

Oxygen Partial Pressure - Hazards and Safety

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Abstract

The partial pressure of oxygen (PO₂) is a critical consideration in diving, particularly technical diving. Oxygen supports life and can enhance decompression efficiency, but it can also produce life-threatening toxicity. Dive planning for oxygen can be challenging with conflicting limits, recommendations, and practice. This paper reviews the role of oxygen, known and potential hazards of extreme partial pressures, changes in prescribed upper limits and the rationale for them, and knowledge gaps to be overcome. The goal is to provide insight to aid in the navigation of best practice regarding oxygen to help optimize diving safety.

Keywords: closed-circuit, decompression, diving safety, open-circuit, rebreather, toxicity

Introduction

Oxygen is necessary to sustain life, but it plays a more complex role in diving safety. Increased fractions can improve decompression efficiency by reducing the inert gas fraction of inspired gas, and a high partial pressure of oxygen (PO₂) is a staple in the treatment of decompression sickness. Problematically, however, too much oxygen can also create health and life-threatening toxicity.

The consideration of oxygen toxicity related to diving is typically focused on pulmonary and central nervous system (CNS) effects. Pulmonary toxicity is irritation of the lungs caused by prolonged breathing, often many hours, of substantially elevated oxygen concentrations. This can develop with a PO₂ as low as 0.5 ATA given sufficient exposure time. Signs and symptoms include persistent unproductive cough, a sense of tightness in the chest or discomfort during inspiration, and retrosternal pain. CNS toxicity involves an insult to the brain caused by exposure to higher PO₂ and can develop after relatively short periods given high enough values. Signs and symptoms include twitching of facial muscles, tunnel vision, nausea, paresthesia, unconsciousness, and convulsions. Subtle manifestations may appear first, but cases can evolve rapidly or immediately present with fully incapacitating effects.

Pulmonary oxygen toxicity is generally not a major concern in diving. While the lungs can be stressed, mild symptoms typically resolve without consequence following a return to air breathing. CNS toxicity represents a much greater threat since a loss of consciousness underwater is accompanied by a high risk of drowning.

Oxygen toxicity is generally not a concern in air diving since the risk of impairment from nitrogen narcosis normally restricts maximum depths to those where PO₂ is not problematic. Oxygen is a greater hazard with enriched air nitrox since the threshold for toxicity is found at much shallower depths. Oxygen content is a critical consideration in technical diving, for concerns of both hypoxia and hyperoxia.

Both practice and guidance regarding the safe upper limits of oxygen breathing have evolved over time. A host of variables must be considered to assess the effective risk. The goal here is to provide the background needed to understand PO₂ choices and related practice.

The Oxygen Continuum

Figure 1 depicts a continuum of the physiological effects and common operational guidance concerning oxygen. PO₂ is the standard measure, representing the product of the fraction of oxygen (FO₂) multiplied by the ambient pressure (PO₂ = FO₂ * ambient pressure). PO₂ is normally reported in atmospheres absolute (ATA). For example, the PO₂ of air at sea level pressure is 0.21 ATA (0.21 * 1 ATA). The PO₂ of air at 20 meters of seawater (msw; 66 feet of seawater [fsw]) is 0.63 ATA (0.21 * 3 ATA).

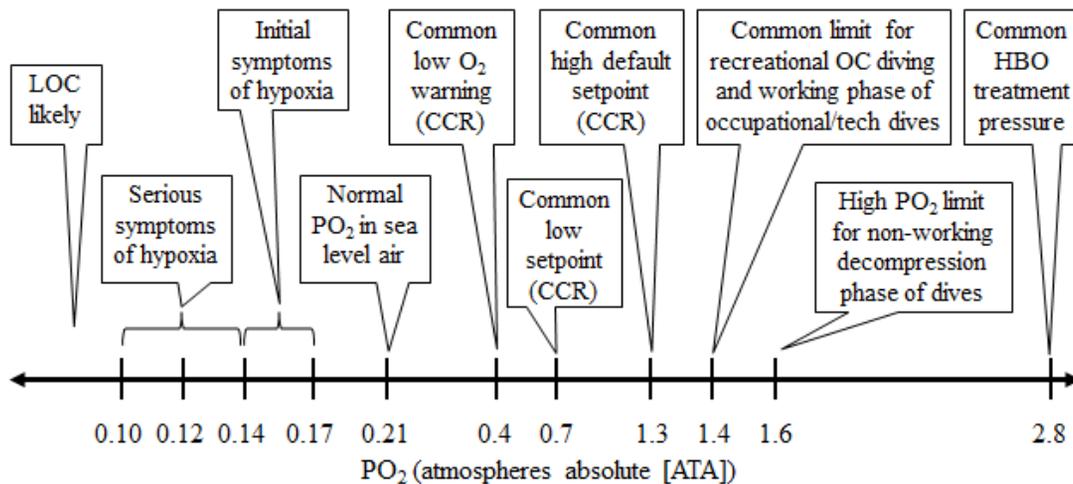


Figure 1. Continuum of physiological effects and operational guidance for oxygen partial pressure in diving and hyperbaric applications. Abbreviations: LOC - loss of consciousness; CCR - closed-circuit rebreather; OC - open-circuit; HBO - hyperbaric oxygen

Initial symptoms of hypoxia are expected when PO₂ falls to or below 0.17 ATA. This is approximately equal to the oxygen concentration in air at an altitude of 1676 m (5500 ft). Loss of consciousness is an increasing risk as PO₂ falls below 0.10 ATA, equivalent to the oxygen concentration in air at an altitude of 5800 m (19,000 ft).

The normal state of divers breathing gas underwater is hyperoxic, thus well able to meet normal metabolic needs. For example, the PO₂ of air breathed at 1 msw (3 fsw) is already 0.23 ATA (0.21 * 1.1 ATA). The PO₂ of air breathed at 3 msw (10 fsw) is 0.27 ATA (0.21 * 1.3 ATA). Breathing modestly elevated PO₂ is not problematic. It would typically take many hours of exposure to develop symptoms of pulmonary irritation at a PO₂ of 0.5 ATA (14 msw/45 fsw; 0.21 * 2.4 ATA). The time it takes for pulmonary toxicity to develop decreases at higher PO₂, but the much more immediate concern is the risk of CNS toxicity at higher PO₂. Symptoms of CNS toxicity have been reported with pure oxygen breathing at 3 msw (10 fsw; 1.3 ATA) (Arieli et al., 2006), but they are much more likely to be experienced by divers breathing a PO₂ of 1.6 ATA or higher. Harabin et al. (1995) reviewed 42 cases with CNS toxicity in 688 hyperbaric chamber exposures (6% incidence). All subjects were immersed and exercising discontinuously at moderate levels (4 min on, 2 min off) in 22°C water. While the exposures varied in complexity, nine cases with symptoms were recorded at 1.6 ATA PO₂, one with convulsions. The rest of the cases were documented at higher PO₂.

Underwater Breathing Apparatus

Open- and closed-circuit breathing systems deliver oxygen to divers in fundamentally different ways. Open-circuit systems deliver a fixed FO₂ and a variable PO₂, with the PO₂ increasing as a function of depth. Closed-circuit systems deliver a variable FO₂ and a fixed PO₂, with the PO₂ stable across depth.

Open-circuit systems do not usually measure the oxygen content delivered to divers. Closed-circuit rebreathers monitor PO₂ in the breathing loop as a priority. Manual rebreathers typically introduce a modest continuous flow of oxygen into the loop, often through constant flow orifice or needle valves, intended to meet minimal estimated basal needs, with divers manually injecting additional oxygen as required. Electronic rebreathers employ computerized controllers to monitor and automatically inject oxygen through electromechanical (solenoid) valves to tightly maintain PO₂ at a setpoint.

There are typically two oxygen setpoints used by electronic rebreathers, low and high. The low setpoint, typically 0.7 ATA PO₂, is needed on the surface or in shallow water where the maximal achievable PO₂ is limited. If a setpoint exceeds the maximal achievable PO₂, the solenoid valve will fire continuously and ineffectively. The high setpoint, often 1.3 ATA PO₂ but variable with unit and/or user-selected settings, can be maintained as long as the ambient pressure is greater than the setpoint.

Rebreathers provide visual and sometimes audible warnings when the PO₂ falls outside the acceptable range. A low oxygen warning is typically triggered at a PO₂ of 0.4 ATA. This is a hyperoxic state, but active warning at this point allows the diver time to respond before hypoxic conditions can develop.

High Oxygen Concerns

A high PO₂ setpoint is desirable to reduce decompression stress on divers. The higher the setpoint, the less inert gas will be taken up during the descent and bottom phase, and the more that will be eliminated as the diver ascends, particularly so in the latter part of the ascent. The PO₂ of air breathed on open-circuit is 1.3 ATA at roughly 52 msw (171 fsw). A closed-circuit rebreather operating on a fixed 1.3 ATA setpoint would deliver a progressively lower partial pressure of inert gas from this point through the ascent. With a PO₂ setpoint of 1.6 ATA, a rebreather diver would inspire virtually no inert gas at a depth of 6 msw (20 fsw).

Minimizing decompression stress is important, but oxygen toxicity is also a critical consideration of high PO₂. It is tempting to downplay concerns of high PO₂ when hyperbaric oxygen (HBO) therapy used to treat decompression sickness can expose patients to a PO₂ of 2.8 ATA, but there are critical differences that reduce the CNS toxicity risk in a dry chamber. HBO treatment includes scheduled air breaks; patients remain at rest and, most importantly, a patient convulsing in a chamber can be simply managed by removing the oxygen mask or hood and clearing the airway as required.

Divers in the water face much greater hazard with any impairment of consciousness. Self-rescue quickly becomes untenable and rescue by others is far from assured. Full-face masks or mouthpiece retaining straps reduce the likelihood of losing the mouthpiece (Haynes, 2016), but they do not address the airway compromise associated with vomiting into a mouthpiece or buoyancy issues. The threshold for seizures may also be lower in the water, driven by exercise, rising arterial CO₂ (associated with exercise or inadequate CO₂ removal), increased work of breathing, general stress, cold (or cool) stress, and a wide range of medications or drugs.

The potential for visual changes following prolonged exposure to high PO₂ is also known. Reversible hyperbaric myopia can develop in both HBO patients receiving multiple treatments and active divers. More worrisome are the reports that high PO₂ may also promote cataract formation in an irreversible manner in patients receiving prolonged series of HBO treatments (Palmquist et al., 1984; Gesell and Trott, 2007). There is little evidence to date, but there are legitimate concerns that frequent and long duration dives at high PO₂ could create a cataractogenic risk for divers.

High Oxygen Limits

The PO₂ limit of 1.6 ATA has a long history in scientific, occupational, and technical diving. There is, however, a broad shift in practice towards more conservative bounds. The default high setpoint on many modern rebreathers is 1.3 ATA. It is not uncommon for lower PO₂ setpoints to be used for part or all of long exposures.

The guidance on PO₂ limits has been modified by some institutions within the past five years. The US National Oceanic and Atmospheric Administration (NOAA) reduced the maximum PO₂ during the working phase of the dive from 1.6 to 1.4 ATA, effective September 5, 2015. The 1.6 ATA limit continued to be allowed during the decompression phase of dives. The Canadian Standards Association Z275.2 committee, which develops standards accepted for commercial and scientific diving in Canada, ended a long period of debate quickly after the NOAA decision was announced. The maximum PO₂ was similarly reduced from 1.6 to 1.4 ATA for the working part of the dive, effective November 18, 2015.

Rationale for Reduced PO₂ Limits

PO₂ limits should reflect a compromise between the benefit of reducing decompression stress and the hazards of pulmonary, CNS, and ocular toxicity. High PO₂ values that are attractive from a decompression point of view can push other safety boundaries. The NOAA oxygen exposure limits are quite restrictive at high PO₂, allowing a maximum single exposure of only 45 minutes at 1.6 ATA (Dinsmore and Bozanic, 2013). The allowable time increases to 150 minutes at 1.4 ATA and 210 min at 1.2 ATA (Table 1). While these limits were incompletely tested, they do provide points of reference for safety planning. A good risk-benefit balance can be found by moderating PO₂ during the deep phase of a dive where the absolute reduction in inert gas uptake is modest and the life threat from compromised consciousness is extremely high. A lower set point at depth would allow more flexibility to increase the PO₂ during the shallower portion of the ascent where the decompression benefit of high PO₂ is great and the life threat of compromised consciousness somewhat reduced.

Table 1. NOAA oxygen exposure limits (from Dinsmore and Bozanic, 2013).

Pressure (ATA)	Maximum Single Exposure (min/h)
1.6	45 / 0.75
1.5	120 / 2.0
1.4	150 / 2.5
1.3	180 / 3.0
1.2	210 / 3.5
1.1	240 / 4.0
1.0	300 / 5.0
0.9	360 / 6.0
0.8	450 / 7.5
0.7	570 / 9.5
0.6	720 / 12.0

The argument that the 1.6 ATA PO₂ limit is well-tested through time must be considered cautiously. The existence of a limit is immaterial unless a large number of exposures are made to the limit. Exposures conducted partially or wholly at lower PO₂ will not provide insight into the safety of higher PO₂. There is a sufficient combination of documented evidence and theoretical concerns to consider more than the benefits of high PO₂ for safe practice. Conservatism is warranted from an institutional perspective, since standards should provide reasonable protection for individuals with an unknown level of susceptibility and a normal range of activity.

Overcoming Knowledge Gaps

More research is needed to better understand both the hazards and benefits of high PO₂. It is difficult to conduct research on potentially high consequence events, but a combination of observational and laboratory studies could contribute to the effort. Observational studies should include close, long-term surveillance of PO₂ profiles, capturing details on normal practice and problematic events. Data from closed-circuit systems with tightly controlled set points and continuous records would be most useful. The information typically available with open-circuit exposures, particularly related to manual breathing gas switches, is much less reliable. Integrating dive records and health data would help to characterize risk profiles. The American Academy of Underwater Science could play an important role in gathering relevant field data, evaluating cases related to oxygen problems, and reporting in the literature.

Laboratory studies are needed to further assess inter- and intra-individual variability in oxygen toxicity response and interactions that may affect severe toxicity risk. Variables and interactions of interest include absolute PO₂, exposure time, rate changes in PO₂, exercise intensity, PCO₂, thermal state, breathing resistance, and common medications, drugs, nutritional supplements, and other dietary elements. Addressing all would be a huge undertaking. Animal studies could be useful to investigate some relationships, but substantial human research is required for maximal relevance to human divers.

Conclusions

High PO₂ offers both benefit and risk to divers. A reduction in decompression stress must be balanced against an increased risk of oxygen toxicity. Change in practice has led to some decrease in PO₂ norms, but further research is needed to better understand variables that affect susceptibility to oxygen toxicity. Additional information could facilitate further refinement of guidelines and practice.

Literature Cited

- Arieli R., T. Shochat, and Y. Adir. 2006. CNS Toxicity in Closed-circuit Oxygen Diving: Symptoms Reported from 2527 Dives. *Aviation, Space and Environmental Medicine*, 77:526–532.
- Dinsmore, D. A., and J. E. Bozanic. 2013. *NOAA Diving Manual: Diving for Science and Technology*, 5th ed. North Palm Beach, FL: Best Publishing Company.
- Gempp, E., P. Louge, J.E. Blatteau, and M. Hugon. 2011. Descriptive Epidemiology of 153 Diving Injuries with Rebreathers Among French Military Divers from 1979 to 2009. *Military Medicine*, 176:446–450.
- Gesell, L. B., and A. Trott. 2007. De Novo Cataract Development Following a Standard Course of Hyperbaric Oxygen Therapy. *Undersea and Hyperbaric Medicine*, 34:389–392.

- Harabin, A.L., S.S. Survanshi, and L.D. Homer. 1995. A Model for Predicting Central Nervous System Oxygen Toxicity from Hyperbaric Oxygen Exposures in Humans. *Toxicology and Applied Pharmacology*, 132:19–26.
- Haynes, P. 2016. Increasing the Probability of Surviving Loss of Consciousness Underwater when Using a Rebreather. *Diving and Hyperbaric Medicine*, 46:253–259.
- Palmquist, B. M., B. Philipson, and P. O. Barr. 1984. Nuclear Cataract and Myopia during Hyperbaric Oxygen Therapy. *British Journal of Ophthalmology*, 68:113–117.