

NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

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THE EFFECTS OF SIMULATED BREATHHOLDING DIVES IN THE DRY
AND WET CHAMBERS ON BLOOD SHIFTS INTO THE THORAX

by

Karl E. Schaefer, Robert D. Allison, Charles R. Carey and Richard Strauss

Bureau of Medicine and Surgery, Navy Department Research Work Unit M4306.02-7060BAK9.02

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J. H. Baker, CAPT MC USN Officer in Charge Naval Submarine Medical Research Laboratory

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SUMMARY PAGE

THE PROBLEM

Blood shifts into the thorax have been shown to play a significant role in allowing the breathhold diver to go to deeper depth than could be predicted from measurements of total lung capacity and residual volume. Studies were carried out to determine whether the blood shifts are caused by hydrostatic pressure only and whether there is an increasing blood shift with increasing depth.

FINDINGS

Measurements of heart rate, blood pressure and transthoracic resistance (impedance plethysmograph) were made during simulated breathhold dives to 25, 50 and 90 feet in the wet and dry chamber. During the former, blood shifts into the thorax occurred and the heart rate fell below initial levels. During the dives in the dry chamber no systematic changes in the transthoracic resistance were seen. The heart rate did not decrease significantly below initial levels. Data provide evidence that the blood shifts into the thorax during breathhold diving are caused by the hydrostatic pressure.

APPLICATIONS

These findings are of interest to Navy Medical Officers involved in diving instruction and scientists concerned with diving medicine.

ADMINISTRATIVE INFORMATION

This investigation was conducted as a part of Bureau of Medicine and Surgery Research Work Unit M4306.02-7060BAK9-Regulation of Respiration, Circulation and Body Temperature at Rest and During Exercise in Naval Diving Operations. The present report is the second on this work unit. The manuscript was submitted for review on 7 July 1972, approved on 26 October 1972, and designated as Naval Submarine Medical Research Laboratory Report Number 729.

Robert D. Allison, Ph.D., is a member of the staff at Scott & White Clinic, Temple, Texas; LT Richard Strauss, MC, USN, was a Submarine Medical Officer Candidate at the time of this investigation.

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ABSTRACT

Heart rate, blood pressure and transthoracic resistance changes were measured in 5 trained divers during simulated breathhold dives to 25, 50 and 90 feet in a wet and a dry chamber. During the "wet" dives heart rate fell significantly below control values and the transthoracic resistance changes provided conclusive evidence for blood shifts into the thorax. Reverse blood shifts out of the thorax developed at the end of breathholding on return to the normal atmospheric pressure. The "dry" dives on the other hand did not show any systematic changes in transthoracic resistance and the heart rates did not decrease significantly below control levels.

The results of these studies indicate that the hydrostatic effect of immersion on legs and abdomen forces the blood into the thorax. Blood shifts amounted to about 500 ml during breathholding when immersed on the surface and were in excess of one liter during breathhold dives to 25, 50 and 90 feet.

The existence of these blood shifts during breathhold dives makes it highly unlikely that there is an abrupt depth threshold at which "thoracic squeeze" would occur under actual diving conditions.

THE EFFECTS OF SIMULATED BREATHHOLDING DIVES IN THE DRY AND WET CHAMBERS ON BLOOD SHIFTS INTO THE THORAX

INTRODUCTION

Blood shifts into the thorax have been shown to play a significant role in allowing the diver to go to greater depth than could be predicted from measurements of total lung capacity and residual volume or the ratio of compressible to non-compressible air-containing spaces.⁵

In previous studies it was observed that blood shifts into the thorax at 90 and 130 feet of depth amounted to 1047 and 850 ml, respectively, based on measurements with the impedance plethysmograph.6 Craig3 reported evidence suggesting a blood shift of 600 ml in dives to 4.75 m following maximal exhalation. He found no significant difference between esophageal and ambient pressure at this depth, indicating that the subject's residual volume of 2.0 liters must have been compressed to 1.4 liters, owing to corresponding blood shift. Further studies were done on 5 divers in a wet and dry chamber during simulated breathholding dives to 25, 50, and 90 feet to determine whether (1) the blood shifts are caused by hydrostatic pressure alone and (2) whether there is an increasing blood shift with increasing depth. The results are presented in this report.

METHODS

Five veteran navy divers in swim trunks were studied separately in a compression chamber in the dry state under increased atmospheric pressures equivalent to 25, 50 and 90 foot water depths. Subsequently they were submerged singly in a tank of water inside the compression chamber for study at the same pressure equivalents. Heart rate changes were recorded from electrodes placed on the precordium following suitable skin preparations. Systemic blood pressure was measured with a sphygmomanometer attached to the right brachial region. Corrections for the blood pressure measurements carried out in the wet tank were made for the positioning of the cuff above heart level. This amounted to a subtraction of 15-20 mm Hg from the observed value.

A four-electrode, 120-Hertz impedance plethysmograph was used to measure changes in the subject's thoracic resistance as related to displaced blood volume during breathhold dives. In the four-electrode system, current (0.12 ma at 120 my) was introduced to an outer set of electrodes (I1, I2) and variation in conduction of the current as a function of thoracic gas or blood volume was detected between inner electrodes $(E_1, E_2)^2$. Two pairs of lead strips (1 cm wide by 20 cm long) were fastened horizontally to a sponge pad (30 by 20 by 1 cm). The two strips of a pair were separated by one cm, and one pair was spaced 30 cm from the other. The pad was placed on the back of the subject with the inner strip (E1) of the upper pair at the level of C7 (7th cervical vertebra) and the inner strip (E2) of the lower pair of T12 (12th thoracic vertebra). Velcro straps, fastened to the upper and lower edges of the sponge pad, crossed over the front of the chest and held the electrode pad in place under the diver's wet-suit jacket. The lead strips were soldered to insulated wires which terminated in a four-pin attachment for connection with a shielded cable leading to the impedance plethysmograph placed outside the chamber.

After application of electrodes to the subject, the plethysmograph unit was balanced by means of a variable potentiometer. This null balance represented the resistance of the subject's thorax between detecting electrodes (E₁-E₂). Evidence for the validity of the impedance method to detect blood volume changes in the lungs has been described.² The question could be raised whether movements of the dia-

phragm would affect the electrical resistance measured which could be interpreted as blood shifts. However in our experiments the chest was held in a fixed position during breathholding. During the periods of breathholding, no involuntary diaphragmatic contractions were observed. It is therefore concluded that under the conditions of our experiments diaphragmatic movements did not affect the measurements of thoracic resistance.

RESULTS

Table I exhibits the <u>vital statistics</u> of the subjects. Croft, the world record holder in deep breathhold diving, was one of the subjects (R.C.). His vital capacity was 7.8 liters. Another diver subject (W.E.) also had an unusually large vital capacity. Table II

Table I. Vital Statistics

Subject	age (years	height (inches)	weight (lbs)	Vital Capacity L _{BTPS}
С	34	69	185	7.8
F	28	72	175	4.31
N	36	70	200	5.65
Wa	32	70	185	4.71
We	28	72.5	210	7.41
MEANS	31,6	70.7	191	5.98

Table II. Diving Profile and Times of Breathholding during Simulation Dives to 25, 50, and 90-Foot Depths in Dry and Wet Chambers

Condition		Time of Descent (Seconds)	Time at Depth (Seconds)	Time of Ascent (Seconds)	At Surface (Seconds)	Total Dive Time (Seconds)
25 Feet						
A. Dry	Mean	22.0	18.0	17.4	56.0	67.0
Chamber	S.E.M.	1.83	1.9	1.7	1.4	1.9
B. Wet	Mean	24.0	23.0	16.2	60.0	74.0
Chamber	S.E.M.	4.2	3.7	1.4	4.7	4.4
50 Feet						
A. Dry	Mean	31.0	22.0	24.2	80.0	92.0
Chamber	S.E.M.	1.8	3.8	7.3	6.7	7.3
B. Wet	Mean	25.0	21.0	26.0	78.0	87.0
Chamber	S.E.M.	2.7	3.5	4.0	3.3	3.0
90 Feet						
A. Dry	Mean	43.0	26.0	34.2	102.0	118.0
Chamber	S.E.M.	2.7	5.4	5.7	6.1	10.0
B. Wet	Mean	46.0	20.0	33.5	100.0	111.0
Chamber	S.E.M.	4.0	1.3	1.3	2.8	4.9

presents the diving profiles in the wet and dry chamber dives. They were in good agreement with each other. The times of descent and ascent increased with depth. Time at depth was approximately 20-25 seconds. Total dive time depended on depth and ranged from 1-2 minutes. Changes in heart rate and thoracic resistance observed in 4 subjects during 2 minutes of breathholding on the surface are presented in Figure 1 for: (1) breathholding in air, and (2) breathholding with the body immersed in water with the subject in a sitting position.

It has been observed that there is a pronounced decrease in heart rate (bradycardia) and in thoracic resistance during submersion in water. During

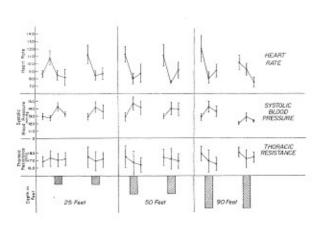


Fig. 3. Effect of simulated breathhold dives in the wet chamber at 25, 50 and 90 feet on heart rate, blood pressure and thoracic resistance.

resulted in a significant fall of pulse rate and decrease in thoracic resistance. At the end of breathholding on return to the surface, pulse rate increased again at 25 feet. However, pulse rates remain at a lower level at 50 and 90 feet breathhold dives. The thoracic resistance returned regularly to higher levels upon return to the surface and normal breathing.

Figure 4 shows a comparison of the effects of breathhold dives in the wet and dry chamber. Values are expressed in percent changes.

The dives in the wet chamber exhibited (a) a reproducible pattern of a

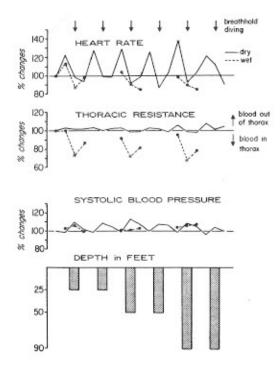


Fig. 4. Comparison of the effects of simulated dives in the wet and dry chamber on percentage changes of heart rate, blood pressure and thoracic resistance.

decrease in thoracic resistance during the dive and (b) a subsequent increase in thoracic resistance on returning to the surface immediately after the end of the breathholding.

Blood pressure did not change significantly. Heart rate decreased significantly during some of the dives in the wet chamber but not during some of the dives in the dry chamber.

All the heart rate data on hyperventilation were obtained under normal atmospheric pressure, the only difference being that in the wet chamber the subjects were immersed up to the neck in water. The increase in heart rates produced by hyperventilation of about 2 minutes was found to be significantly higher in the dry chamber, averaging 27% as compared to an average of 11% in the wet chamber.

Actual calculations of blood shifts were performed for one subject, in whom all the data of gas exchange at various depths was available (Croft, Table III).

Calculations of blood volume were obtained in the following manner:

$$V_{o} = p (L^{2}/R_{o})$$

$$V = (150/R_{o}) \times [(L^{2} \times \Delta R)/R_{o}]$$

p = resistivity of blood at 37°C, 150 (ohm/cm)

 L^2 = distance (cm) between detecting electrodes (E₁-E₂)

R_o = resistance (ohms) of the thorax when the plethysmograph is balanced.

During breatholding at the surface with the subjects immersed in water, the blood shifts amounted to 500 ml. During breathold dives to 25, 50 and 90 feet, the blood shifts were above one liter. The reversed changes in thoracic resistance (increases at the end of breathholding - indicating shift of blood out of the thorax) corresponded with the blood shifts into the thorax during breathhold diving.

DISCUSSION

These data provide evidence based on transthoracic electric resistance changes that blood shifts into the thorax occur during breathholding when immersed in water and reverse blood shifts out of the thorax develop at the end of breathholding on return to normal atmospheric pressure.

The dry dives on the other hand did not show any systematic changes in thoracic resistance. Although there was a tendency toward a decrease during breathholding from the level attained during hyperventilation prior to the dive, there was no regular response toward an increase at the end of breathhold dives.

When the subjects were breathholding in the dry chamber, their heart rate
did not fall significantly below control
levels, although the decrease was significant when compared with the increased heart rate values attained
during hyperventilation prior to the
dives.

In the "wet dives" heart rate fell significantly below control values measured at the surface and at each depth, with the exception of the dives to 90 feet, at which time the subjects showed indications of exhaustion.

The slight rise in blood pressure observed in all breathhold dives in the wet and dry chambers never reached statistical significance. Direct

Table III. Blood Shifts In and Out of the Thorax During Simulated Breathhold Diving to 25, 50 and 90 Feet Depth and Immersion at the Surface (Subject: Croft)

	Conditions	Thoracic Resistance (ohms)	Thoracic Conductive Volume (liters)	Correction For Gases (CO ₂ , O ₂) Conduct. Vol. Changes	Blood Volume Shift · ML
1.	Surface (Control)	10.6	12.738		
2.	Breathholding 2' Surface Immersed in Water.				
	Position Sitting	10.2	13.235		+ 500
3.	Surface (Control)	14.0	9.600	-	
4.	25 Feet Depth	12.2	11.065 + 1.465	134	<u>+1330</u>
5.	Surface Follow- ing Dive to 25 Feet	13.6	9,926 - 1,339	-	-1339
6.	50 Feet Depth	11.9	11.340 + 1.740	245	+1485
7.	Surface Follow- ing Dive to 50 Feet	14.0	9.600 - 1.740	-	-1740
8.	90 Feet Depth	11.3	11.946 2.346	388	+1958
9.	Surface Follow- ing Dive to 90 Feet	13.4	10,075	-	-1553

measurements of arterial blood pressure might have indicated differently, but this is purely conjectural.

The results of these studies demonstrate that the hydrostatic effect of immersion on legs and abdomen forces the blood into the thorax.

The immediate blood shift into the thorax upon immersion in water at the surface demonstrated that for such a shift of approximately 500 ml the external hydrostatic pressure is responsible.

During the surface breathholding, CO₂ accumulates in the lungs and only oxygen leaves the lung. But during descent to 25, 50, 90 feet, both CO₂ and oxygen leave the lung a situation which requires additional movement of blood into the lungs for pressure compensation.

Whether this is the cause of the larger blood shifts amounting to about 1.5 liters found in Croft¹⁰ at 25, 50 and 90 feet breathhold dives or whether other factors play an additional role cannot be stated at this time.⁸

At any rate, Croft's lung volume was not compressed to residual volume in any of these dives. Therefore, it would seem that such a compression of the lung is not a requirement for the occurrence of the blood shift. Blood shifts appear to be quite similar in magnitude at the three depth levels measured. This indicates that the blood volume entering the thorax is not a function of depth.

Agostoni lobserved a decrease in residual volume when subjects were submerged to the neck; and he assumed that this gas volume was replaced by an equivalent amount of blood shifted into the thorax. Rahn⁵ had also postulated a blood shift into the thorax in breathhold diving.

Harding et al⁴ observed a 17% fall in heart rate during breathholding for 25 seconds in trained underwater swimmers, a fact which corresponds with the 20.5% fall in heart rate measured in the subjects of this study, who were trained divers.

These authors also found that breath-holding on the surface without submersion resulted in tachycardia and decreased blood pressure in most of the trained divers with the exception of one who exhibited consistently a decrease in heart rate under these conditions. Harding et al postulated a blood shift into the thorax due to hydrostatic effects of immersion and explained the fall in heart rate as a reflex response of the arterial baroreceptors to the increased blood pressure.

The consistent but small blood pressure increases found in our experiments in practically all dives could have contributed to the decreases in heart rate observed. Other factors like face immersion must also be considered. The blood shifts into the thorax observed in the "wet dives" may have been responsible for the significant fall of the heart rate below control values in these dives.

Some practical conclusions can be drawn from these observations on the blood shifts in breathhold dives in the wet environments. The existence of these blood shifts makes it highly unlikely that there is a typical and well demarcated depth threshold at which a thoracic squeeze occurs. A number of cases of "thoracic squeeze" were observed at the Escape Training Tank of the Naval Submarine Base, in Groton, Connecticut, over the years and these cases involved pain and hemoptysis, But there were never any abnormal signs on the chest x-rays taken and the patients were usually released the following day. The only reported case of fatal thoracic sequeeze involving breathhold diving was one reported by Strauss and Wright.7

It appears that there is no basis for emphasizing the thoracic squeeze, as far as breathhold diving is concerned, as has been done in some diving courses. The "casual" reference given to "thoracic squeeze" in the Navy diving reference books 8.9 is supported on the basis of experience and the observations on blood shifts.

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The results of these studies clearly indicate that the hydrostatic effect of immersion on legs and abdomen forces the blood into the thorax. Blood shifts amounted to about 500 ml during breathholding when immersed on the surface and were in excess of one liter during breathhold dives to 25, 50 and 90 feet.

The existence of these blood shifts during breathhold dives makes it highly unlikely that there is an abrupt depth threshold at which "thoracic squeeze" would occur under these conditions.

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