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CARDIOVASCULAR RESPONSES TO PARTIAL AND TOTAL IMMERSION IN MAN

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SUMMARY

1. Short-term cardiovascular effects of partial and total immersion of eighteen human subjects in the horizontal plane have been examined. Brachial arterial pressure, heart rate, forearm blood flow and respiratory movements were monitored simultaneously throughout the experiments. Forearm vascular resistance was calculated from the mean blood pressure and mean flow.

2. Total immersion, including the face, with breath-holding resulted in a $61 \pm 43\%$ increase in forearm vascular resistance with an associated $29 \pm 15\%$ reduction in forearm blood flow. The concurrent bradycardia was significantly different from the heart rate changes during breath-holding with the torso only immersed, or during total immersion with snorkel-breathing. Neither breath-holding in air or with only the torso immersed, nor total immersion with snorkel-breathing produced such a diving response.

3. Breath-holding, after several minutes of total immersion and snorkel-breathing, produced an attenuated diving response. It therefore appears that a full diving response can be obtained only when the apnoea commences at the moment of face immersion.

4. The present investigation supports the concept that in man face immersion is an essential predisposing factor for the diving response, and cortical inhibition of the respiratory centre is important for its initiation and maintenance.

INTRODUCTION

Paul Bert (1870) was the first to record that in animals such as the duck a marked bradycardia occurs upon immersion in water. A similar diving bradycardia has since been observed in many other vertebrates. Irving

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(1934) proposed that bradycardia is only one component of widespread reflex cardiovascular adjustments that take place upon immersion. The functional significance of such reflex adjustments, he suggested, is to conserve the limited oxygen reserve during the period of immersion, while maintaining normal perfusion through the cerebral and coronary vascular beds. To achieve this, the flow through peripheral and abdominal vessels must virtually cease. If the flow were only moderately reduced, the oxygen extraction by the tissues could correspondingly increase and no oxygen conservation would occur (Folkow, Nilsson & Yonce, 1967).

Investigation of the diving response in man has been less extensive than in animals and most studies have been concerned with changes in heart rate (Irving, Scholander & Grinnell, 1940; Scholander, Hammel, Le-Messurier, Hemmingsen & Garey, 1962; Craig, 1963; Harding, Roman & Whelan, 1965). Examination of changes in the peripheral circulation in the forearm and calf has been confined to situations where only the face has been immersed (Elsner, Garey & Scholander, 1963; Brick, 1966).

In the present investigation forearm blood flow, heart rate and blood pressure have been recorded during immersion of the whole body, with and without immersion of the face and with and without breath-holding, in an attempt to analyse the effects of these different procedures on the cardiovascular responses to immersion.

METHODS

The subjects were eighteen healthy males aged between 18 and 25 years. Two were experienced underwater swimmers.

A 3·7 m diameter pool containing water to a depth of 1·3 m was used. Subjects lay supine on a light aluminium stretcher supported on trestles, initially in the air above the water and then immersed with the uppermost portions of the body no more than 6 in. below the surface. Only shallow immersion was attempted so that the subjects would not experience difficulty in breathing. The face was either supported above the level of the water or immersed with the rest of the body. With immersion of the face, the breath was either held or the subject continued to breathe through a mouthpiece and snorkel tube. The water temperature was maintained at $34 \pm 0{\cdot}5°$ C. The laboratory air temperature was $24 \pm 1°$ C.

Forearm blood flow. Blood flow was measured in the right forearm by venous occlusion plethysmography. A pneumatic cuff was inflated around the wrist, to a pressure of 200 mm Hg, to exclude hand circulation. A second cuff around the upper arm was inflated intermittently to a pressure of 60 mm Hg to occlude venous but not arterial flow. The plethysmograph was a modified Whitney mercury-in-rubber strain-gauge consisting of a small plastic block into which were sealed the ends of two insulated copper wires 2 m long. A 35 cm loop of siliconized rubber tubing of wall thickness 0·8 mm and bore 0·5 mm containing an unbroken column of mercury was attached to the ends of the wires in the block. All conductive surfaces were sealed so that the instrument was completely insulated from the water. The plastic block of the strain-gauge was placed at the point of maximum convexity of the forearm and the tubing applied around the limb. The free end of the loop was fixed in place by an adjustable clamp to a pin extending from the plastic block. The tubing was always kept

slightly taut. The strain-gauge formed one arm of a low impedence Wheatstone bridge and the output was fed to a Beckman four-channel pen recorder.

Calibration was carried out by stretching the rubber tubing by known increments in a screw gauge and measuring the deflexion of the pen recorder.

Heart rate. The electrocardiogram was monitored by two precordial electrodes and an earth (Hardin *et al.* 1965). The precordial electrodes were mounted on a thin rubber sheet through which the leads were sealed. These two electrodes were placed over the sixth left intercostal space, and a rubber strip wound around the chest to ensure electrical contact between the skin and the electrodes. The rubber strip did not hinder the subject's respiratory excursions. An indifferent electrode was fixed to the left upper arm.

These electrodes provided an electrocardiogram with a strong R wave component, which was directed into a Beckman cardiotachometer coupling and the heart rate recorded on one channel of the pen recorder.

Respiration. Respiratory excursions were followed by means of a thoraco-abdominal stethograph and recorded on one channel of the recorder. The record served simply to differentiate periods of apnoea from those of normal respiration.

Blood pressure. In most experiments left brachial arterial pressure was measured by auscultation, using an anaeroid manometer, both with the subject in air and under water. Korotkow sounds were sufficiently distinct to make the accuracy of the method approximately ± 5 mm Hg. Blood pressure was recorded once every 30 sec during the resting phases and approximately once every 15 sec immediately before, during and after experimental procedures. The increased environmental pressure during immersion accounted for 5 mm Hg of the elevation in arterial blood pressure.

As a check on the auscultatory method, the brachial arterial pressure was recorded directly in one subject. A 21-gauge needle was inserted into the artery under local anaesthesia and connected by 60 cm sterilized, heparinized, saline-filled polyethylene tubing to a Statham pressure transducer. Since the transducer and cable could not be immersed in water, they were fixed above the pool on a 40 cm long steel post extending from the stretcher. In this way the subject's arm and the transducer remained in the same relative position independent of movement of the stretcher. The relative pressure changes produced during the various procedures were essentially the same as those obtained by auscultation.

Procedure. All experiments began outside the pool, where the electrocardiogram electrodes, thoraco-abdominal stethograph and pneumatic cuffs for both plethysmograph and blood pressure recording were fitted. The subject then entered the pool and lay supine on the stretcher above the surface of the water (Plate 1).

The right arm was supported by a rest in a slightly elevated position relative to the heart, and the strain-gauge applied to the forearm. The experiments which followed were divided into two series.

Series I

This series consisted of twelve experiments in which the subject breathed through the nose between procedures.

Breath-holding in air. After a 5 min control period, during which all parameters were recorded and the subject settled, instruction was given to hold the breath in 'moderate' inspiration for 1 min. Following this procedure, recording continued for a further 5 min to allow the parameters to return to base line.

Transfer to water. Recording stopped and the pneumatic cuffs were deflated. The stretcher and subject were then transferred to two lower trestles so that the torso was immersed but not the face. The head was supported by an adjustable rest and the torso was held under water by weight belts laid across the pelvis and ankles.

Breath-holding, face out. Following the transfer to water, records were obtained over a 5 min control period. Then the subject performed a 1 min breath-hold with the face still in the air but the torso immersed.

Breath-holding, face submerged. After a 4 min rest period a nose clip was applied, the subject inspired, the head rest was lowered, and light pressure on the subject's forehead produced total submersion of the face without voluntary effort by the subject. The subject was now totally submerged. After 1 min the face was brought out of water and supported again on the head rest.

Snorkel-breathing, face submerged. Following a further 4 min rest period the subject's face was again submerged for 1 min but during this time the subject breathed continuously through a short snorkel (internal volume 32 ml). This procedure was followed by a 4 min rest period with the face in air once more and the subject breathing through the nose without snorkel.

Transfer to air. The subject and stretcher were then lifted out of the water and placed on the higher trestles. Recording was continued for 2 or 3 min until shivering precluded accurate data.

Series II

A further five experiments were performed which were the same as Series I except that the subject breathed through the snorkel between procedures and the final snorkel-breathing, face-submerged procedure was modified. This modification was designed to test the effects of breathing with the face submerged for several minutes, followed by superadded apnoea of 1 min duration. After the 1 min of breath-holding, the subject remained totally immersed for several minutes and then the face was brought out of the water.

The base line mean for each parameter was calculated by averaging the readings during the first two of the 3 min before each procedure. The mean level of each parameter during the actual procedure was calculated from the readings taken during the last 30 sec of the procedure.

Mean blood pressure was calculated from the formula: $\frac{1}{3}$ (systolic pressure + 2 (diastolic pressure)).

Vascular resistance was calculated by dividing mean blood pressure by mean blood flow.

RESULTS

Series I

The mean results of the twelve experiments in this series are summarized in Fig. 1.

The variation in individual base line means precluded useful analysis of the changes during procedures by the usual standard deviation test. However, differences were readily apparent when changes in the level of a parameter relative to the subject's individual base line were analysed by two tailed paired *t* test. This test was also used to compare changes in each parameter between consecutive procedures. A result referred to as 'significant' in the following text therefore means 'significant by two tailed paired *t* test' (Hill, 1966).

Breath-holding in air. This produced a slight rise in heart rate, blood pressure and forearm blood flow, while the forearm vascular resistance decreased slightly. The only significant change was the rise in blood pressure (Table 1).

Transfer to water. Immersion of the body produced a significant increase in blood flow and blood pressure. Heart rate was unaltered.

Breath-holding, face out. The effect of breath-holding with the body immersed but the face out of water produced similar responses to breath-holding in air, except for a significant tachycardia.

Breath-holding, face submerged (Fig. 2). Breath-holding with simultaneous total immersion produced a rise in blood pressure similar to that seen with the two earlier procedures, but instead of a rise in heart rate there was now a bradycardia (-6 ± 15 %). This was not a significant

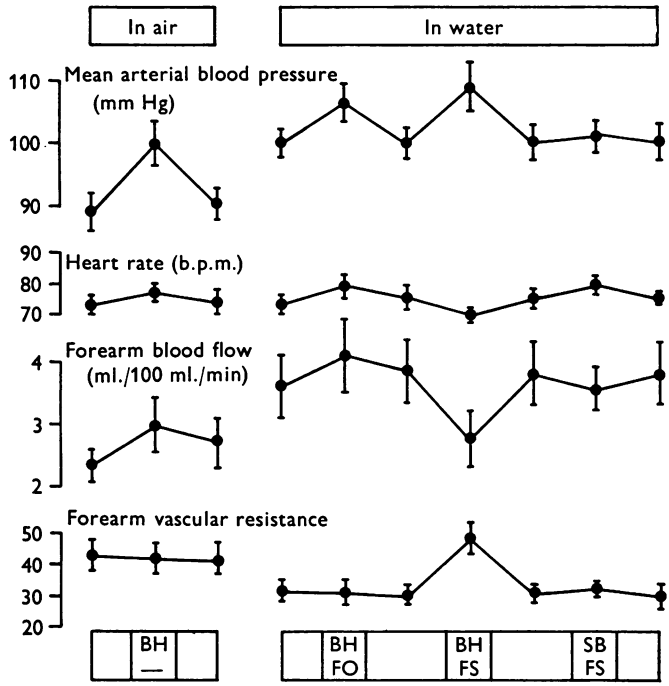

Fig. 1. Averaged results of Series I experiments. Vertical lines through each point represent the S.E. of the means.

Abbreviations: BH, breath-hold (1 min), FO, face out with torso immersed, FS, face submerged as well as torso, SB, snorkel-breathing, b.p.m., beats per minute. The increased environmental pressure during immersion accounted for 5 mm Hg of the elevation in arterial blood pressure.

change from the preceding base line, but was a significant fall when compared with the response elicited by the previous procedure. The most dramatic changes were a fall in forearm blood flow (-29 ± 15 %) and an increase in vascular resistance (61 ± 43 %).

Snorkel-breathing, face submerged. This 1 min period of total immersion and continuous snorkel-breathing produced no significant change. Moreover, the lack of response observed during this procedure was significantly different from the response obtained during breath-holding, face submerged.

Because of the inconsistent nature of the recordings following the

TABLE 1. Experiments of Series I

	In air Breath-hold		Transfer to water		In water: Breath-hold face out		In water: Breath-hold, face submerged		In water: Snorkel breathing, face submerged	
	% change with S.D.	$2P$	% change with S.D.	$2P$	% change with S.D.	$2P$	% change with S.D.	$2P$	% change with S.D.	$2P$
Mean blood pressure	12 ± 7	$< 0{\cdot}001$	11 ± 5	$< 0{\cdot}001$	7 ± 4	$< 0{\cdot}001$	9 ± 8	$0{\cdot}001 \rightarrow 0{\cdot}005$	1 ± 3	$0{\cdot}60 \rightarrow 0{\cdot}70$
Heart rate	7 ± 14	$0{\cdot}10 \rightarrow 0{\cdot}20$	1 ± 10	$1{\cdot}00$	9 ± 13	$0{\cdot}02 \rightarrow 0{\cdot}05$	-6 ± 15	$0{\cdot}10 \rightarrow 0{\cdot}20$	3 ± 8	$0{\cdot}20 \rightarrow 0{\cdot}30$
Forearm blood flow	23 ± 35	$0{\cdot}05 \rightarrow 0{\cdot}10$	56 ± 54	$0{\cdot}02 \rightarrow 0{\cdot}05$	12 ± 23	$0{\cdot}05 \rightarrow 0{\cdot}10$	-29 ± 15	$< 0{\cdot}001$	-7 ± 22	$0{\cdot}10 \rightarrow 0{\cdot}20$
Forearm vascular resistance	-3 ± 26	$0{\cdot}60 \rightarrow 0{\cdot}70$	-25 ± 22	$0{\cdot}05 \rightarrow 0{\cdot}10$	2 ± 24	$0{\cdot}80 \rightarrow 0{\cdot}90$	61 ± 43	$< 0{\cdot}001$	11 ± 21	$0{\cdot}40 \rightarrow 0{\cdot}50$

Twelve subjects, except the final procedure in which there were ten.
$2P < 0{\cdot}05$ implies significant by two tailed paired t-test.
$(n-1)$ degrees of freedom where n is the number of subjects.

transfer to air no statistical evaluation of the responses during this time was attempted.

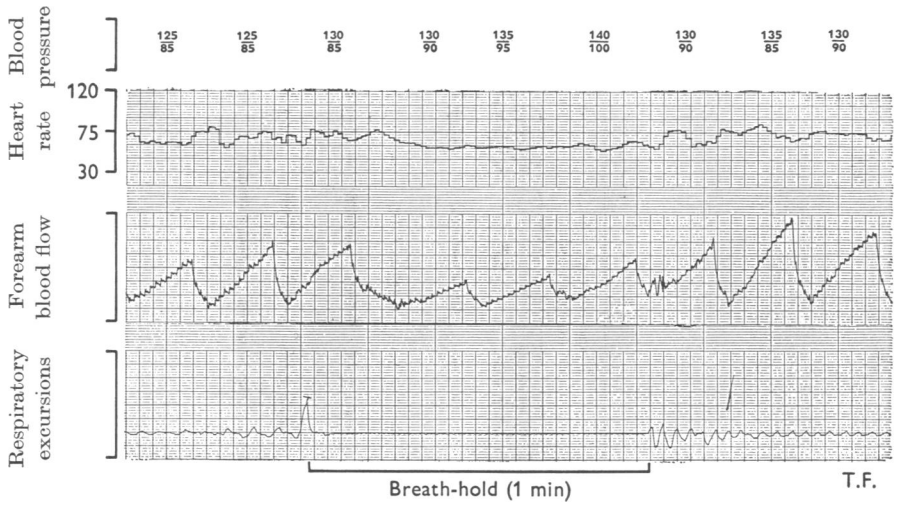

Fig. 2. Typical record obtained during breath-holding with face submersion to produce total immersion.

Series II

In these experiments in which the subjects breathed through a snorkel between procedures instead of through the nose, breath-holding in air produced a significant rise in blood pressure only as in Series I. The transfer to water produced a response similar to Series I, but the rise in blood pressure was not significant. Breath-holding with the face out of water evoked no significant response (Table 2).

Breath-holding with the face submerged produced significant changes in all parameters. The responses were identical with those obtained in Series I, except that now the bradycardia was significant.

Submersion of the face while respiration was continued through the snorkel caused a small but significant increase in blood pressure almost exclusively in the systolic element.

Breath-holding produced a further increase in mean blood pressure, but a reduction in heart rate. Forearm blood flow was reduced and vascular resistance increased, but none of these changes was significant. When compared with the preceding breath-hold, face submerged, the changes in blood flow and vascular resistance were significantly different. The reduction in blood flow and the rise in vascular resistance were both smaller during this final breath-hold procedure.

Variability between subjects. One of the subjects in Series II was an amateur competitive swimmer (J.P.). His responses were different from

TABLE 2. Experiments of Series II

	In air Breath-hold		Transfer to water		In water: Breath-hold, face out		In water: Breath-hold, face submerged		In water: S.B., F.S./ B.H., F.S.	
	% change with S.D.	2P	% change with S.D.	2P	% change with S.D.	2P	% change with S.D.	2P	% change with S.D.	2P
Mean blood pressure	9±6	0·02→0·05	4±3	0·05→0·10	8±8	0·05→0·10	8±4	0·01→0·02	15±12	0·05→0·10
Heart rate	−7±18	0·40→0·50	3±6	0·20→0·30	−8±13	0·10→0·20	−15±10	0·02→0·05	−13±15	0·05→0·10
Forearm blood flow	−21±33	0·20→0·30	27±12	0·005→0·01	−38±28	0·05→0·10	−51±11	0·001→0·005	−16±40	0·20→0·30
Forearm vascular resistance	62±85	0·10→0·20	−17±9	0·01→0·02	110±107	0·05→0·10	133±69	0·001→0·005	64±88	0·10→0·20

Same five subjects for all procedures. $2P < 0·05$ implies significant by two-tailed paired t test.

any other subject tested in these experiments. Whenever he held his breath, in air or water, face under or face out of water, he showed only one response, namely, a rise in blood pressure, a fall in heart rate and forearm blood flow and a rise in forearm vascular resistance.

DISCUSSION

The essential finding in this study was that total immersion, including the face, with breath-holding resulted in a marked diving response, i.e. a bradycardia and an increased forearm vascular resistance with an associated fall in forearm blood flow. Breath-holding either in air or with only the torso immersed did not produce such a response. Neither did total immersion with snorkel-breathing. Separation of breath-holding and face immersion by immersing the face and snorkel-breathing for several minutes before breath-holding, resulted in an attenuated diving response during the apnoeic period. It therefore appears that the full diving response can be obtained only when apnoea commences at the moment of face immersion.

Series I

In both series of experiments the subjects inspired only moderately immediately before the breath-holding period to avoid a Valsalva manoeuvre. This was considered important, since Sarnoff, Hardenbergh & Whittenberger (1948) demonstrated that elevation of intrapulmonary pressure by 40 mm Hg markedly reduces the systemic arterial pressure in dogs. In the present experiments a significant increase in blood pressure occurred during the final 30 sec of the breath-holding procedures. A typical Valsalva manoeuvre is also characterized by a reduction in forearm blood flow (Roddie, Shepherd & Whelan, 1958), whereas in the current experiments no significant change in flow occurred during breath-holding in air. Thus, it appears that the subjects did not raise intrathoracic pressure sufficiently to evoke a Valsalva response.

However, the variability in the response between different subjects may have been due partly to the inability to control the degree of intrathoracic pressure. Craig (1963) showed that the tachycardia characteristic of breath-holding in air was directly proportional to the increase in intrathoracic pressure. A similar tachycardia in air was observed by Harding *et al.* (1965). In the present study there was no significant change in heart rate during breath-holding in air. This difference may be related to posture since, in the studies of Harding *et al.* and Craig the subjects stood vertically, whereas in the present experiments the subjects were horizontal.

The elevation of mean blood pressure following immersion in water may be related to two factors. First, since the rise in blood pressure was signifi-

cant in Series I, but not Series II where all subjects were repeating the experiment, an emotional factor may have been involved. Secondly, once transferred, the subject breathed with the chest immersed under one or more inches of water which was equivalent to mild negative pressure breathing. Kilburn & Sieker (1960) demonstrated a small but consistent elevation of both systolic and diastolic brachial arterial pressure during similar negative pressure breathing.

The finding of a significant increase in forearm blood flow on immersion in water can be largely ascribed to the difference in temperature of the laboratory air and the pool water.

Breath-holding, torso immersed with the face out of the water resulted in changes similar to those during breath-holding in air. Craig (1963) and Harding *et al.* (1965) found that the tachycardia during breath-holding decreases as the depth of immersion of the vertical subject is increased. In the current experiments with the subjects horizontal, there could be no facilitation of venous return by adding an external hydrostatic pressure gradient.

Breath-holding, face immersed produced a marked diving response. Elements of this diving response have been demonstrated by other authors during face immersion alone and also during total body immersion (Irving *et al.* 1940; Scholander, Hammel, Le Messurier, Hemmingsen & Garey, 1962; Craig, 1963; Harding *et al.* 1965). The present investigation appears to be the first in which several major cardiovascular parameters had been measured simultaneously during total immersion in man.

As soon as the procedure commenced, a diving response was observed, which indicates that the triggering mechanism is probably neural rather than changes in blood gas tensions.

From the investigations of Kawakami, Natelson & DuBois (1967) and Brick (1966), it was expected that, if face receptors are of importance in man, a diving response should be initiated upon snorkel breathing with the face and trunk fully immersed. However, no significant change in any parameter was observed in the present study during this procedure.

Series II

During the final procedure, i.e. continuous snorkel breathing with the face submerged, there was a small but significant elevation of arterial blood pressure almost exclusively in the systolic element, probably due to the added stress of the procedure.

The measurements following the onset of voluntary apnoea showed a marked attenuation of the cardiovascular response when the immersion of the face was separated from the onset of asphyxia. This may have resulted from partial adaptation of facial receptors.

The initiation of the response still followed immediately upon apnoea, and the changes in parameters evoked by this procedure rapidly returned to pre-breath-holding levels when voluntary respiration was recommenced.

Andersen (1963), from his work with ducks, proposed that abolition of activity in the medullary respiratory centre is a determinant factor for initiation of the diving response. A similar situation apparently applies in man, although in this latter case the apnoea is voluntary instead of spontaneous.

The results of the present study support the theory that in man face immersion is an essential predisposing factor for the diving response, while cortical inhibition of respiration is important in its initiation and maintenance.

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EXPLANATION OF PLATE

General view of the experimental layout. The subject is performing a breath-hold in air above the water. Later the stretcher with the subject will be shifted to the lower supports, one of which is seen on the left, producing complete immersion of the torso.