0.5 m/min. For the November 1961 *T. borchgrevinki*, all 63 fish swam at −1.8° to −1.7°C at an average of 7.3 m/min. The difference between the −1.8° to −1.7°C Table 1 data for the two seasons is only partly explained by the differences in acclimation times.

For *T. hansoni*: 3 of 8 (−1.8° to −1.7°C) swam at an average of 2.1 m/min; 1 of 8 (−0.8°C), at 2.4 m/min; none of 7 (+0.2° to +0.4°C) swam; 1 of 6 (+1.2°C) swam at 1.4 m/min; and none of 5 (+2.1° to +2.2°C) swam.

For *T. loennbergi*: 3 of 11 (−1.8° to −1.7°C) swam at an average of 3.5 m/min; 3 of 8 (−0.9° to −0.6°C), at 3.3 m/min; 3 of 4 (+0.2° to +0.3°C), at 3.2 m/min; 2 of 5 (+1.1° to +1.3°C), at 3.1 m/min; and the single fish at +2.4°C did not swim. The high b_s of 0.1124 appears to be a consequence of "labored" swimming activity.

Thus both the propensity to swim and the average swimming rate tend to decline with temperature over a very narrow temperature range. The "level of no excess activity" (*7*) with O_2 at near-saturation appears to be at about +2°C for the three antarctic species. By contrast, the tabulated data for the more eurythermal temperate species indicate that these fishes have lower swimming rates at lower temperatures. Possibly because the water viscosity increases greatly with decreasing temperature, the respiration-swimming coefficients tend to be larger at the lower temperatures and of the same order for both temperate and antarctic species (*8*).

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Alveolar Pathways during 90-Foot, Breath-Hold Dives

Abstract. The gas tensions of mixed expired and alveolar air were measured at various depths during descent and ascent. A reversed carbon dioxide gradient from the lungs into the blood was demonstrated at the 50-foot depth. At 90 feet, 45 percent of the pre-dive carbon dioxide content of the lungs had disappeared. There was no indication of a reversed oxygen gradient during ascent.

During the training of submarine crews in submarine escape procedures, such as "free or buoyant ascent," instructors at the escape training tank frequently hold their breath under water and perform "skin" dives to depths as great as 90 feet. The ascent is carried out by climbing up a line. (These diving maneuvers are similar to those practiced by sponge and pearl divers.) The escape tank at the New London Submarine Base afforded us an opportunity to study the pulmonary gas exchange during this type of diving.

In previous investigations (*1*, *2*) alveolar gas samples were obtained from divers at the surface, at 90-foot depth, and after their return to the surface of the escape training tank. Results showed that a considerable amount of the predive CO_2 content in the lungs had disappeared during descent to 90 feet, thus indicating a transfer of CO_2 from the lungs to the blood. On the basis of theoretical equations, DuBois (*3*) had predicted such changes in pulmonary gas exchange during diving. They were subsequently confirmed in simulated breath-hold dives in dogs (*4*) and recently in men (*5*). Our report presents, for the first time, detailed data on alveolar pathways during breath-hold dives and gives direct evidence of the existence of a reversed CO_2 gradient during descent.

The experiments were carried out with an experienced diver who was well trained as a subject in respiratory experiments and whose lung volumes had been determined repeatedly. Prior to the descent the diver exhaled to residual volume and then inhaled 4 liters from a spirometer. After reaching a predetermined stopping point, he exhaled the major part of his expiratory volume through a mouth-piece into the first bag used for the collection of mixed expired air; he exhaled the remainder into a second bag used to collect "alveolar air." (The latter usually contained 10 to 20 percent of the total expiratory volume.) The bags were brought to the surface. Gas samples from the bags were collected in mercury tonometers and the volumes of the bags were measured. Gas analysis was carried out in duplicate with a Scholander 0.5 gas analysis apparatus. The CO_2 and O_2 content (STPD) in the lungs at various depths was calculated from the measured gas tensions and volumes of mixed expired and alveolar air, the known volume of residual air, and the total dry gas pressure in the lungs.

Results plotted in the O_2–CO_2 diagram show the alveolar pathways during natural breath-hold dives to a 90-foot depth (Fig. 1). The resting alveolar pCO_2 of 40 mm-Hg was lowered to 29.5 mm-Hg by the inhalation of 4 liters of air. The alveolar pO_2 increased correspondingly. During descent, the rising ambient pressure compresses the lungs and the alveolar gas tensions are quickly elevated. At 25 feet, alveolar pCO_2 reached 46 mm-Hg. The normal "virtual" venous (oxygenated venous blood) pCO_2 is 48 to 50 mm-Hg, which corresponds to the crossover point between CO_2 elimination and reabsorption (Fig. 1). At this point, CO_2 is already transferred from the lung alveoli into the pulmonary capillary blood, a condition which is indicated by the disappearance of the normal gradient between CO_2 tensions in mixed expired and alveolar air. At 50 feet, pCO_2 in mixed expired air is 6 mm-Hg higher than the alveolar pCO_2. At 90 feet, only mixed expired samples could be obtained because of the small lung volume. However, it can be assumed that under these conditions mixed expired air and alveolar gas tensions have reached an equilibrium. In spite of the large ambient pressure increase, there is little change in the measured alveolar

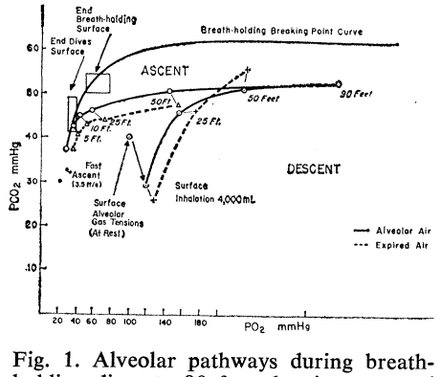

Fig. 1. Alveolar pathways during breath-holding dives to 90 feet showing reversed CO_2 gradient. At 50 feet pCO_2 mixed expired air is 6 mm-Hg higher than pCO_2 "alveolar air." Surface breath-holding breaking point curve drawn for comparison with diving breath-holding curve. End dive alveolar pCO_2 decreased with increasing rate of ascent reaching 30 mm-Hg at 3.5 ft/sec.

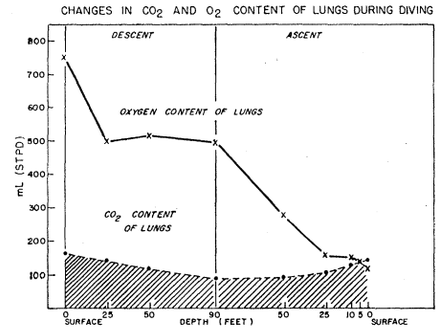

Fig. 2. CO_2 and O_2 content of lungs during diving (milliliters, STPD) calculated from measured gas tensions and volumes of mixed expired and alveolar air at various depths, the known residual volume and the total dry gas pressure in the lungs.

pCO_2 between 50 and 90 feet of depth because of the CO_2 uptake in the blood. During ascent alveolar pCO_2 falls less than would be predicted from the ambient pressure decrease because CO_2 is again entering the lungs from the blood. The influx of CO_2 into the lungs during ascent is regulated, at least in part, by the speed of ascent.

Figure 1 shows alveolar CO_2 and O_2 tensions measured after surfacing from dives in which three different rates of ascent were used. The highest alveolar CO_2 tensions were obtained in a group of six subjects (standard deviation of the mean indicated in box form) whose ascent rate averaged 1.9 ft/sec. The subject, for whom the whole alveolar pathways during diving were determined, had an ascent rate of 2.3 ft/sec and his end dive values were lower. In three cases the rate of ascent was increased to 3.5 ft/sec resulting in alveolar pCO_2 tensions as low as 30 mm-Hg.

Table 1. Pulmonary CO_2 and O_2 exchange during breath-hold dives to 90 feet. The number of experiments is shown in parentheses.

Depth (ft)	Alveolar partial pressure (mm-Hg)		Change in gas (%)	
	Measured	Theoretical	Tension	Content
	CO_2 exchange			
0	30 (3) 1			
25	46 (7) 2	52	-11	-12
50	50 (3) 1	74	-32	-27
90	52 (2)	110	-52	-45
	O_2 exchange			
0	118 (3) 1			
25	153 (7) 13	206	-26	-33
50	227 (3) 6	295	-23	-32
90	344 (2)	437	-28	-34

Breath-holding experiments on the surface equal in time to the diving experiments were carried out on the same six subjects. The alveolar pCO_2 values at the breath-holding end points were significantly higher (7 mm-Hg) than the end dive CO_2 tensions (Fig. 1). Both sets of data (shown in the boxes) fall on the breath-holding point curve of Otis, Rahn, and Fenn (*6*). The diving breath-holding curve reaches a plateau at around 50 mm-Hg, which is 10 mm-Hg lower than the breath-holding breaking point curve and 10 mm-Hg higher than the normal alveolar air curve.

The lower alveolar pCO_2 values during breath-hold dives are caused by the CO_2 transfer from the lungs into the blood. A quantitative estimation of the CO_2 transfer is given in the changes in the CO_2 content of the lungs (Fig. 2). The CO_2 content decreased during descent to 90 feet linearly from 163 ml to 89 ml and remained approximately at this level during the first part of ascent to 50 feet. Only in the latter part of ascent was the normal CO_2 gradient re-established as the lungs started to refill with CO_2.

The O_2 content of the lungs does not change linearly. During the 15 seconds of effortless descent to 25 feet, 250 ml of O_2 are transferred from the lungs while the estimated O_2 consumption (based on O_2 uptake during resting) is 59 ml during this period. No further O_2 transfer occurs during descent from 25 to 90 feet. Climbing up the line during ascent results in another decrease in O_2 content which does not cover the O_2 cost during this period. Toward the end of the dive, the O_2 transfer is reduced to minimal values. These peculiar changes in O_2 content cannot be explained adequately at this time. It was speculated that the rapid decrease in O_2 tension during the last part of ascent might lead to a reversed O_2 gradient from the blood to the lungs (*5*). However, the measurements of expired and alveolar O_2 tensions obtained in breath-hold dives did not show any evidence for this hypothesis. The extremely low O_2 tension found after surfacing indicates the existing danger of hypoxia. One of our subjects became briefly unconscious upon reaching the surface, but recovered after the first deep breath.

In Table 1 the measured alveolar CO_2 and O_2 tensions are listed together with the gas tensions which should theoretically exist at various depths, based on alveolar gas tensions measured at the surface prior to the dive. The differences give an estimation of the changes in O_2 and CO_2 content of the lungs which compare reasonably well with the changes calculated from the measured expiratory and alveolar volumes and gas concentrations and the gas content of the known residual volume (*7*).

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Reinforcing Brain Stimulation in Competition with Water Reward and Shock Avoidance

Abstract. Employing response rate as the index of reinforcing strength in self-stimulation experiments is questioned. With water reward or shock avoidance placed in competition with brain stimulation, self-stimulation rate does not reflect relative reinforcement value. The results agree with preference tests which show that, for a given electrode site, stimulus intensity, not rate, is directly related to reward strength.

An assumption underlying most self-stimulation studies is that performance rate reflects the strength of the reward. However, it has recently been demonstrated that with rewarding, intracranial stimulation (ICS) rate may be a misleading index (*1*). When provided with a choice, animals often exhibit a preference for either an electrode placement or a stimulus intensity which supports a significantly lower rate than