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DIVING BRADYCARDIA IN MAN

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It has been known for many years that aquatic animals show a marked fall in heart rate on immersion in water. Paul Bert (1870) observed that in the duck 'the heart, which was beating before immersion about 100 times per minute, does not give more than fourteen regular and deep beats'. Diving bradycardia has since been described in the seal, duck, water snake, porpoise and musk rat, and Irving, Scholander & Grinnell (1941) have reported its occurrence in man.

The mechanism of the phenomenon does not appear to have been elucidated. That slowing is produced by increased vagal activity is illustrated by the finding that it is abolished by atropine (Murdaugh, Seabury & Mitchell, 1961), but the afferent side of the reflex remains obscure. Irving, Scholander & Grinnell (1942) postulate a conditioned response due to higher centre activity inducing bradycardia and peripheral vasoconstriction in order to conserve oxygen stores; Johansen (1959) finds in the snake that the apnoea and wetting of the nostrils play a part.

In the present investigation of diving bradycardia in the human subject, the cardiovascular responses to breath-holding have been examined both in air and in water at various depths of immersion. The results provide an explanation of the mechanism of the response. A preliminary account of this work has been presented to the Australian Physiological Society (Harding & Roman, 1961).

METHODS

The subjects were healthy males between the ages of 20 and 40 years. Two groups were studied. One group of five consisted of our colleagues and ourselves, all unaccustomed to underwater swimming. The other group consisted of seven experienced amateur skin divers. These could be considered as 'untrained' and 'trained' groups.

Heart rate was derived from a continuous electrocardiogram by calculating the reciprocal of the time occupied by each group of five beats. Recordings were taken with a direct writing electrocardiograph at a paper speed of 50 cm/min.

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The electrode system consisted of two precordial electrodes and an earth. The electrodes were two disks of 18-gauge copper 1¼ in. (3·2 cm) in diameter. Each was backed by a disk of $\frac{1}{16}$ in. rubber 2½ in. (6·4 cm) in diameter, made adhesive with rubber solution. The electrodes were mounted 4 in. (10 cm) apart on a thin rubber sheet 6 in. (15 cm) broad and 9 ft. (2·74 m) long, through which the leads were sealed. The earth, a small brass plate, was taped on to the skin in the epigastric fossa. After the electrodes had been applied below and behind the left nipple, the rubber strip was wound around the subject's chest and secured with adhesive tape. This system provided the electrodes with sufficient insulation, but did not hinder the breathing of the subject.

Respiratory movements were followed by means of thoraco-abdominal stethograph and a float volume recorder writing on a kymograph drum.

Underwater experiments were conducted in a cylindrical tank 6 ft. (1·83 m) high and 2 ft. (61 cm) in diameter, constructed from two 44 gallon oil drums; in all experiments the subjects stood upright. The water temperature was maintained at approximately 28° C; subjects wore only swimming trunks.

After the recording apparatus had been applied, the subject stood quietly at rest, breathing normally, on the floor of the laboratory. Electrocardiograph and stethograph recordings were begun, and after a few minutes of control observations the subject, on being told, took a moderately deep inspiration and held his breath for 45 sec, recordings being continued throughout the breath-holding period and the minute following. The subject then climbed into the tank, and stood with his feet on the bottom and with the water at the level of his sterno-clavicular joint. Control records were again taken in this position, and the subject then inspired and, by flexing his knees, submerged completely for 45 sec, records being taken throughout the immersion, and during the first minute following return to the previous position. The average heart rate during the last 30 sec of breath-holding was expressed as percentage change from the previous control level. Breath-holding in air with the knees bent gave the same response as breath-holding when standing erect.

In seven experiments the effect of different levels of immersion was observed by recording the response to 45 sec breath-holding while the subjects stood, first in the empty tank, and then at successively deeper levels of immersion as the tank was gradually filled with water.

In seven experiments the arterial blood pressure was recorded during breath-holding in air and during immersion in the tank. A 23-gauge short bevel needle was inserted into the brachial artery under local anaesthesia, and connected by 300 cm of saline-filled polythene tubing (1 mm internal diameter) to an inductance manometer and ultra-violet recorder (New Electronic Products Ltd). The system was critically damped and had a frequency response flat to 125 c/s. The subject's elbow was splinted to prevent movement. It was desirable that the manometer head should remain at the same horizontal level as the needle in the artery during the subject's movements in the tank, so as to prevent artifacts due to hydrostatic pressure changes. Since the manometer head could not be immersed in water, this was achieved by means of an inverted U-shaped steel rod, one end of which was built into a plaster cast fastened about the subject's shoulders. The metal rod projected up over the rim and down the outside of the tank, the manometer head being attached to it at the level of the needle in the artery (Fig. 1). In these experiments the heart rate was derived from the blood-pressure record, and the respiratory movements were also recorded through a second manometer channel.

In one experiment on each of the five untrained subjects the effect on the heart rate response to breath-holding of inflating a pneumatic anti-G suit applied to the legs and abdomen was studied. The effect of general body cooling was also observed in these subjects, the procedure being carried out in a cold room at 9° C, in which the subject had been at rest for at least 20 min.

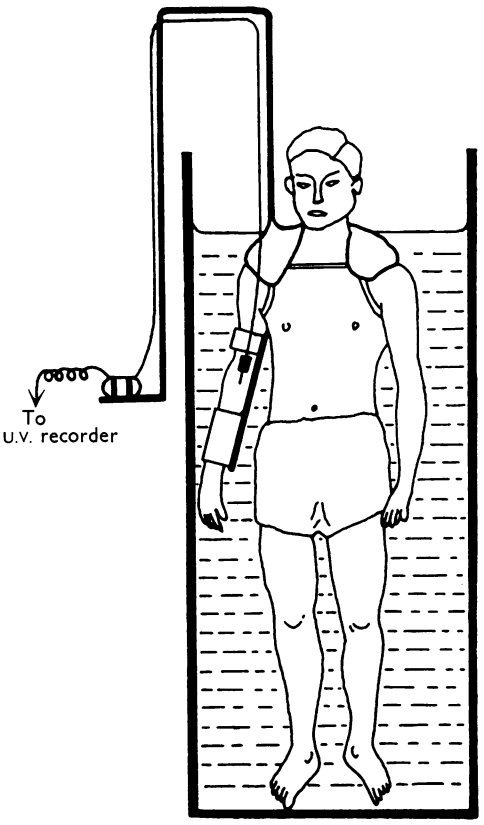

Fig. 1. Diagram of a subject standing in the tank, wearing the apparatus for underwater measurement of the blood pressure.

RESULTS

All five 'untrained' subjects showed a tachycardia on breath-holding in air for 45 sec, there being an average increase in heart rate of 19% (18 runs; average increase 15 beats/min; S.D. ± 8). Six of the seven 'trained' underwater swimmers also showed a tachycardia under these conditions, averaging 26% (8 runs; average increase 21 beats/min; S.D. ± 15). One of the 'trained' group, however, showed a fall in heart rate of 21, 18, and 15 beats/min respectively on the three occasions on which he was tested, the average fall from the resting rate being 24%.

Complete immersion in water reversed the response to breath-holding from a tachycardia to a bradycardia. In the 'untrained' group there was an average fall of 15% from the resting value (22 runs; average fall 11 beats/min; S.D. ± 13). In two of these subjects the response was not consistent, and they sometimes showed a slight increase of 6–10% in

rate. In the 'trained' group all seven subjects showed a bradycardia, averaging 17 % (14 runs; average fall 13 beats/min; S.D. ± 16).

Figure 2 shows a typical record from an experiment on one of the 'untrained' group, and in Fig. 3 the data from one run on each of the twelve subjects has been pooled; where more than one run was carried out on a given subject the data from the first run of the series have been

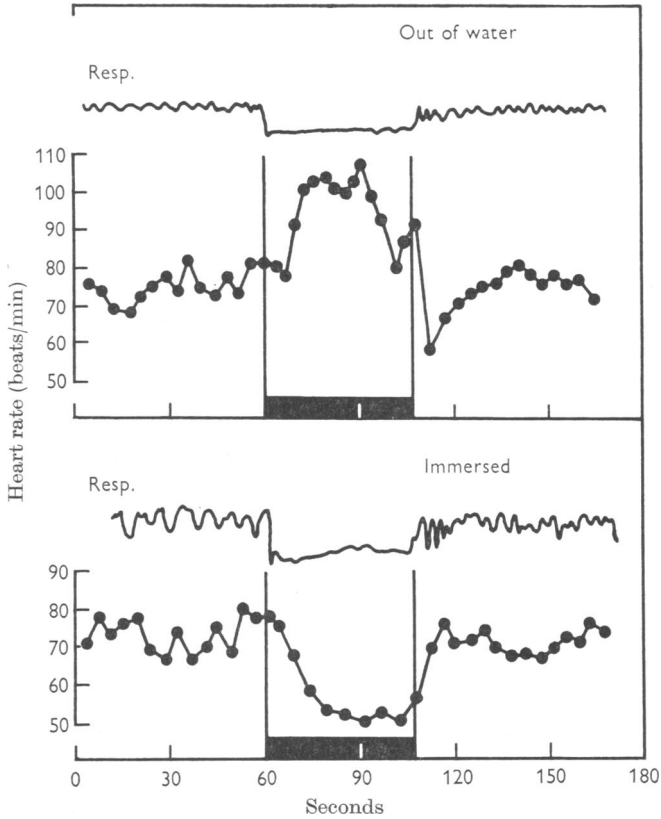

Fig. 2. Heart-rate response to breath-holding for 45 sec; upper frame standing in the laboratory, lower frame fully immersed in water, but with water at neck level during control and recovery periods. The black rectangles represent periods of breath-holding.

taken. In order to ascertain the level of immersion at which this reversal of the response to breath-holding occurs, seven experiments were carried out in which the water level in the tank was gradually raised. The subject held his breath for 45 sec with the water at five standard anatomical levels; Figure 4 shows a typical result. The tachycardia on breath-holding gradually diminished with the increasing depth of immersion, until it became very slight when the water reached the level of the xiphisternal

joint. With the water at the level of the neck, a bradycardia resulted when the breath was held. It was observed that the resting heart rate progressively fell with the increasing depth of immersion.

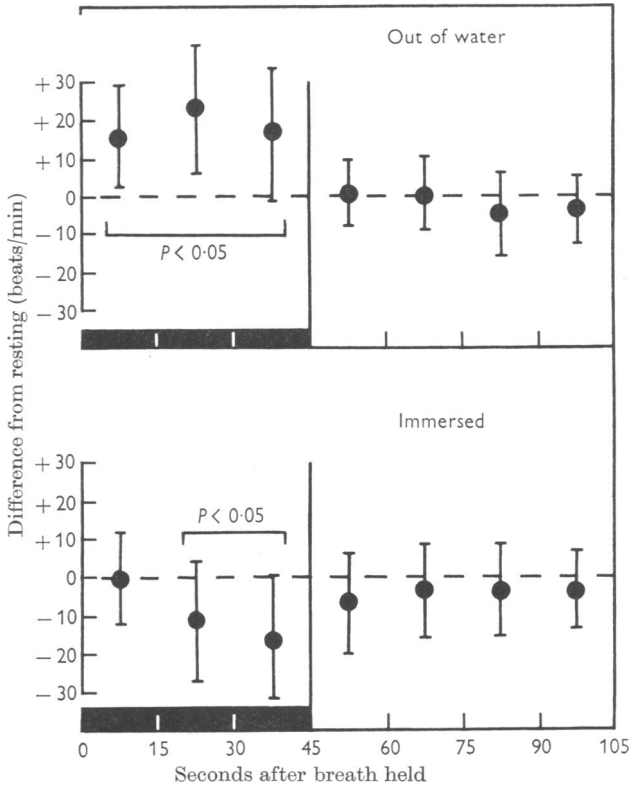

Fig. 3. Pooled data from one run in each of twelve subjects of the heart-rate response to breath-holding out of water (upper frame), and while fully immersed (lower frame). The vertical lines through the points represent the standard deviations from the means. The black rectangles represent periods of breath-holding.

The arterial blood-pressure response to breath-holding was also found to be profoundly affected by immersion. Figure 5 illustrates the changes in blood pressure which accompanied breath-holding under the two conditions. Voluntary apnoea in air produced a pattern of response identical with that seen during the Valsalva manoeuvre: there was a profound fall in both systolic and diastolic pressures, which slowly returned towards the resting level by the end of the breath-holding period. Afterwards a marked 'overshoot' in pressure occurred. The tachycardia which accompanied the fall in blood pressure was diminished as the pressure returned towards normal and a sharp drop in heart rate accompanied the 'overshoot'.

The blood-pressure response which accompanied breath-holding while fully immersed was quite different. A rise occurred in both systolic and diastolic pressure, which gradually increased over the period of the breath-holding; as the pressure increased, so the accompanying bradycardia became more pronounced.

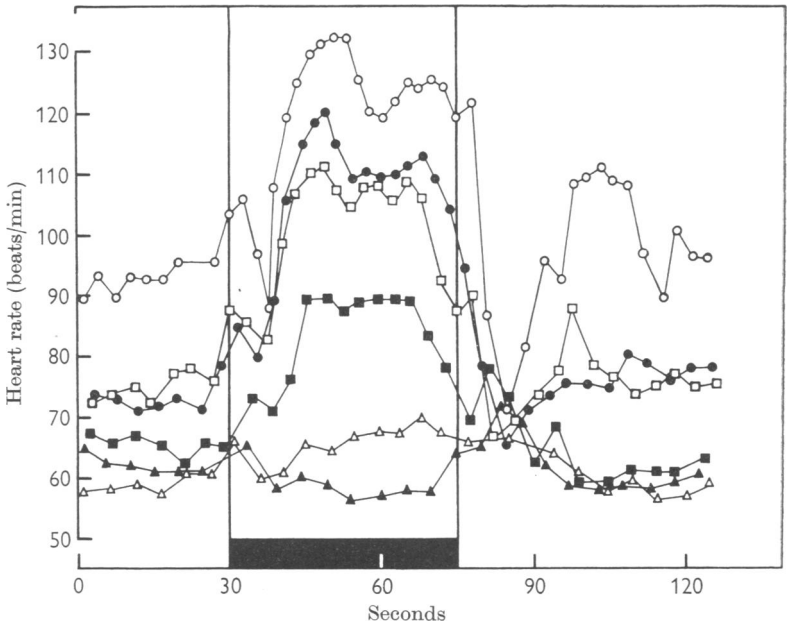

Fig. 4. Heart-rate response to breath-holding at various depths of immersion in one subject: ○ - - - - - ○, no water; ● - - - - - ●, water to knees; □ - - - - - □, water to hips; ■ - - - - - ■, water to navel; △ - - - - - △, water to xiphisternal joint; ▲ - - - - - ▲, water to neck. The black rectangle represents the period of breath-holding.

In twelve experiments the subject stood in the tank, with the water at the level of his neck, and breathed through a 1 in. diameter corrugated rubber tube and mouthpiece (a snorkel). After control recordings in this position had been taken, the subject immersed completely, but continued to breathe as normally as possible through the tube. The change occasioned by this manoeuvre was an average tachycardia of 3 % in the 'untrained' group, and of 1 % in the 'trained' group; these changes were not statistically significant.

Inflation of a pneumatic pressure suit to 100 mm Hg pressure about the legs and 40 mm Hg pressure about the abdomen caused a marked reduction in the tachycardia of breath-holding. The average increase in rate for the five subjects with the suit inflated was 6 % compared with the

increase of 19 % seen in these subjects under the same conditions without the pressure suit. This difference was highly significant ($0{\cdot}01 > P > 0{\cdot}001$). The pressures used in the suit were selected as being approximately equal to the water pressures exerted on the same parts when the subject was immersed in the tank.

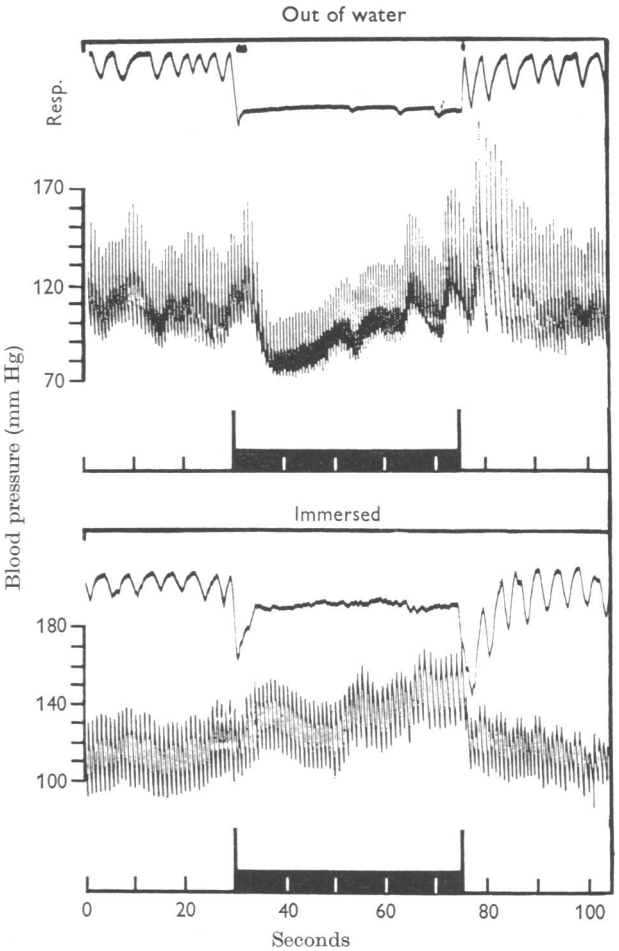

Fig. 5. Blood-pressure response to 45 sec breath-holding out of water (upper frame), and while fully immersed (lower frame), but with water at neck level during control and recovery periods. The black rectangles represent periods of breath-holding.

General body cooling at an atmospheric temperature of 9° C caused in two subjects a reversal of the normal tachycardia on breath-holding in air to a bradycardia of 1 and 3 % respectively (both the average of 3 runs). In the remaining three subjects the tachycardia responses to breath-

holding were 14, 12 and 23 %, respectively, which values were not significantly different from those obtained in these subjects when at a room temperature at 27° C.

DISCUSSION

The heart-rate response to holding the breath in air is a tachycardia, and this is converted to a bradycardia on immersion in water. This reversal of the response occurs when the water is approximately at the level of the xiphisternal joint. The experiments with the snorkel demonstrate that the appearance of a bradycardia is dependent on the breath being held, rather than on the further immersion of the head; this conclusion can also be drawn from the experiments in which the subject held his breath with the water at the level of his neck, when a bradycardia usually occurred.

The blood-pressure response which accompanies the heart-rate changes was also reversed on immersion. The pattern of the response to breath-holding in air was identical with that seen during the Valsalva manœuvre. In this procedure there is a striking fall in pressure, which is accounted for by the rise in intra-thoracic pressure impeding the venous return and causing a drop in cardiac output. The increase in heart rate is attributed to a reflex response resulting from the effect of a fall in blood pressure on the arterial baroreceptors (Lee, Matthews & Sharpey-Schafer, 1954).

When the breath was held during immersion there was a sustained rise in blood pressure, and the fall in heart rate can be explained as a reflex response to this raised arterial pressure. The cause of the reversal of the blood-pressure response to breath-holding on immersion cannot be determined with certainty from the present experiments. It is probably due to the increased volume of blood in the thorax as a result of the hydrostatic effect of the immersion on the legs and abdomen. Glaser, Berridge & Prior (1950) found that the amount of blood in the lungs was increased on body cooling and the effect of an atmosphere of 9° C in reversing the tachycardia of breath-holding in two subjects may have been due to this effect. The reduction in the degree of breath-holding tachycardia by inflation of a pressure suit may also be attributed to squeezing peripheral blood into the central pool, with a consequent reversal of the fall in blood pressure. Ross, Maddock & Ley (1961) demonstrated an increase in pulmonary blood volume on pressure suit inflation.

In those experiments in which the tank was filled with the subject in position so that he was immersed in a step-wise fashion successively from the ankles to the neck the resting level of the heart rate diminished with increasing depth of immersion and the tachycardia in response to

breath-holding became less. With the water level at about the xiphisternal joint no change in rate occurred, and above this a bradycardia resulted. In one subject in whom the arterial pressure was also recorded during this procedure the pressure rose with each succeeding depth of immersion and presumably this rise accounted for the increasing reflex bradycardia.

SUMMARY

1. The heart rates and arterial blood pressures of adult male subjects were measured in the upright position before, during and after a 45 sec voluntary apnoea, performed both in air and while fully immersed in water.

2. Holding the breath in air caused a fall in blood pressure resembling the response to the Valsalva manœuvre, and a tachycardia. The response was reversed when the breath was held when immersed; the blood pressure rose and a bradycardia occurred.

3. No difference was found between those accustomed and those unaccustomed to skin diving.

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