

# DIVING (UNDERWATER) FOR PULMONARY PEARLS

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Because man is not made for the aquatic environment, he stretches the limits of cardiopulmonary adaptation by diving underwater for recreational, research, military or occupational purposes.

## Breathhold Diving

Korean and Japanese women who dive for a living have long had a reputation of being able to breathhold dive for prolonged periods at depths down to 20m (66 feet). Even more amazing, some competitive breathhold divers can descend down to 112m (370 feet). What sets the depth limit for man is uncertain. It used to be thought that the crushing effect of hydrostatic pressure on the thorax would be the limiting factor. Assuming a diver takes a breath to total lung capacity (TLC) before diving, the volume of gas in the lungs will decrease below TLC upon descent on account of the surrounding hydrostatic pressure. Thus by Boyle's law, a depth (and critical pressure, Pcrit) will be reached when TLC is reduced to the volume of residual volume (RV) at sea-level:

$$TLC \times P_b = RV \times P_{crit}$$

where  $P_b$  is the barometric pressure at sea-level. Based on average figures, a Pcrit of 4 atmospheres (ATA) would reduce TLC to RV, and this pressure is reached at about 30m (since each 10m underwater adds 1 ATA to the ambient pressure):

$$P_{crit} = P_b \times TLC/RV$$

Taking average values for TLC and RV as 6 L and 1.5 L respectively, and since  $P_b$  is 1 ATA,

$$P_{crit} = 6/1.5 = 4 \text{ ATA}$$

However, competitive divers can descend to 112m or 12 ATA. Assuming an average TLC of 6 L, the calculated lung gas volume at 12 ATA would be well below RV, about 0.5 L. If there was a proportionate reduction of thoracic volume, the chest would be crushed! There is evidence to suggest that although the gas volume is reduced greatly by the pressure, the thoracic cage is spared the crushing reduction of volume by an increase in thoracic blood volume<sup>(1)</sup>. It is estimated that about a 500 - 1,000 ml of blood may enter the thorax as a result of the ambient hydrostatic pressure<sup>(2, 3)</sup>.

How long can a diver remain underwater? This depends on the rate of rise of PaCO<sub>2</sub>. The diver senses his need to

surface when the PaCO<sub>2</sub> approaches the break point of about 55 mmHg. Assuming that PaCO<sub>2</sub> rises at 4-6 mmHg/min in the absence of ventilation, the time limit would be about 2-3 minutes. It is important to reiterate that it is the PaCO<sub>2</sub>, not the PaO<sub>2</sub>, which warns the diver of the need to surface. Voluntary hyperventilation preceding a breathhold dive is therefore dangerous and believed to be responsible for some deaths<sup>(4)</sup>. In the hope of prolonging dive duration, inexperienced swimmers sometimes hyperventilate before submerging. This results in a greater loss of CO<sub>2</sub> than in a gain of O<sub>2</sub> since the body has a low O<sub>2</sub> storage capacity. The low starting PaCO<sub>2</sub> would then give the diver more dive time before break point PaCO<sub>2</sub> is reached. However the PaO<sub>2</sub> may fall to perilous levels before the CO<sub>2</sub> break point is reached and loss of consciousness may then ensue. Thus hyperventilation should not be practised prior to dives.

At dives greater than 1.2m (4 feet), alveolar pressure rises (secondary to ambient hydrostatic pressure) such that the alveolar CO<sub>2</sub> is absorbed back into the blood<sup>(5)</sup>:

$$\begin{aligned} \text{PACO}_2 \text{ at } 1.2\text{m} &= (1.12 \times P_b - 47) \times \text{PACO}_2 \text{ at } 1 \text{ ATA}/P_b \\ \text{Where } P_b &= 760 \text{ mmHg} \\ \text{and } \text{PACO}_2 \text{ (alveolar CO}_2 \text{ tension) at } 1 \text{ ATA} &= 40 \text{ mmHg,} \\ \text{PACO}_2 \text{ at } 1.2\text{m} &= 45 \text{ mmHg} \end{aligned}$$

Since mixed venous O<sub>2</sub> tension is also 45 mmHg, CO<sub>2</sub> transfer cannot occur at this depth. At greater depths, the diver not only fails to excrete CO<sub>2</sub>; he may actually absorb some CO<sub>2</sub>! Another remarkable phenomenon is the effect of high ambient pressures on O<sub>2</sub> exchange in the lungs. The increased alveolar O<sub>2</sub> pressure as a result of progressive descent is advantageous, as this allows O<sub>2</sub> to be driven into the blood even at low fractional concentrations. Thus, even if alveolar O<sub>2</sub> concentration is only 3%, at depths of 30m (4 ATA) O<sub>2</sub> transfer is still possible because the alveolar partial pressure of O<sub>2</sub> (PAO<sub>2</sub>) would be:

$$0.3(4 \times 760 - 47) = 98 \text{ mmHg.}$$

However, this advantage quickly turns into a hazard during ascent, when the absolute PAO<sub>2</sub> falls (as a result of falling ambient pressure).

## Snorkelling

In this situation, the lungs are exposed to barometric pressure at the surface (1 ATA) through a breathing tube, but the body, being immersed in water, is subjected to hydrostatic pressure as well. A dramatic (but unrealistic) picture of this is often seen in the movies when the hero, being hounded by hordes of villains, dives into a nearby river and hides underwater for a prolonged period while breathing through a hastily plucked hollow reed. He resurfaces only to energetically pursue his exploits. In real life, the hero would have to be careful not to submerge too deep and has to select a reed which is short (low dead space) and wide (low resistance), otherwise the reed would pose a high resistance and an additional dead space which the encumbered inspiratory muscles have to overcome. The

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maximum inspiratory pressure a normal person can generate is 100 - 150cm H<sub>2</sub>O, thus the maximum depth tolerable would be about 1 m only.

### Scuba Diving

Self-contained underwater breathing apparatus (SCUBA) overcomes the problem of snorkelling by providing gas at the appropriate pressure to the lungs, and partly overcomes the problem of breathhold diving by providing supplies of O<sub>2</sub> through a gas tank and allowing ventilation for the removal of carbon dioxide. However, all is not smooth sailing just because of SCUBA.

There is still the problem of barotrauma. Air in partially enclosed spaces such as the sinuses are also compressed on descent, and since the bones that wall the sinuses cannot collapse, the consequent gas volume loss is made up for by mucosal oedema and haemorrhage. If the middle ear drainage vents are not equally efficient in equilibrating pressures, the difference in pressure of both sides can give rise to severe giddiness. On ascent, expanding gas in the middle ear can rupture the tympanum. Pulmonary barotrauma occurs when the diver does not exhale to vent the expanding gas. The result could be pneumothorax, mediastinal emphysema, subcutaneous emphysema, or even arterial air embolism.

Another peril is narcosis from inert gas at high partial pressures. Nitrogen narcosis can occur at any depth below 30m (100 feet) and is called "rapture of the depths". The diver appears intoxicated and has a temporarily impaired intellect. The "rapture" rapidly disappears on ascending.

High ambient pressures result in increased gas density. This in turn causes airflow resistance to rise in the airways with turbulent flow regimes. Breathing helium-oxygen mixtures overcomes this to a certain extent.

Finally, SCUBA diving may be complicated by decompression sickness which is due to the rapid fall in ambient pressure on ascent. Whereas the complications resulting from the high ambient pressures per se may be explained by Boyle's law, decompression sickness is explained on the basis of

Henry's law. Henry's law states that the amount of gas dissolved in a liquid is directly proportional to the partial pressure of the gas. Thus, upon descent the ambient pressure (and lung gas pressure) rises and forces more gas into solution in the blood. While the increased amount of dissolved oxygen is used up in metabolism, nitrogen is not. The SCUBA gear allows the diver to stay underwater for prolonged periods, so that nitrogen gradually accumulates in solution, especially in adipose tissue. If subsequent ascent is too rapid, the fall in ambient pressure will be accompanied by a fall in the partial gas pressure. The lower gas pressures will then be unable to sustain the large amounts of dissolved gas (resulting from earlier high partial pressures) and the supersaturated gas solution may liberate the gases much in the same way gas is liberated in a Coke bottle when the cap is removed (and the ambient pressure in the bottle suddenly released). The clinical manifestations depend on the site of the gas formation and embolisation, and include joint pains (bends) and chest discomfort (chokes).

Divers are therefore taught to follow strict schedules of ascent with stops in between. However, even adherence to such schedules does not guarantee freedom from complications. This is evident in the reports in this issue of the journal<sup>(6)</sup> where some of the victims were trained and experienced naval divers.

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