Partial Pressure Physics
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Partial-pressure effects: The partial pressure of a gas is determined by the concentration of the gas and the ambient pressure, eg, the concentration of O2 in air is about 21%, and the partial pressure of O2 in air at surface (1 atm abs) is about 0.21 atm. The concentration of O2 in air remains the same at depth, but the partial pressure reflects the increasing pressure and compression of the gas. At 2 atm abs, the number of O2 molecules per unit volume is twice what it is at the surface, and the partial pressure is double.

The physiologic effects of gases are related to their partial pressure and change according to depth. Toxic effects appear as the partial pressure of O2 increases. Pulmonary oxygen toxicity can cause lung damage with extended exposure to a PO2 above 0.6 atm (equivalent to 60% O2 at surface or 30% O2 at 33 ft). Oxygen convulsions may occur, especially in working dives, if the PO2 approaches or exceeds 2 atm (eg, 100% O2 at 33 ft or 50% O2 at 99 ft).

Increased partial pressures of N2 produce nitrogen narcosis, a condition resembling alcohol intoxication. In divers who breathe air, this effect becomes noticeable at 100 ft or less. It is generally incapacitating at about 10 atm abs (300 ft), where it produces an anesthetic effect resembling that of 30% nitrous oxide at sea level. (Helium lacks this anesthetic property and is used in place of N2 as the diluent for O2 in deep diving.)

Partial pressures of O2 and CO2 in alveolar gas are modified by the
pressure of depth in breath-hold diving and in underwater swimming without breathing apparatus. The impulse to return to the surface and resume breathing depends largely upon CO2 buildup in the body. A breath-holding diver may hyperventilate beforehand to extend time underwater; this blows off CO2 but adds little to stores of O2, and may then cause unconsciousness from hypoxia without warning before PCO2 rises enough to become an effective stimulus.

Diving to a significant depth during the breath-hold complicates the situation by elevating the PO2 and permitting extended O2 uptake at depth. A diver who has "pushed the limits" under those circumstances may lose consciousness when alveolar PO2 falls to a low level on ascent. This phenomenon is probably responsible for many unexplained drownings among spearfishing competitors and others who do extensive breath-hold diving. The term shallow-water blackout is sometimes applied, but it is best reserved for its original meaning: unconsciousness from CO2 buildup in rebreathing types of scuba. (Hypoxia is also a potential problem in rebreathing units if O2 is displaced by excess N2.)

Carbon dioxide poisoning: In normal individuals on land, hyperpnea or breathlessness usually provides ample warning of increased CO2 in inspired gas. Such a response may be more the exception than the rule under water, especially where high PO2 and exertion are also factors. Some individuals develop spontaneous CO2 retention through an inadequate increase in pulmonary ventilation during exertion. Whatever the source, abnormally high PCO2 per se can cause loss or impairment of consciousness at depth and can also increase the likelihood of O2 convulsions and augment the severity of nitrogen narcosis. The tendency to retain CO2 may be suspected in divers who frequently experience post-dive headaches or pride themselves on low air-use rates.