Facial Cold Receptors and the Survival Reflex “Diving Bradycardia” in Man

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Abstract We measured heart rate (HR), stroke volume (SV), systemic arterial blood pressure (BP), and mean arterial pressure (MAP) in 7 healthy volunteers in response to face immersion in water with concomitant breath-holding at different lung volumes. The subjects were at rest in the prone position. During breath-holding at total lung capacity (TLC), baseline HR (70 to 75 beats/min) fell by 10% within fractions of a second, both in the control preimmersion state when the head was surrounded by room air, and when it was immersed in water of 33°C. This response was associated with rises in MAP and in SV. Immersion of the face in 10°C water while breath-holding, was associated with a strong, negative chronotropic effect (22% fall in HR), which developed within 10 s. Breath-holding at functional residual capacity (FRC) reduced HR substantially only in 10°C water, and in contrast to that at TLC, the response was slowly developing with a latency of 10–15 s. All these reductions in HR were significant and accompanied by increases in BP and MAP. The strong, negative chronotropic effect of cold water was typically linked to a rise in SV. The study identified two temporal components of HR reduction to face immersion: a fast parasympathetic response dependent on the input from the high pressure baroreceptors, and a late response mediated, in all likelihood, by sympathetic efferent activity. Facial receptors sensitive to cold seem to be vital in the largest responses observed. The fast response to breath-holding with the face in water of neutral temperature was equal to that in air. Thus “diving bradycardia” is in fact a basic survival response independent of water.

Key words: apnea, baroreceptors, bradycardia, cold receptors, cardiac output.

A cardiovascular diving response, consisting of bradycardia and peripheral vasoconstriction in diving animals, can be mimicked in man by immersion of the face in water (OLSEN *et al.*, 1962; IRVING, 1964; ELSNER and SCHOLANDER, 1965; PAULEV, 1968; FUREDY *et al.*, 1983). During face immersion and the accompanying breath-holding, the blood flow is redirected to oxygen-sensitive organs, such as the brain, whereas less-sensitive organs must rely on anaerobiosis (ANDERSEN, 1966; FEDAK *et al.*, 1988). This is a so-called "oxygen-conserving response," which always has been considered of importance for diving homeotherms (for references see BLIX and FOLKOW, 1983).

Despite numerous reports, the exact determinants of the response to face immersion have been unsettled. Why the potential for successful resuscitation is increased when young victims of near-drowning have been in cold water has also yet to be settled. In the present study, we reexamined the nature of the cardiovascular events during face immersion in man performed in cross-combination of two water temperatures and two lung volumes of breath-holding. We confirmed the previous findings which showed a negative, chronotropic response during immersion of the face in water. We found that the afferent inputs from facial cold receptors largely determined the magnitude of HR reduction, and we also identified two temporal components of the HR reduction response.

METHODS

Subjects. Seven subjects aged 22–52 years volunteered for the study. The procedure complied with the rules of the Helsinki Declaration, informed consent was obtained, and the study was approved by the institutional ethical committee for human research. All volunteers were in physically good condition, and acquainted with the procedure described below.

The subjects were lying prone with their foreheads supported by an uninsulating band, and the face just above the water surface. The water bath bottom was provided with a snorkel and a separate water source, enabling us to fill the bath quickly with water when needed.

Recording and equipment. Four stainless steel strips (5 mm wide and 0.1 mm thick) were arranged around the neck and the chest wall of the subject, constituting the tetrapolar system of an impedance cardiograph. Stroke volume (SV), heart rate (HR), and cardiac output ($\dot{Q}$) were determined from the electrode signals with a Minnesota Impedance Cardiograph (304-A), and the electrocardiogram (ECG) was monitored with a San-Ei, Cardiosuper (2E 31A). The variables were continuously recorded and processed on-line by a microcomputer system (MIYAMOTO *et al.*, 1981, 1982). Triggered by the ECG R-wave, the computer sampled the first

time derivative (dZ/dt) and the basic transthoracic impedance (Z_0), at a rate of 200 times/s. The sampling was repeated for 5 cardiac cycles to obtain an average waveform with a favorable signal-to-noise ratio. The validity of the system was previously confirmed by the Fick principle, and also by a thermodilution method (MUZI *et al.*, 1985). In our subjects the whole body hematocrit was assumed to be 43%. All direct or computed data were recorded on a multichannel recorder (San-Ei, RECTI HORIZ-8K), except the intrapulmonic pressure, which was measured with a mercury manometer as the mouth pressure. With the glottis open, this mouth pressure represents the pressure of the pulmonary air during breath-holding against the mercury column. End-expiratory gas concentrations were measured with an expired gas monitor (San-Ei, 1H 21A) using the infrared (carbon dioxide) and the polarographic (oxygen) principle. The alveolar oxygen uptake and the carbon dioxide elimination were calculated according to the alveolar gas exchange equation in the period before breath-holding, and the net transfer of gases from the lungs to the body during 15-s breath-holding was calculated according to principles already published (PAULEV *et al.*, 1988). The arterial blood pressure was continuously measured in the finger non-invasively by applying a pneumatic cuff (Finapres, Ohmeda, U.S.A.). The mean arterial pressure (MAP) was calculated as the diastolic pressure plus 1/3 of the pressure amplitude.

Protocol. Just prior to breath-holding, the subject took a deep inspiration of normoxic room air (approximating the vital capacity, here called total lung capacity, TLC). The subject was asked to hold his breath for 15–20 s on inspiration, maintaining a mouth pressure of 10 to 20 mmHg on the mercury manometer, with his expiratory muscles, and then to breathe spontaneously for 1–2 min. Then the subject was instructed to hold his breath following a normal expiration for 10–15 s (at FRC). During breath-holding at FRC the subject relaxed, and thus showed an intrapulmonic pressure around zero, avoiding Valsalva and Mueller maneuvers.

The main protocol consisted of repeating the above breath-holding twice at TLC and twice at FRC. This was done in three different environments for the facial skin: room air; water at 33°C; and water at 10°C, regulated with ice cubes. For comparison, we also raised the water level to the ears of the subjects for 30–40 s, while they continued breathing through the snorkel. In the control period the subject breathed spontaneously and the facial skin was dried. The room temperature was 22–23°C.

Statistics. The level of significance was chosen as two-sided $\alpha=0.01$. The distribution of the variables was, with good approximation, shown to be normal. Changes of the variables from the preceding control period to a given maneuver were calculated as percentages in order to eliminate individual differences in absolute values of the control levels for HR, SV, and MAP. Accordingly, statistical comparisons were performed using a paired *t*-test, where $2p < 0.01$ was considered statistically significant.

RESULTS

Breath-holding

Breath-holding in air at TLC elicited a statistically significant reduction in HR (Table 1). A fall in HR was usually accompanied by a rise in MAP (Table 3). The average fall in HR for the group of 7 persons was 11% to 89% of control value

Table 1. Percentage change in heart rate from baseline (100%) during breath-holding (BH in air and in water at 33 and 10°C) and during immersion of the face in water while breathing spontaneously (FI).

	Air (%)	Water 33 (%)	Water 10 (%)
BH at TLC	89.0 (1.4)*	91.1 (0.8)*	77.8 (1.3)*
BH at FRC	101.2 (7.1)	96.1 (2.3)*	82.2 (1.7)*
FI		95.0 (0.6)*	85.0 (1.5)*

Each value represents a mean (standard error) of 14 observations from seven subjects.

*Statistically significant from baseline ($p < 0.01$).

Fig. 1. Original recording from a subject showing the short-lasting inspiratory (*I*) tachycardia immediately followed by a drop in HR during breath-holding at TLC in air. The FRC breath-hold is initiated by a normal expiration (*E*). Tracings from above are: stroke volume (*SV*), cardiac output ($\dot{Q}$), beat-by-beat HR, pneumotachogram (*Pn.*), and arterial blood pressure in mmHg.

(Table 1). The deep inspiration before TLC breath-holding caused a drastic rise in HR, followed by a steep fall toward 50 beats/min (Fig. 1). This negative chronotropic effect developed within a fraction of a second. Breath-holding in air at FRC had no significant effect on either heart rate (Table 1) or MAP (Table 3). SV changed on only a few occasions (Table 3). Just following breath-holding at TLC, the deep inspirations always elicited a positive chronotropic effect due to the respiratory work; this rise in HR was also found at the end of some FRC breath-holds followed by deep respirations (Fig. 1).

Breath-holding plus immersion

When the subjects kept the breath at TLC, while the water bath was elevated to the ear level (33°C), we found an average reduction of HR to 91.1%—similar to the 89% in air (Table 1). This reduction was equal to the previously obtained results (PAULEV, 1969) with 28 and 36°C water (Fig. 2). A negative chronotropic effect was accompanied by a rise in MAP at 33°C, and by a significant rise of SV at 10°C (Table 3). During breath-holding at FRC, our subjects reduced only HR to 96.1% in 33°C water (Table 1), whereas MAP increased to 105.3% (Table 3), and SV was unchanged compared to control (Table 3). However, face immersion in cold water (10°C) induced a highly significant HR reduction during breath-holding both at TLC and at FRC (Table 1). Estimation of the total peripheral vascular resistance (TPVR) from the ratio of MAP and $\dot{Q}$ from Tables 1 and 3 showed a rise in TPVR of 10–15%. The net transfer of oxygen over the pulmonary barrier tripled and that of carbon dioxide doubled during apneic face immersion at TLC (Table 2). Consequently, the alveolar gas exchange ratio ($R = \dot{V}_{\text{CO}_2}/\dot{V}_{\text{O}_2}$) fell (Table 2).

Face immersion with spontaneous breathing preserved.

A negative, chronotropic reaction to face immersion was found in all subjects (four examples shown in Fig. 3), when they continued their breathing through the tube in the water bath. The fall in HR was evident after a delay of 10–15 s (Bin

Table 2. Oxygen uptake and carbon dioxide output in a 15-s control period before breath-holding (control) compared to 15-s face immersion in 10°C water with breath-holding (BH) at TLC.

Subjects	$\dot{V}_{\text{O}_2}$		$\dot{V}_{\text{CO}_2}$		R	
	Control	BH	Control	BH	Control	BH
NIS	97	330	82	168	0.85	0.51
NAK	65	215	66	159	1.0	0.74
PEP	84	310	68	135	0.81	0.44

Oxygen uptake ($\dot{V}_{\text{O}_2}$) and carbon dioxide output ($\dot{V}_{\text{CO}_2}$) are in ml STPD per 15s. R denotes the alveolar gas exchange ratio.

Fig. 2. Decrease of HR from baseline as a function of water temperature during face immersion with spontaneous breathing preserved (upper curve) and during face immersion with breath-holding (lower curve). The present mean values are from 14 observations in 7 subjects. The earlier data are from PAULEV (1969), where each point is the mean value of 50 observations from 30 subjects. All vertical bars indicate S.E.M.

No. 3–4) and was sustained throughout most immersion periods in 10°C water (Fig. 3). In the cold water, the close location of ice cubes near the face was sometimes enough to reduce HR even before the immersion started. However, all the subjects showed a typical, delayed fall in HR even from a low control level (Fig. 3). We found a reduction of HR to 85% in 10°C water, and only to 95% at 33°C (Table 1). Such HR reductions were already established years ago (PAULEV, 1968, 1969). The present and earlier results are compared in Fig. 2. The HR reduction was evidently temperature dependent and more pronounced below 28°C (Fig. 2).

Our young subjects (INA 23 and ISH 22 years of age) had relatively large respiratory variations at rest, and they also demonstrated the largest negative,

Fig. 3. The fall in HR during 50-s immersion of the face in water during spontaneous snorkel breathing. Three of the subjects were immersed in both 33 and 10°C water, while subject INA only did the cold water test. Each BIN represents 5 s.

chronotropic effects, particularly in cold water face immersion (Fig. 3). Simultaneously with the fall in HR, the respiratory frequency increased from an average (S.E.M.) of 12 (1) to 23 (2) respiratory cycles per min ($n=14$). Since tidal volume also increased, the resulting hyperventilation was pronounced especially in the young subjects. During face immersion with breathing preserved in 33°C water, SV increased by an average (S.E.M.) to 107.3% (1.9) in the 7 subjects (Table 3), as compared to the 5% HR reduction (Table 1). Face immersion also led to an increase in TPVR—approximately 10% as estimated from the ratio of $MAP/\dot{Q}$ from Tables 1 and 3.

DISCUSSION

This study showed a negative, chronotropic effect of breath-holding at high lung volume, and a further potentiated effect by face immersion in cold water. When face immersion was performed with spontaneous breathing preserved, HR

Table 3. Percentage changes of stroke volume (SV) and mean arterial pressure (MAP) from baseline (100%) during breath-holding at TLC and FRC in air, and with the face immersed in water at 33 and 10°C. Face immersion with breathing preserved is shown below.

	BH air (%)	Water 33 (%)	BH water 10 (%)
TLC			
SV	103.1 (2.1)	101.2 (1.8)	118.4 (2.4)*
MAP	105.2 (0.8)*	107.4 (1.1)*	102.1 (1.2)
FRC			
SV	101.3 (1.6)	100.2 (0.9)	124.3 (2.3)*
MAP	100.1 (0.9)	105.3 (0.7)	101.4 (1.0)
Face immersion		33°C (%)	10°C (%)
SV		107.3 (1.9)*	97.4 (0.6)*
MAP		101.2 (0.6)	107.3 (1.8)*

Mean of double determinations in 7 subjects ($n = 14$). The standard error is in parentheses. *Statistically significant from baseline ($p < 0.01$). *Results from only subject PP ($n = 5$).

reduction displayed a different temporal pattern. The study thus implicates the presence of different mechanisms of HR decline due to breath-holding and to the activation of facial cold receptors, with a possible interaction. During breath-holding at TLC with and without face immersion, we found evidence of generalized vasoconstriction, since $\dot{Q}$ fell (i.e., a 10% increase in TPVR). This small vasoconstriction is not comparable to the substantial limb blood flow reduction elicited by Valsalva maneuvers (PAULEV, 1969) and hardly of consequence during skin diving. The small vasoconstriction found during face immersion with breathing preserved cannot be a high pressure baroreceptor-mediated phenomenon, but must be elicited from the inputs of facial cutaneous nerves.

Afferent pathways during breath-holding. Firstly, the deep inspiration and subsequent breath-holding at TLC stimulate the pulmonary stretch receptors, resulting in an immediate inhibition of the activity of vagal cardiac motoneurons. We consistently found a rapid HR acceleration, most likely due to a reduced activity of cardiac vagus. This HR acceleration was never observed during breath-holding at FRC, when the pulmonary stretch receptors were not stimulated. However, a reduced respiratory drive from the cerebral cortex or from the respiratory, medullary neurons due to stretching of the pulmonary receptors will also diminish the tone of the vagal cardiac motoneurons and thus contribute to the HR acceleration observed during inspiration (Fig. 1).

Secondly, an abrupt fall in HR and an increased SV at a negative intrathoracic pressure (i.e., the pressure in the pleural space minus the atmospheric pressure) occurs during breath-holding at TLC (Fig. 1). This observation agrees with the

Starling's law of the heart: a facilitated diastolic filling and venous return of blood to the right half of the heart will increase SV. With a short latency this must lead to a rise in systemic arterial pressure (MAP), which we and other investigators observed here (Fig. 1) and in the previous studies (KAWAKAMI *et al.*, 1967; PAULEV, 1968, 1969; WHAYNE *et al.*, 1972). The rise in MAP in turn becomes an adequate stimulus of the high pressure baroreceptors (aortic and carotid) which increase the tone of the vagal cardiac motoneurons, and thus explain the abrupt fall in HR (Fig. 1).

Efferent pathways mediating the bradycardia during breath-holding. A negative, chronotropic response during breath-holding alone occurred within fractions of a second. The almost instantaneous changes in HR in the present study is highly suggestive of neural efferent pathways being involved. HR responses mediated by the parasympathetic nervous system are very fast (WARNER and COX, 1962). Furthermore, atropine abolished the bradycardia evoked by breath-holding (GALLO *et al.*, 1988). Therefore, the fast-reacting vagal motoneurons are most likely to mediate the bradycardia during breath-holding.

Afferent pathways mediating the bradycardia during face immersion. Stimulation of the facial skin with cold without wetting (e.g., by cold air) during breath-holding induced a fall in HR similar to that which occurred when the face was immersed in cold water (KAWAKAMI *et al.*, 1967; PAULEV, 1968; HONDA *et al.*, 1987). Recently, SCHUITEMA and HOLM (1988) found a significant HR reduction during breath-holding with a bag of ice water applied to the forehead or to the eyes. Both areas elicited a more pronounced HR reduction than breath-holding alone. They found clear evidence of regional differences by facial cold exposure in eliciting the bradycardia. The forehead and the eyes are especially sensitive to cold, perhaps due to a high density of cutaneous cold receptors. The cold-induced bradycardia was not specific to the sensitive facial skin (HR fell by 7%), but was also observed when the same skin area of the hand and forearm (HR fell by 3.5%) were immersed in 15°C water (PAULEV, 1968). HONDA *et al.* (1987) found a 3% reduction in HR and in $\dot{Q}$ during facial cold stimulation with spontaneous breathing preserved, but 10–18% fall during facial cold stimulation with breath-holding. The above observations are explicable by cold stimuli to the thermal receptors of the skin, transferred through afferent pathways along the trigeminal nerve to the hypothalamic temperature area.

Efferent pathways during face immersion. During face immersion with spontaneous breathing preserved, the fall in HR developed after a time lag of 10–15 s. The relative fall in HR in warm water was only half as large as the rise in SV. Parasympathetic blockade (atropine sulfate) did not reduce the magnitude of HR reduction in 2 of 3 subjects studied (GALLO *et al.*, 1988). Accordingly, this late response could be due to either a slow inhibition of the sympathetic activity to the sinus node of the heart (TODA and SHIMAMOTO, 1968) or to other factors. During face immersion with breath-holding, the efferent pathway for the fast HR-reduction must be vagal cardiac motoneurons in the medulla.

Temperature receptors in the face. The HR reduction observed during face immersion with spontaneous breathing was also seen during face immersion with breath-holding (Fig. 2). At each water temperature the total HR response to facial immersion with breath-holding equaled the sum of the "breath-holding bradycardia" and the "cold bradycardia" (Fig. 2). Although the cold bradycardia has been well established in man (ANDERSEN, 1966; HONDA *et al.*, 1987; HONG, 1988; OLSEN *et al.*, 1962; PAULEV, 1968, 1969; SCHUITEMA and HOLM, 1988), GALLO *et al.* (1988) reported the lack of temperature dependency in the bradycardia during face immersion while breathing spontaneously. However, close examination of their data indeed reveals clear evidence of the temperature dependency (GALLO *et al.*, 1988; Fig. 2).

Other phenomena. Substantial reductions in $\dot{Q}$ must represent a fall in blood oxygen transport. In the present study, $\dot{Q}$ during breath-holding was not reduced more than 10% from the baseline level, and the oxygen uptake tripled due to the high lung diffusion capacity at TLC (PAULEV *et al.*, 1988, 1990). Therefore, in our subjects the utilization of oxygen for the whole body was considered not to decrease, contrary to what was claimed in diving physiology (OLSEN *et al.*, 1962; IRVING, 1964). We found one case of profound bradycardia (with one cardiac cycle of more than 2-s duration) during breath-holding at TLC in cold water, but hypoxia was totally ruled out by the high oxygen tension measured. The bradycardia was interpretable as due to a strong cold reflex from the facial skin. Similar low HR's have been observed in small children who have been victims of cold water near-drowning for periods of up to 40 min, but such low HR's are allied to those found during hypothermic surgery (HAYWARD *et al.*, 1984). Previously reported arrhythmias were seldom of serious consequence to $\dot{Q}$ or seldom malignant (PAULEV, 1969), but the incidence of malignant arrhythmias increased in cold water (HONG, 1988). These arrhythmias and bradycardia phenomena are not part of the "diving bradycardia." Arrhythmias are just provokable through cold receptors in the skin. Thus, bradycardia, as well as malignant arrhythmias leading to fibrillation or cardiac arrest, can be elicited by cold stimuli both in our everyday life and in surgical anesthesia.

HAYWARD *et al.* (1984) studied maximum breath-hold duration and diving bradycardia in cold water near-drowning. Peripheral cold stimulation decreased breath-holding capacity and facilitates drowning. They compared the average HR response of young persons submerged in water to the neck during breath-holding. Twenty subjects were exposed to each of 8 temperatures. The average HR of the group exposed to 15°C was reduced by breath-holding to 60% of control (52/87 beats/min), and that of another group exposed to 30°C was reduced to 73% (61/84 beats/min). Whether a 40% HR reduction in cold water is considered statistically significant from a 27% reduction in another group of subjects is not crucial to the conclusion; their findings do not support the postulate that the "diving bradycardia response" has an important role in the enhanced resuscitability of persons who have nearly drowned in cold water. Small children have relatively good prognosis

for resuscitation, which is consistent with their faster cooling rate in cold water.

BJERTNAES *et al.* (1984) also found a reduction in $\dot{Q}$, while systemic vascular resistance increased during face immersion with breath-holding; they assumed the myocardial oxygen demand to be reduced. The stroke work of the left ventricle was reduced during breath-holding in air, just as was $\dot{Q}$ (PAULEV *et al.*, 1988). In the present study a similar reduction in $\dot{Q}$ was found in air (Fig. 1), and also with the face in water we found an unchanged or slightly reduced $\dot{Q}$ during breath-holding.

Conclusion. The HR alterations during breath-holding with face immersion in water consist of at least three components: 1) a breath-holding bradycardia is mediated by increased vagal tone through the action of the carotid sinus; 2) a temperature-dependent "cold bradycardia" showing a delayed temporal pattern, elicited from cutaneous cold receptors; 3) an inspiratory tachycardia in the control and recovery phase due to stretching of the pulmonary receptors.

The "diving bradycardia" occurs in most species (FEDAK *et al.*, 1988), not only in emergency situations in the water, but also on land (HONDA *et al.*, 1987; PAULEV, 1968, 1969; PAULEV *et al.*, 1988, 1990). Breath-holding often takes place during the activities of everyday life both as a Valsalva maneuver (e.g., running, lifting, straining, etc.), and without a rise in intrapulmonic pressure. In all situations, the "diving bradycardia" in man represents a circulatory survival response, more than a genuine "oxygen-conserving diving reflex" confined to the water environment. A further HR reduction is elicited from cutaneous cold receptors. The generally accepted diving reflex is in fact two separate phenomena. Although the fast HR phenomenon preserves blood pressure and life, the slow temperature phenomenon can elicit malignant arrhythmia.

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