation during immersion on a scale from 1 (very uncomfortable) to 10 (very comfortable) was made.

EQUIPMENT

VC, inspiratory (LV_{in}) and expiratory volumes (LV_{exp}) were measured using a spirometer (Vitalograph Compact II, Buckingham, England). A CO_2/O_2 analyser (Normocap Oxy, Datex Ohmeda, Helsinki, Finland) was connected to the mouthpiece of the spirometer to measure pre- and post-apnoeic end-tidal CO_2 and O_2. Heart rate (HR) and arterial oxygen saturation (S_aO_2) were measured continuously, with the averaging function set on 6 s, via pulse oximetry (Biox 3700e, Ohmeda, Madison, USA). Mean arterial pressure (MAP) was measured using an automated finger photoplethysmometer (Finapres 2300, Ohmeda, Madison, USA). Skin blood flow (SkBF) was measured using a laser-Doppler flow meter (Advance Laser Flowmeter 21, Advance Company, Japan). Respiratory movements were registered with laboratory-developed pneumatic chest bellows connected to a pressure sensor. An analogue event marker marking the apnoeic time (from the last inspiration before to the start of the first expiration after the breath-hold), was stored together with other data using a data acquisition system (MP100A-CE, Biopac Systems Inc, USA).

DATA ANALYSIS

Subjects served as their own control. To study the effect of position, resting values were obtained from a 60 s period starting after 10 min in that position (Figure 1). Control values for HR, MAP, SkBF and S_aO_2 were obtained from the period 90–30 s before each apnoea. Continuous graphs for the period from 25 s before until 40 s after the apnoea were made for HR, MAP and S_aO_2 by re-sampling to obtain a mean value for every 5 s, and calculating relative changes from the control taken before each apnoea. For SkBF, continuous graphs of the absolute data were used, since control values were very different because of cold-induced

Table 1
Resting values (mean ± SD) of heart rate (HR), mean arterial blood pressure (MAP), skin blood flow (SkBF), arterial oxygen saturation (S_aO_2), end-tidal fraction of oxygen ($F_{ET}O_2$) and carbon dioxide ($F_{ET}CO_2$) for dry-body apnoea (DA), dry-body face-immersion apnoea (DFIA), immersed-body apnoea (IA) and immersed-body face-immersion apnoea (IFIA), and the pooled averages for the dry- and immersed-body conditions for 16 subjects
*** $P < 0.01$, † $P < 0.001$**

	HR	MAP	SkBF	S_aO_2	$F_{ET}O_2$ (%)		$F_{ET}CO_2$ (%)	
	(bpm)	(mmHg)	(ml·min^{-1}·100 g^{-1})	(%)	pre apnoea	post apnoea	pre apnoea	post apnoea
DA	69 ± 10	100 ± 14	5.8 ± 7	97.4 ± 1	16.2 ± 0.9	12.5 ± 1	5.0 ± 0.6	6.3 ± 0.4
DFIA	70 ± 8	103 ± 11	5.0 ± 8	97.6 ± 1	16.2 ± 0.9	12.4 ± 2	4.9 ± 0.6	6.3 ± 0.5
Δ DA-DFIA	-1.0 ± 2	-3.0 ± 1	0.9 ± 0.5	-0.2 ± 0.2	0.01 ± 0.1	0.1 ± 0.2	0.07 ± 0.07	-0.003 ± 0.06
IA	74 ± 10	96 ± 13	1.5 ± 3	97.9 ± 1	16.1 ± 0.9	10.7 ± 1.7	4.9 ± 0.6	6.6 ± 0.5
IFIA	75 ± 12	95 ± 14	1.9 ± 3	97.5 ± 2	15.8 ± 2.6	11.5 ± 1.8	5.0 ± 0.9	6.6 ± 0.5
Δ IA-IFIA	-0.6 ± 2	0.9 ± 4.1	-0.4 ± 0.5	0.5 ± 0.4	0.4 ± 0.3	-0.7 ± 0.2†	-0.12 ± 0.09	-0.02 ± 0.04
Drybody	70 ± 9	101 ± 12	5.4 ± 7	97.5 ± 1	16.2 ± 0.9	12.4 ± 1.5	4.9 ± 0.6	6.3 ± 0.5
Immersed	74 ± 11	96 ± 13	1.7 ± 3	97.7 ± 1	15.9 ± 2	11.1 ± 1.8	5.0 ± 0.8	6.6 ± 0.5
Δ Dry-imm	-4.6 ± 2*	5.5 ± 4.5	3.7 ± 1.4†	-0.2 ± 0.3	0.2 ± 0.2	1.3 ± 0.3†	-0.02 ± 0.1	-0.3 ± 0.08†

vasoconstriction during immersion. To compensate for the difference in duration of apnoeas between subjects the first 30 s and the last 15 s of the apnoeas were aligned for HR, MAP and SkBF, and S_aO_2 was aligned with the end of the apnoeas. To determine if there were differences between HR, MAP, and SkBF measurements, the last 15 s of apnoeas were analysed. For determination of differences in S_aO_2 a 20 s period that encompassed the nadir was analysed.

STATISTICAL ANALYSIS

Data were further analysed for differences between DA versus DFIA, IA versus IFIA and the pooled dry versus immersed conditions, and these differences are given where appropriate, including 95% confidence intervals (CI). Comparisons between apnoea and face-immersion apnoea in both body conditions, and between the pooled values for dry-body and immersed-body conditions were also made using 2-sided paired Student *t*-tests. Significance was accepted at $P < 0.05$.

Results

Testing of one subject was terminated due to cold discomfort, and no results from this subject were included. No signs of shivering were observed in any of the remaining subjects. Some individual apnoeas were excluded from analysis because of obvious recording lapses, but calculations always

Figure 2
Mean (SEM) changes from reference in (A) heart rate (*n* = 15); (B) mean arterial pressure (*n* = 11); and (C) absolute values for skin blood flow (*n* = 14) for dry-body and immersed-body conditions, averaged over 5 s periods; vertical dashes indicate the beginning and end of apnoeas
■ apnoea in air; □ apnoea with face immersion; * $P < 0.001$

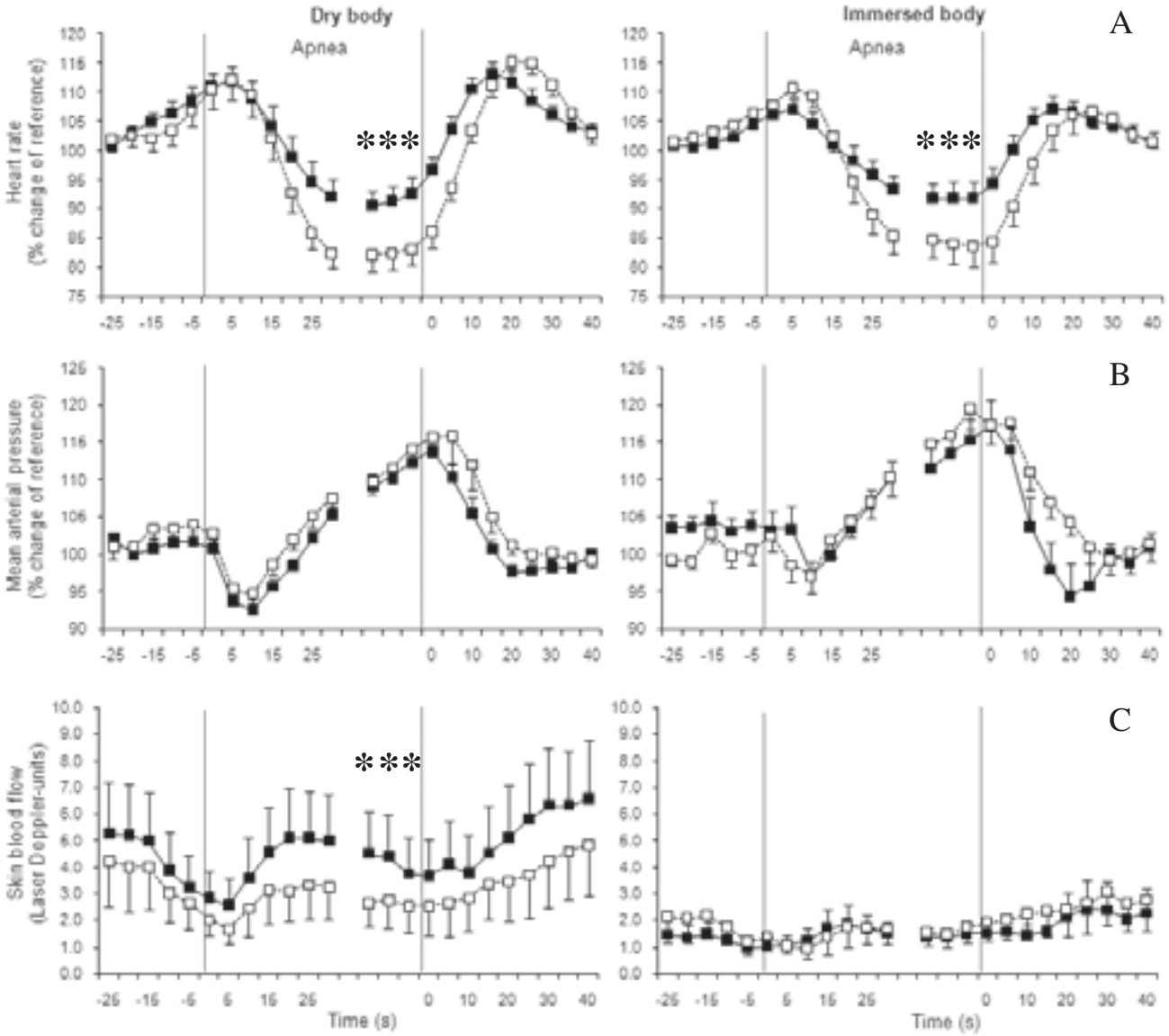
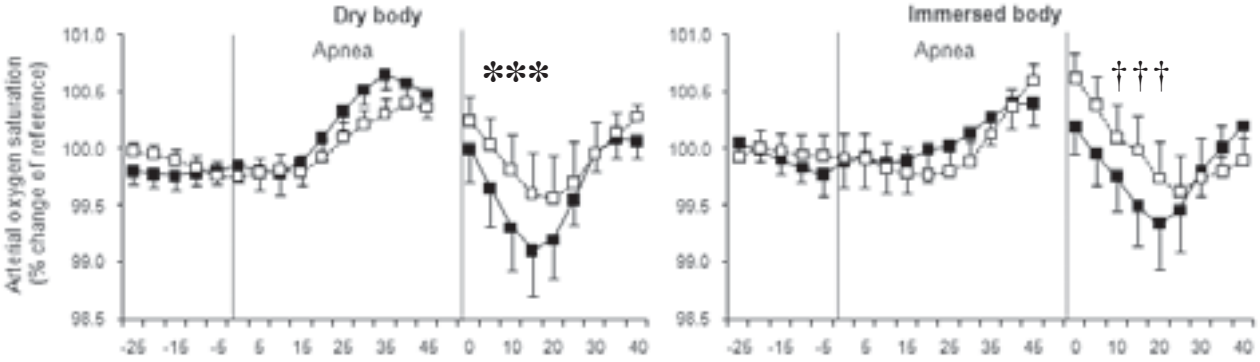


Figure 3
Mean (SEM) changes from reference in arterial oxygen saturation for 14 subjects for the dry-body and the immersed-body conditions, averaged over 5 s periods; the nadir corresponds with the end of the apnoea but is delayed due to circulation time; vertical dashes indicate the beginning and end of apnoeas; ■ apnoea in air; □ apnoea with face immersion; * *P* < 0.001; † *P* < 0.05



include at least two apnoeas per subject per series, as a basis for an individual mean. All subjects were able to perform their predetermined apnoea duration, except one who had some problems with the first apnoeas.

EFFECTS OF BODY IMMERSION

No differences in MAP or S_aO_2 were noted during any of the rest periods before the apnoeas, but HR was slightly elevated (4.6 bpm; 95% CI 3.5, 0.1; $P < 0.001$) and SkBF reduced (3.7 $ml.min^{-1}.100g^{-1}$; 95% CI 2.7, 0.09; $P < 0.001$) in the immersed-body condition (Table 1).

DIVING RESPONSE

The HR and MAP during apnoea showed similar patterns in all series. The bradycardia was more pronounced in the DFIA (18%) and IFIA (17%) series when compared to the DA (9%) and IA (9%) series (difference DA–DFIA 9.3%, 95% CI 3.5, 0.14; difference IA–IFIA 7.9%, 95% CI 4.8, 0.2; Figure 2A). The enhanced bradycardia during face immersion was consistent across the series of apnoeas in both the dry-body and immersed-body conditions. There were no differences in HR between the pooled dry-body and immersed-body series (-0.4%, 95% CI 3.2, 0.1). There were no differences in MAP between DA and DFIA (-1.6%, 95% CI 2.7, 0.1), or IA and IFIA (-3.7%, 95% CI 6.2, 0.2). However, the MAP increased more in the pooled, immersed-body series (6.8%, 95% CI 3.1, 0.1; Figure 2B). The SkBF patterns were similar in the dry-body series, but SkBF was reduced more in DFIA compared to DA (16.0%, 95% CI 8.4, 0.3). SkBF was lower in the immersed-body conditions compared to the dry-body conditions (-39.3%, 95% CI 28.2, 0.9; $P < 0.01$) and no differences in SkBF were found between IA and IFIA (2.7%, 95% CI 29.6, 0.9; Figure 2C).

ARTERIAL OXYGEN SATURATION

The more pronounced diving response during face immersion (Figure 2) was associated with less arterial desaturation, regardless of body immersion status (Figure 3). The S_aO_2 nadir, corresponding to the end of the apnoea, showed 50% less desaturation in DFIA compared to DA, and in IFIA compared to IA (absolute differences were -0.4%, 95% CI 0.2, 0.01 and -0.4%, 95% CI 0.4, 0.0 respectively; Figure 3).

RESPIRATORY PARAMETERS

The mean (± SD) VC for the prone position was 94 (4)% of the standing VC ($P < 0.001$) and, in the immersed position, it was 88 (6)% of the standing VC ($P < 0.001$); VC in the immersed position was 94 (7)% of that in the prone position ($P < 0.01$). There were no differences between series in LV_{in}, end-tidal fraction of O_2 ($F_{ET}O_2$) and end-tidal fraction of CO_2 ($F_{ET}CO_2$) at the last breath before apnoea. The relative inflation of the lungs was 61 (21) % in the dry-body condition and 64 (20)% in the immersed-body condition (Δ4.3%; 95% CI 4.9, 0.2; NS). At the end of the apnoea, there was no difference in LV_{exp} between all series, but differences were found in $F_{ET}O_2$ and $F_{ET}CO_2$. The $F_{ET}O_2$ was higher after IFIA compared to IA (Δ0.7%; 95% CI 0.4, 0.01; $P < 0.001$), while there were no differences between DFIA and DA, or in $F_{ET}CO_2$ (Table 1). In the pooled dry-body series, the $F_{ET}O_2$ was higher after apnoeas (Δ1.3%; 95% CI 0.5, 0.02; $P < 0.001$) while the $F_{ET}CO_2$ was lower compared to the pooled immersed-body series (Δ0.3%; 95% CI 0.16, 0.005; $P < 0.001$; Table 1).

COMFORT RATING

The subjects rated the immersed position in the tank less comfortable than the mean (SD) prone position on the

bed (6 (2) for tank and 8 (1) for bed; $P < 0.001$). Without the subject who interrupted tests due to cold discomfort, the group expressed no subjective cold problems (thermal confort mean score 8, range 5–10) despite the chilling effect on the skin evident from the reduced skin blood flow.

Discussion

From this study, one may conclude that face immersion during apnoea causes a more powerful diving response, reducing blood flow and O_2 consumption further compared to apnoea alone.[2,3] The results indicate that the diving response reduces oxygen consumption in the immersed diver in a similar manner to that in the dry, simulated diving model, which has not previously been shown. Even though both ambient and water temperature were 23°C, the diving response was more pronounced when apnoea was combined with face immersion, regardless of body immersion. This shows that the chilling effect of the water on the facial skin was sufficient to elicit a stronger diving response despite the strong pre-apnoeic vasoconstriction during body immersion. The enhanced diving response during face immersion was consistent throughout the series, showing that sufficient facial re-warming occurred in the two-minute intervals between apnoeas. For the diver, this suggests that it is important to expose the facial area involved in triggering the diving response for achieving maximal O_2 conservation. The main neural input from the face is through the ophthalmic branch of the trigeminal nerve, i.e., the forehead and eye region.[19] The face mask should, therefore, not cover all of this area (Figure 4). The slight increase in heart rate observed just before the apnoeas has been previously ascribed to an anticipatory response.[16] The initial increase in S_aO_2 during apnoea is related to the large inspiration.

The arterial haemoglobin desaturation was less pronounced during face immersion, in both dry- and immersed-body conditions, indicating that O_2 consumption was reduced by the increased diving response. This oxygen conservation would occur mainly via the following mechanisms:

- By reduced perfusion of organs that can withstand transient hypoxia by relying on anaerobic pathways, preserving most of the available oxygen for the use of the heart and brain;[1]
- By reduced myocardial O_2 demand through the reduction in HR, which further reduces oxygen usage;[9]
- The chilling of peripheral tissues may also in itself reduce local and thereby overall metabolism, as long as shivering is not induced.

While this study reveals an oxygen-conserving effect when apnoea is performed with the body immersed, thus adding body cooling and eliminating the effects of gravity compared to the dry model, it does not study the effects of pressure present during deep dives. It has been shown by earlier studies that cardiac performance during apnoeic exposure to increased ambient pressure at 20 metres' (m) depth is similar to that during apnoea at the surface.[17] Bradycardia was also shown to be similar during controlled dives in a diving tank at 10 m and 16 m depth.[20] Studies of the effects of lung volumes on the diving bradycardia show that the reduction in lung volume due to increased pressure at depth would likely enhance the diving response.[21] In addition, the diver in the field will in most situations encounter colder water when leaving the surface, which in turn should enhance the diving response.[5,22] Taken together, this supports the conclusion that the diving response conserves O_2 in the immersed diver to at least the same extent as in simulated dives, both during shallow and deep dives.

The reduction in SkBF before the apnoeas in the immersed-body condition, caused by strong vasoconstriction because of the chilling effect of the water, does not appear to have an effect on the magnitude of the overall diving response. Despite the constant low skin perfusion during body immersion, the blood pressure increased more during these apnoeas, compared to the dry-body condition, when normal skin perfusion periods preceded the apnoeas. This shows that other vascular beds, i.e., in the abdominal organs, may constrict more, and sufficiently compensate for the minor additional skin vasoconstriction during immersion, causing a similar effect on total peripheral resistance as during the non-immersed condition.

The resting HR in the immersed-body situation is slightly higher than in the dry-body situation, which may result from increased muscular tension due to cold and an increased effort from maintaining body position while floating. The latter explanation seems supported by the results from the questionnaire, which showed that subjects were less comfortable in the immersed situation due to factors not associated with chilling. This may also explain the differences between values in $F_{ET}O_2$ and $F_{ET}CO_2$ found after the apnoea, where increased muscular tension probably

Figure 4
A diver who exposes the facial area triggering the oxygen-conserving diving response (left) and a diver who covers all of the upper face with mask and hood (right), unlikely to benefit from the effect of chilling

caused an increase in O_2 usage and CO_2 production during body immersion. The increased metabolism does not seem to affect the extent of bradycardia during apnoea. This agrees with earlier findings of bradycardia during apnoeas with moderate exercise and swimming, reaching heart rates of at least as low levels as during inactive dives.[8,23]

Conclusions

Cold stimulation of the face plays an important role in the extent of the 'diving response' developed despite constant body immersion in cool water, and this leads to O_2 conservation in the diver. The oxygen-conserving effect of the diving response in the immersed diver is of the same magnitude as that observed in the dry, horizontal-body laboratory model used to simulate diving, which suggests that the simulated diving model is valid and can be used for further studies of diving-response functions in divers.

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