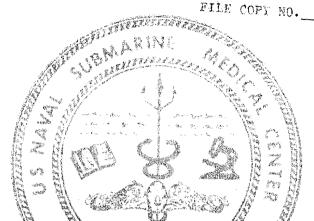
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Submarine Base, Groton, Conn.

REPORT NUMBER 531

PULMONARY AND CIRCULATORY ADJUSTMENT DETERMINING THE LIMITS OF DEPTHS IN BREATHHOLD DIVING

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Bureau of Medicine and Surgery, Navy Department Research Work Unit MR005.04-0054.02

Approved and Released by:

Gerald J. Duffner, CAPT MC USN COMMANDING OFFICER Naval Submarine Medical Center

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SUMMARY

THE PROBLEM

To determine pulmonary and circulatory adjustments determining the limits of depths in breathhold diving.

FINDINGS

Measurements of thoracic resistance using the impedance plethysmograph provided evidence for a blood shift from the periphery into the thorax during breathhold dives. These findings explain the extension of the depth threshold in breathhold diving beyond the calculated depth threshold based on the compression ratio of total lung volume/residual volume. Moreover, gas exchange data indicate that neither increased CO₂ nor hypoxia determined the depth limits in maximal breathhold dives.

APPLICATION

This information is of importance for Diving Medical Officers and investigators of diving physiology.

ADMINISTRATIVE INFORMATION

This investigation was conducted as a part of Bureau of Medicine and Surgery Research Work Unit MR005.04-0054.—Physiological Alterations in Free Diving. The present report is No. 1 on this Work Unit. The manuscript was approved for publication on 5 June 1968 and designated as Submarine Medical Research Laboratory Report No. 531.

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ABSTRACT

Data on pulmonary gas exchange were collected in breathhold dives to 90 feet at the Escape Training Tank, Naval Submarine Base, Groton, Connecticut, and in open sea breathhold dives, off Ft. Lauderdale, Florida, to a depth of 217.5 feet on diver R. Croft. Thoracic blood volume displacements were measured at depths of 25, 50, 90, and 130 feet, using the impedance plethysmograph. The open sea dives were carried out with an average speed of descent of 3.95 feet per second using a 65-pound cable brake and an average rate of ascent of 3.50 feet per second pulling up the line.

End-of-dive alveolar oxygen tensions did not fall below 40 mm Hg, while alveolar CO₂ tension did not rise above 40 mm Hg, except in one case. These findings indicate that neither hypoxia nor hypercapnia determined the depth limits under those conditions. At depths of 90 and 130 feet, blood was forced into the thorax, amounting to 1,047 and 850 ml, respectively. This is the first direct evidence of intrathoracic blood pooling in breathhold diving at greater depths. It explains the extension of Croft's 197-foot depth threshold calculated from his total lung volume/residual volume ratio to the depth of 217.5 feet actually reached in his world record dive.

PULMONARY AND CIRCULATORY ADJUSTMENT DETERMINING THE LIMITS OF DEPTHS IN BREATHHOLD DIVING

INTRODUCTION

Recent exploits in breathhold diving have shown that man has the capacity to dive to depths in excess of 200 feet (1). This raises questions about the validity of the generally held assumption that the depth threshold is determined by a point at which total lung volume* (the amount of gas contained in the lungs at the end of maximal inspiration) is compressed to the residual volume (volume of gas in lungs at end of maximal expiration). Both recent record holders in breathhold diving, Robert A. Croft—217.5 feet and Jacques Mayol—231 feet, went to considerably greater depths than could be predicted on the basis of their total lung volume/residual volume ratio. Additional factors must, therefore, be considered. It has been suggested that at greater depths blood is forced into the thorax replacing air and resulting in a decrease of residual volume and thereby extending the depth limit (2,3).

This report presents the results of the first measurements of thoracic blood volume made in breathholding dives, demonstrating a shift in blood volume into the thorax at great depths. Moreover, end-of-dive alveolar gas tensions were obtained in dives up to 217.5 feet in the open sea, considerably extending the depth range of information in pulmonary gas exchange in breathhold diving.

METHODS

Studies were carried out on Robert Croft, a U.S. Navy diver, to depths of 90 feet at the Escape Training Tank, Naval Submarine Base, Groton, Connecticut, and extended to open sea dives off Ft. Lauderdale, Florida, culminating in Croft's world record dive to 217.5 feet.

TABLE I-VITAL STATISTICS OF DIVER R. CROFT

		Predicted normal (and range)
Age	33 years	
Height	69 inches	
Weight	175 pounds	
Vital Capacity	7.8 L (BTPS)	4.9 (3.8 — 6.1) L (4)
Res. Volume	1.3 L (BTPS)	2.0 (1.1—2.9) L. (5)
Total Lung Volume (Capacity)	9.1 L (BTPS)	6.9 (5.4 — 8.4) L. (4.5)
Total Lung Volume (Capacity) /Residual Volume Ratio.	6.96	3.17 (2.43 — 4.59) (5)
Compression Ratio	6.96=197 feet depth	72 (46 — 118) Feet
Max. Expiratory Pressure	290 mm Hg at 6.4 L inhalation.	(60 — 100 mm Hg) (5)
Max. Inspiratory Pressure	117 mm Hg at 3.2 L inhalation	(60 — 100 mm Hg) (5)

^{*}Total lung volume corresponds to the term total lung capacity as used in standard pulmonary physiology texts. The term volume is used in this report to maintain consistancy in the description of gas and blood volume changes.

The vital statistics of R. A. Croft (Table I) show his unusually large vital capacity and small residual volume. Based on the ratio of total lung volume (total lung capacity, TLC) to residual volume his depth threshold would be 197 feet, which is considerably deeper than that of the average person (80-100 feet). Moreover, R. Croft is able to exert much larger expiratory pressures and somewhat larger inspiratory pressures than those found in normal healthy subjects (the expiratory and inspiratory pressures were measured at different lung volumes, varying from residual volume to total lung capacity).

Before the dive in the open sea, the subject sat on a platform up to his waist in water. He lowered himself into the water up to the neck and held onto the raft in front of him, which supported two investigators who collected alveolar and mixed expired gas samples. The breathing valve was attached to the raft in a low position. The subject breathed through a 11/4 inch diameter tube into rubber bags. A two-way stop-clock allowed switching from one bag to another at the end of collection periods. The filled bags were clamped and stored until the end of the experiment. The volume of the bag was determined with a dry gas meter. Gas analysis was carried out with an infrared CO2 meter (Godard) and a Beckman Model 777 Oxygen meter.

At the end of the dive, the subject made his first exhalation into a two-bag system previously used in diving studies (6) or a one-bag system with a Hans-Rudolph low dead space (18 ml) valve. The latter is demonstrated in Figure 1 which shows the collection of alveolar gas samples following the world record dive to 217.5 feet. Following the collection of the end dive sample, the subject continued to breathe into the plastic bags until the end of the experiment. Expired air was collected at one-half or one minute intervals during the first five minutes and then in two-three minute intervals.

A four-electrode, 120 KHz impedance plethysmograph was used to measure changes in the subject's thoracic resistance related to displaced blood volume during breathhold dives. In the four electrode system, current

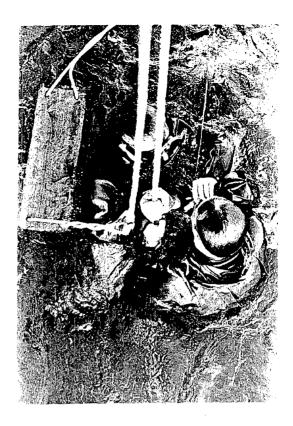


Figure 1. Collection of alveolar gas samples from Robert Croft following his world record dive to 217.5 feet.

(.12MA-120 MV) is 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 is detected between inner electrodes (E₁, E₂) (7). Two pairs of lead strips (1 cm wide-20 cm long) were fastened horizontally to a sponge pad (30x 20x1 cm). Each of the strips in each pair was 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 (E_1) of the upper pair at the level of C₇, and the inner strip (E₂) of the lower pair at T₁₂. 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 connector brought from beneath the wet suit jacket for connection with a shielded cable of approximately 300 feet in length

which was connected to the impedance plethysmograph.

Following 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 $(E_1 - E_2)$.

Under the experimental conditions of this study, movement artifacts were encountered as a major difficulty.

Calculations of blood volume were obtained in the following manner:

$$\begin{split} V_o &= \frac{PL^2}{R_o} \\ V &= \frac{150}{R_o} \quad \times \quad \frac{L^2 \times \triangle R}{R_o} \end{split}$$

P = resistance of blood at 37° 150 (ohm cm)

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

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

Output of the plethysmograph was recorded on a Sanborn polygraph series 150. Expiration and increased blood volume were associated with decreased thoracic resistance. No attempt was made to interpret superimposed pulsatile changes related to the cardiac cycle in this study.

Evidence for the validity of the impedance method to detect blood volume changes in the lungs has been described (7,8,9). Measurements of blood, pulse rate and blood flow using electromagnetic flow meters and direct bleed-out methods simultaneously with impedance plethysmograph studies in dogs agreed within four percent ± five percent (7).

After a typical period of hyperventilation prior to a dive in the open sea, alveolar CO_2 tension has decreased to 22.1 mm Hg while alveolar O_2 tension has risen to 132 mm Hg, Figure 2. At the end of breathhold dives to various depths both alveolar CO_2 tension and alveolar O_2 tension remain at approximately the same level averaging 35 mm Hg P_{CO_2} and 40 mm Hg P_{O_2} respectively, Figure 2. The

constancy of the end dive alveolar gas tensions is astonishing in view of the different demands on energy expenditures made during dives to different depths.

END DIVE ALVEOLAR GAS TENSIONS

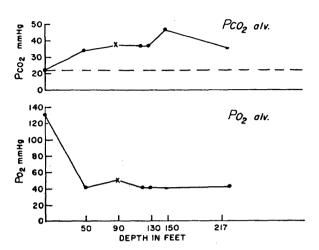


Figure 2. End dive alveolar gas tension obtained after rapid ascent at an average rate of 1.2 m/sec. from various depths. Control values-alveolar gas tensions after rapid exhalation following maximal inhalation.

Since Croft exhaled about the same amount of air at the end of the dives, both O_2 and CO_2 content of the lungs remained approximately the same, averaging 230 ml of O_2 and 190 ml of CO_2 . This oxygen content is considerably higher than the 100 ml left at the breaking point of breathholding in dives previously found in Croft (10).

Oxygen used and carbon accumulated during individual breathhold dives were calculated from the differences between the gas content of the lungs before and after the dives and plotted in Figure 3. The gas exchange values exhibit the same constancy for different dives as the alveolar gas tensions at the end of the dives. About 1000 ml of O_2 are used while practically no CO_2 is accumulated, resulting in a respiratory exchange ratio near zero.

The excess oxygen, calculated by subtracting control values from those determined during the dives and during the recovery period, provides an estimate of the oxygen requirement for the dive.

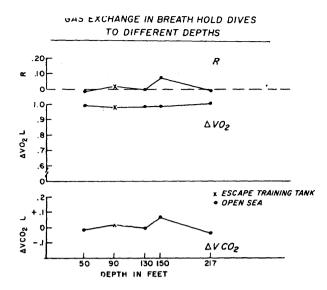


Figure 3. Gas exchange in breathhold dives to different depths. Oxygen consumed and CO₂ accumulated during the individual dives has been determined by subtracting the end dive oxygen and CO₂ content of the lungs from control values measured prior to the dive.

Data obtained on R. Croft during breathhold dives with maximal ascent rates (1.07— 1.4 meter/sec) to 50, 90, 130 and 145 feet are shown in Figure 4. The literature contains only values on breathhold dives to shallower depths and at a slower ascent rate (.6 -.67) meter/sec) by Craig (11) and Yokoyama (12). These data are included for comparison in Figure 4, together with other values collected by us in previous studies on R. Croft, and another group of 8 divers in breathhold dives to 90 feet using slow and fast ascent rates. It is readily apparent, that the oxygen requirements do not increase linearly with depth, but instead level off at greater depths for both slow and maximal ascent rates, which is in line with observations of Craig at shallower depths (11). It appears that maximum values of oxygen cost in single deep dives lie around three liters of oxygen. The average oxygen cost of Croft's dives to 90, 130 and 145 feet was 2.7 liters. At an average diving time of 86 seconds, the oxygen consumption per minute in these single dives amounted to 1.9 li/min, which is somewhat lower than the reported maximal oxygen consumption (V_{02}) of 3.75 liters/min during exercise (13).

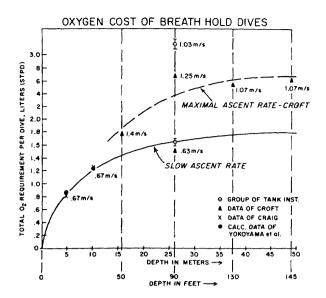


Figure 4. Oxygen cost of breathhold dives. Calculation of the total $V_{0:2}$ needed for dives to different depths were made by subtracting the oxygen consumption measured during the control periods prior to the dive from the oxygen consumed during the dive and during the recovery period after the dive.

The electrical resistance measurements of the human thorax as determined by impedance plethysmography represents a balance between gas, blood and other tissue volumes within the area defined by the detecting electrode positions (E_1-E_2) . Decrease in resistance is associated with decreased gas volume and increased blood volume, and vice versa. Inasmuch as the segmental thoracic resistance relates to the ratio of blood and gas in the thorax, it is correct to speak of a thoracic conductive volume. To arrive at blood volume one has to subtract the changes in gas volumes from the calculated thoracic conductive volume. To accomplish this it is necessary to determine the thoracic resistance at different ratios of gas volume to blood volume in a subject.

Such a calibration was performed with diver Croft in fresh water, sea water and air, thoracic resistance was determined after complete exhalation and inhalation of 1.5, 3.0, 4.5, 6.0, and 7.0 liters of air from a spirometer at ambient temperatures; these were later converted to BTPS. Resistance changed in all three conditions in a linear manner, (see Figure 5), although the basic resistance at end expiratory level was dif-

ferent due to the different temperature conditions. Using these calibration curves, it was possible to calculate the resistance changes produced by, a) compression of gas, and b) transfer of gas. The latter had been determined for depths up to 90 feet. For 130 feet of sea water the compression ratio was known, but the gas transfer had to be approximated.

THORACIC RESISTANCE CHANGES WITH INCREASING GAS VOLUMES FRESH WATER 33°C SEA WATER 5°C SEA WATER 5°C AMBIENT AIR Te. 10 SEND Expiratory Position (Residual Volume) O 1 2 3 4 4 5 6 7 8 LUNG VOLUME

Figure 5. Thoracic resistance rises nearly linearly with increasing gas volumed measured in sea water, fresh water and ambient air at different temperatures. The basic thoracic resistance measured at residual volume varies with the temperature of the medium.

Table II lists the thoracic resistance changes recorded at various depths in fresh water and sea water. Based on the thoracic resistance changes the thoracic conductive volumes were calculated using the formula listed on the bottom of the table. The thoracic conductive volume changes due to compression of gas and gas transfer from the lungs into the blood shown in column 5 were subtracted from the total thoracic conductive volumes changes leaving the thoracic conductive volumes changes due to blood shifts, exhibited in column 6.

TABLE II - EFFECT OF COMPRESSION AT VARIOUS DEPTHS ON THORACIC CONDUCTIVE VOLUME

Condition Fresh Water	Thoracic * Nesis- tance OHMS	Thoracic ** Conductive Volume	A Surface-Depth Thoracic Conductive Volume liters	Calculated Con-*** ductive Volume Changes Due to Gas Volume Changes	Conductive Thoracic Vol- ume Changes Due to Blood Volume Shifts
Surface	39.3	3.950			
25 feet	34.5	4.452	.502	.250	+ .252
50 feet	33,1	4.640	.690	.350	+ .340
90 feet	28.2	5.447	1.497	.450	+ 1.047
Seawater					
Surface	18.0	8.500			
130 feet	13.0	11.850	2.350	1,500	.850

* Resistance (OHMS) between

Electrodes E1-E2 on thorax

**
$$V_0 = \frac{150 \text{ x}}{R0}$$
 (L2 = distance between detecting electrodes)

** $\Delta V = \frac{\Delta R \times V_0}{R0}$

At 90 feet fresh water in the Escape Training Tank, 1047 ml of blood were shifted into the thorax, and 850 ml blood at 130-foot depth of sea water. These blood volume shifts are in the order of magnitude one would expect from other considerations. Jacques Mayol, who set the most recent world record in breathhold diving, reaching 231 feet, was kind enough to let us have the results of his pulmonary function tests carried out at Broward General Hospital in Fort Lauderdale, Florida, under the direction of Dr. W. S. Williams. His vital capacity was 5.281 L. and his residual volume 1.883 L. Adding 60 ml for the sinus dead space, we calculated a total lung capacity (total lung volume) of 7.224 L. The ratio of TLC to residual volume - 3.717 which corresponds to a depth threshold of 90 feet. In order to dive to 231 feet a blood shift of 980 ml into the thorax is required with a corresponding replacement of air and reduction of his residual volume to 0.903 ml. Studies of respiratory mechanics done during submersion and in air, lend further support to the reported blood shifts into the thorax during diving. The intrathoracic pressures remained during submersion at the same level as

during control conditions sitting in air, while the pressures of the extrathoracic vascular system showed an absolute increase (2).

Indirect evidence of intrathoracic blood pooling in breathhold diving may be deduced from the observations of a "diving diuresis" commensurate with dehydration of red cells in Tank instructors carrying out breathhold dives to 90 feet (14). This suggests the operation of the Gauer-Henry effect according to which any measure leading to an expansion of intrathoracic blood volume is accompanied by a diuresis (15). This volume regulation is controlled by stretch receptors in the left atrium, whose activity decreases with increasing thoracic blood volume resulting in a reduction of anti-diuretic hormone secretion and associated diuresis (15).

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