Hypoxia and carbon dioxide retention following breath-hold diving¹

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PAULEV, POUL-ERIK, AND NOE NAERAA. Hypoxia and carbon dioxide retention following breath-hold diving. J. Appl. Physiol. 22(3): 436-440. 1967.—Experimental data from six subjects performing repeated breath-hold dives to 62 ft (18.5 m) in fresh water are presented. Rate of descent was 0.8 m/sec, ascent 1.5 m/sec, and diving time 36-124 sec. Surface intervals varied between 60 and 120 sec. The divers submerged with a lung volume containing residual volume plus 85% of vital capacity. About 650 ml (STPD) oxygen was transferred from the lungs to the body in 42-sec dives (mean of seven dives), while 260 ml carbon dioxide was given off from the body to the lungs. During the first 16 min after a seven-dive series (representing a total submerged time of 330 sec and a total surface time of 420 sec) an excess elimination of approximately 3,500 ml (STPD) carbon dioxide was found. Carbon dioxide retention with acute, respiratory acidosis may—because of the repetitive factor—be more dangerous to breath-hold divers than hitherto believed.

hypoxia; acidosis; repeated skin diving

A REVIEW of the literature shows that the pulmonary physiology of single breath-hold dives has been examined thoroughly, both theoretically (3, 4) and experimentally, covering depths from about 5–30 m (7, 8, 11, 13). However, the physiology of repeated breath-hold diving has not been investigated until recently (10). In connection with a deep breath-hold dive, carbon dioxide retention and delayed CO₂ excretion have been demonstrated (12). This phenomenon might be of importance in repeated dives with short surface intervals, leading to an increasing retention of carbon dioxide with an increasing number of dives. The danger of hypoxia during breath-hold dives has been investigated by Craig (1).

To elucidate the factors which might be responsible for unconsciousness and drowning in particular during

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repeated breath-hold diving, measurements of the oxygen uptake, carbon dioxide retention, and alveolar gas composition during and after a series of breath-hold dives have been performed.

METHODS

Test Subjects and Equipment

The experiments were performed in the tank shown in Fig. 1. Five submarine-escape training tank instructors and one of the authors served as test subjects (ages between 24 and 35 years); all were familiar with the special breath-hold diving technique employed. The main experiments were undertaken with three subjects: EE, height/weight (h/w) 183 cm/76 kg, vital capacity (VC) 6.6 liters; OL, h/w 192 cm/96 kg, VC 7.6 liters; and PP, h/w 179 cm/76 kg, VC 5.4 liters (all volumes BTPS). The residual volumes (RV) were calculated from the VC and the ratio between RV and VC in tank instructors, 0.29 (12). These lung volumes are somewhat—but not critically—greater than if measured while the subject is in water to neck level (11, p. 129). The VC determinations of a given subject agreed within ±0.1 liter by repeated experiments. Volumes of air were measured with two dry, low-resistance gas meters. Gas samples were analyzed simultaneously in a polarographic Clark oxygen cell and a Severinghaus Pco2 electrode (mean difference of duplicate samples 0.07 and 0.1 mm, sp \pm 0.38 and \pm 0.65 mm, n = 56 and 62, respectively). Analysis of the calibration gases was performed with a Scholander microanalyzer. Duplicate analyses of CO₂ and O₂ both agreed within 0.02 % O_2 and CO_2 .

Experimental Plan

Provided with a nose clip and a pair of pressure-compensated goggles (Royal Navy type), the diver performed dives to a thoracic level of 18.5 m in fresh water.

In the main experiments each test subject made a series of seven dives. At the start of each dive the subject expired to RV level and then inspired. The inspired volume was measured. At each surfacing a maximal expiration was performed and the volume measured. Expiratory air was collected in five Douglas bags for 16

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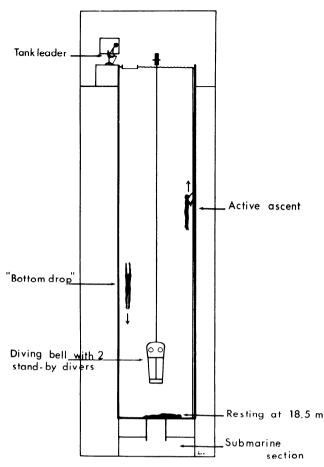


FIG. 1. Principal features of breath-hold dives in a 20-m sub-marine-escape training tank. The diving technique was described earlier (10).

min after the last dive (2 + 2 + 2 + 5 + 5 min), with the test subject resting at the edge of the tank in water up to his neck.

An additional experiment on a single breath-hold dive was performed in each of six subjects. The diver went to the bottom and after a varying period he delivered an alveolar sample to one of the investigators by means of the device shown in Fig. 2. Immediately after sampling the diver was brought into the diving bell (Fig. 1).

RESULTS AND DISCUSSION

The rate of descent was 0.8 m/sec (range 0.5-0.9) and the rate of ascent 1.5 m/sec (range 1.2-1.7). The mean diving time—total time under water—is shown in Table 2. In the single-dive series of the present study the diving time reached 124 sec, but attempts to reach maximal diving times have been discouraged and no cases of total unconsciousness occurred. For the sake of safety, surface intervals, total time between two successive dives, were between 1 and 2 min. Subjectively sufficient restitution to permit a new dive was obtained after the first few respirations in all subjects.

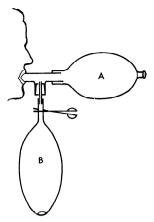


FIG. 2. Alveolar air sample apparatus. A, anesthesia balloon for dead space wash out (1,000 ml capacity in surface sampling, 500 ml in bottom sampling); B, football bladder(s) in single (triplet) mount for alveolar samples. Apparatus dead space 43 ml (water-filled when used at the bottom), id 22 mm. The contents of the rubber bladders and the Douglas bags were analyzed within 2 hr. By repeated examinations a fall of 1–3 mm Pco₂ was found in 1 hr, but no alteration of the Po₂ value could be ascertained.

TABLE 1. Mean alveolar air composition before and after seven repeated dives

Subj	Sample	Po ₂ , mm Hg	Pco2, mm Hg	R
EE	Last	126	27	1.40
$P_B = 746$		(119–133)	(20–32)	
	First	57	43	0.42
	Third	(50-65)	(37–48)	0 =0
	1 nira	104 (95-115)	35 (27–43)	0.78
		(95 115)	(2/ 43)	
OL	Last	134	25	1.60
$P_{\rm B} = 771$		(130-135)	(23–30)	
	First	64	37	0.27
		(48–69)	(32-42)	
	${f T}$ hird	118	28	0.80
		(116–122)	(24-30)	
PP	Last	137	23	1.90
$P_B = 771$	Base	(135-140)	(18–29)	1.3
//-	First	49	39	0.34
		(30-97)	(32-45)	•
	\mathbf{T} hird	III	30	0.69
		(97-118)	(24–40)	

Figures in parentheses are ranges from the seven consecutive dives. Alveolar samples are from the last expiration before a dive and from the first and third expiration after each dive.

Alveolar Gas Composition

Before diving. The results from repetitive diving are seen in Table 1, where the mean predive tensions (last) illustrates the degree of predive hyperventilation (here defined as a ventilation producing a Paco2 below normal level). Most dives were performed after mild hyperventilation (subject EE). Dives after violent hyperventilation were unpleasant (subject PP), mostly because of dizziness during the descent. The mean results of EE and OL show that a certain degree of hyperventilation—balancing between the benefit of an increase in diving time and the

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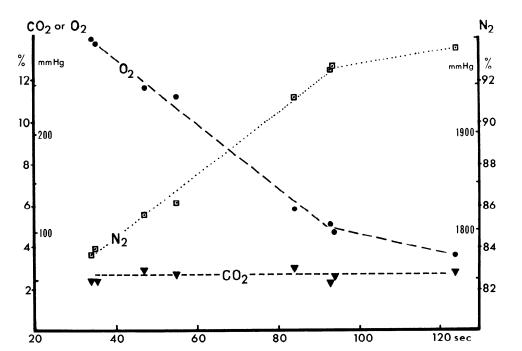


FIG. 3. Relation between diving time and oxygen, carbon dioxide, and nitrogen percentages in alveolar samples taken at 62 ft (18.5 m) of depth during single dives. The alveolar gas tensions at 2.85 atm abs are also shown.

danger of vigorous hyperventilation—was present before such dives. This occurred spontaneously.

At end of dive. Twenty-one samples (7×3) were obtained after repeated dives to 62 ft. Similar to findings by others in 33-ft simulated dives (8), average PA_{CO_2} values were near normal in all subjects (Table 1). In the very first expiration after each dive the PA_{O_2} values were usually about 60 mm. Two breaths later they were near normal. Some of the PA_{O_2} values of PP were very low (minimum 30 mm), primarily because of the longer diving times. Simultaneously the PA_{CO_2} had not even reached normal levels. When surfacing the diver was cyanotic and at the border of unconsciousness (1, 8, 9).

In evaluation of the relative contribution of hypoxia and hypercapnia to unconsciousness it would be desirable to know the gas tensions in the blood perfusing the central nervous system (cerebral cortex and nervous tissue related to respiratory control) at the end of the dive. The alveolar air partial pressures at the time of sampling does not ideally depict these tensions. Thus it must be recalled that the alveolar O₂ and CO₂ tensions have been falling rapidly during the fast ascent due to the alveolar gas expansion accompanying the decompression, whereas the decompression does not by itself affect the tensions in the tissues. Since the tissue gas tensions do not momentarily drop to the tensions of the blood entering them and since the passage of blood from the lungs to the tissues takes time, the tissue tensions must be expected to "lag behind" the tensions of the alveolar air at the time of surfacing.

Single breath-hold dives. The results from samples obtained at the bottom are shown in Fig. 3. The carbon dioxide level is constant and the alveolar oxygen content shows a steady decrease with diving time. If a barometric

TABLE 2. Net changes of alveolar gases during a mean dive

Test Subj EE	Diving time, sec	ΔVo ₂ 665	ΔVco ₂ — 260	R 0.39
OL	(40-45) 47	864	-268	0.31
PP	(44–50) 71 (41–80)	673	-164	0.24

Figures in parentheses are ranges. All volumes are ml (STPD). Oxygen is transferred from the lungs to the body, carbon dioxide is given off from the body to the lungs (negative values).

pressure of 760 mm is assumed, an alveolar CO₂% of 2.7 will correspond to a PACO2 value of 57 mm at 18.5 m (2.85 atm abs). A PACO2 of 57 mm and the corresponding PA_{O2} (85 mm—see below) is in the border area of CO₂ narcosis (9). If the PAO, now is lowered toward anoxic levels, it is possible that the CO2 narcosis may add to or potentiate the effect of the anoxia. Assuming P_{ACO_2} = Pa_{CO_2} and pK = 6.1, the calculated pH of the arterial blood will be 7.30, if blood bicarbonate content is assumed normal. However, a formation of lactic acid occurs in the body during a deep dive as shown by Schaefer (12). A repetitive diving pattern may prevent complete oxidation of lactic acid if surface intervals are short. A nonrespiratory acidosis will then be added to the respiratory acidosis and lower pH further. In the longest of these dives the alveolar O2% is about 4, corresponding to a PAO2 value of 85 mm at 18.5 m. Some of these divers might have fainted if they had tried to ascend (surface PAO2 would have been about 25 mm and PACO2 about 20 mm Hg), but as described earlier they were all brought to the diving bell at the bottom (Fig. 1).

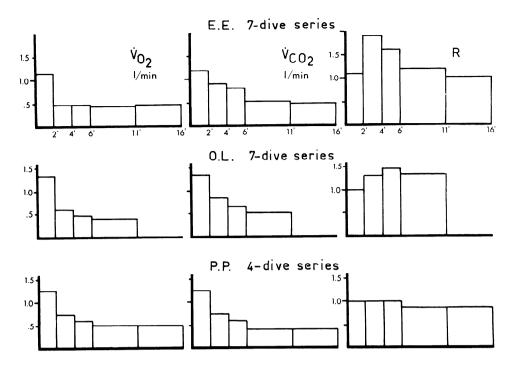


FIG. 4. Oxygen uptake, carbon dioxide elimination (liters STPD/min), and respiratory exchange ratio ($R = \dot{V}co_2/\dot{V}o_2$) after two seven-dive and one four-dive series.

Net Change of Alveolar Gases

The net transfer of gases during a "mean dive" (average of seven successive dives performed by one person since no systematic differences between dives could be elicited) was calculated as the difference between the volume of O₂ or CO₂ in the lungs just after the final inspiration before a dive (VI) and just after the first maximal expiration after the same dive (VE):

$$\Delta Vo_2 \text{ or } \Delta Vco_2 = (RV \cdot FA + Vi \cdot Fi) - (RV + VE) \cdot FA'$$

FA and FA' = fractions in end-expiratory sample from final expiration before the dive and in the first expiration after the dive, respectively. Dead space volume was assumed to be negligible in accordance with DuBois et al. (5). The end-expiratory air was assumed to be representative of alveolar gas concentrations in all parts of the lungs. No external air loss occurred (no air bubbles were observed) during the dives because of the tight nose clips. The results are seen in Table 2. If a respiratory quotient of 0.8-1.0 is assumed, this indicates an over-all retention of 300-600 ml CO₂ (13.5-27 mm) in a mean dive.

Between and After Dives

In a separate experiment mixed expiratory air was collected in one Douglas bag during the first three surface intervals in a series of dives (120–119–123 sec). The first expiration after each dive was included. Calculations showed an oxygen uptake per minute surface interval of 590 ml, a CO₂ elimination of 430 ml, and thus an alveolar exchange ratio (R) of 0.73.

The results obtained by fractional sampling of expired

air after a series of dives are shown in Fig. 4. After a short time R exceeds 1.0, then rises during the next minutes, and ultimately decreases toward normal levels. This pattern is less apparent after only four dives. The Douglas bag-sampling technique is not adequate for a detailed analysis but, in general, the R alterations reflect both a delay in emptying the CO2 stores of the body, in relation to the period for paying off the oxygen debt, and that the excess CO2 is relatively greater than the O2 deficit. Assuming the real, metabolic respiratory quotient to be 0.8 during the 16-min collecting period, subject EE and OL eliminate about 3,500 ml excess CO2 (157 mm) after seven dives and PP about 1,300 ml (STPD) after four dives. Part of this CO2 retention may be beneficial in overcoming the respiratory alkalosis incurred by the predive hyperventilation. However, in the final phase of the longest dives subjective symptoms similar to CO2 poisoning (headache, dizziness, respiratory distress, apprehension, and a fuzzy state with water swallowing) were present. Although the divers had normal alveolar CO2 tensions at the time of surfacing, the corresponding PACO2 values at the bottom would be abnormally high.

In breath holding at sea level and in underwater swimming just beneath the surface, hypoxia may play the dominant role, when swimmers and divers lose consciousness, as pointed out by Craig (1). However, in repeated dives to great depth, loss of consciousness (with the risk of drowning) may—in light of the above-shown CO₂ retention—be influenced by a CO₂ narcosis and acidosis to a greater extent than hitherto believed.

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