Oxygen toxicity in recreational and technical diving

Andrew Fock and Ian Millar

Key words
Oxygen, toxicity, technical diving, mixed gas, review article

Abstract

(Fock A, Millar I. Oxygen toxicity in recreational and technical diving. *Diving and Hyperbaric Medicine*. 2008; 38: 86-90.)
It is increasingly common for recreational scuba divers to use breathing mixtures enriched with additional oxygen ('nitrox' or 'enriched air nitrogen') and for technical divers to be exposed to elevated partial pressures of oxygen for prolonged periods of time. The National Oceanic and Atmospheric Administration oxygen exposure limits have traditionally been used by the recreational diving industry and technical diving communities. Review of the original research into oxygen toxicity brings into question the validity of these limits and would suggest revised limits with a maximum partial pressure of oxygen of 162 kPa (1.6 Ata) and 142 kPa (1.4 Ata) at depth and the use of the repetitive air excursion (REPEX) limits for single and repetitive exposures. Suitable conservatism in case of the need for recompression therapy is recommended.

Introduction

The use of breathing mixtures containing high levels of oxygen ('nitrox', 'enriched air nitrogen' for scuba diving has become routine over the last decade. More recently, the advent of technical diving has seen the use of these mixtures as well as pure oxygen to accelerate decompression. Training agencies for both recreational and technical diving have traditionally used the central nervous system (CNS) limits prescribed by the National Oceanic and Atmospheric Administration (NOAA).[1] These describe a relationship between time and an exposure to a particular partial pressure of oxygen (PPO_2) and are provided for both single exposures and daily exposures. However, with the advent of technical diving, where decompression times may exceed five hours, many divers are routinely exceeding these limits apparently without ill effect. Therefore, it would seem timely to review the origins of these limits as well as newer data on oxygen toxicity more relevant to this style of diving.

Manifestations of oxygen toxicity

At a clinical level, the toxic effects of oxygen are most apparent in the lung, brain and eye. This should not be surprising, given the lung's direct exposure to oxygen, the very high blood flow and vaso-reactivity of the CNS and the unique avascular physico-chemical structure of the lens of the eye. In the lung, oxidative damage results in inflammation, capillary leakiness and ultimately fibrosis. The mechanisms of acute CNS toxicity are extremely complex and incompletely understood. In overview, it is thought that increased reactive oxygen species produced through the metabolism of molecular oxygen cause an imbalance between neurotransmitters, triggering uncoordinated electrical depolarisation (an excellent review is provided by Clark and Thom[2]). This often manifests as loss of consciousness with a grand mal-type convulsion and may commence abruptly, with or without preceding symptoms. The occurrence of such an event whilst diving is likely to be fatal, therefore a good understanding of the CNS oxygen tolerance limits is vital if high PPO_2 is to be

used. With respect to the eye, prolonged exposure to high PPO_2 has been observed to cause a reversible narrowing of the field of vision, whilst in the field of hyperbaric medicine, repeated daily exposure to hyperbaric oxygen for 20–30 sessions or more can induce myopia due to oxidative biochemical change in the lens.[3] Fortunately this myopia usually reverses spontaneously over several months after treatment is completed.

Historical background

Bert, in his seminal work on oxygen in 1878, clearly demonstrated that, whilst oxygen is essential to life, it is lethal at high pressure.[4] He exposed a number of species to high PPO_2 noting convulsions as a manifestation of CNS toxicity. This later became known as the Paul–Bert effect. In 1899, Lorrain Smith reported a series of experiments on rats exposed to raised PPO_2.[5] Smith detailed the pulmonary changes and noted that early changes were reversible, as well as the fact that higher pressures were associated with an earlier onset of symptoms. The pulmonary changes associated with high oxygen exposure are now commonly referred to as the Lorrain Smith effect.

By 1907 when JS Haldane was conducting experiments, which were to lead ultimately to the first successful diving tables, the works of Smith and Bert were well known.[6] In 1908, Haldane both recommends that air diving be limited to 50 fathoms (90 metres' sea water (msw)) to avoid oxygen toxicity and mentions the possibility of using oxygen to accelerate decompression.[6] In the case of the latter, it was felt at the time that the technical difficulties of using oxygen in decompression outweighed any possible benefit. Haldane also confirmed that duration and depth were related to the risk of CNS symptoms resulting from oxygen exposure.

World War Two experience

By the late 1930's the United States Navy (USN) was experimenting with oxygen for both deep diving and recompression therapy.[7,8] At this time it was generally believed that exposures to 100% oxygen at 304 kPa (3 Ata) were usually well tolerated, a limit that was similarly supported in the Royal Navy (RN). After the success of the Italian "human torpedoes" in damaging British battleships at Alexandria in 1941, the British sought to create a similar capability. The bubble-less and decompression-less features of closed-circuit oxygen rebreathers made them ideal for such covert missions. However, after a series of unexplained episodes of unconsciousness associated with the use of oxygen rebreathers in the early stages of World War Two, the RN embarked on an extensive series of human experiments to definitively determine the oxygen exposure limits for divers.[9]

These experiments, conducted by Kenneth Donald, involved more than 2,000 exposures and generally used convulsions as the end point for each experiment. In 60% of cases lip twitching was the first sign of CNS oxygen toxicity; however, in approximately 10% the first sign was a convulsion, often without any preceding symptoms (Table 1).

While CNS oxygen toxicity susceptibility did in general increase with pressure, there was such a large day-to-day variability in the time to convulse for an individual at any given pressure that it was difficult to meaningfully score the differences in susceptibility between individuals. In addition to this variability in individual susceptibility on a day-by-day basis there was also substantial inter-individual variability. However, there did appear to be a minimum threshold of 172 kPa (1.7 Ata) below which convulsions were not seen despite exposures of up to six hours.[10] Donald noted that subjects were less tolerant to oxygen if immersed as compared to in a dry chamber. Susceptibility to toxicity was also increased by exercise, if the water temperature was low or if carbon dioxide levels were elevated.

Donald also found that the addition of nitrogen to the breathing gas mixture increased divers' tolerance to increased partial pressures of oxygen. On the basis of Donald's work, the Royal Navy promulgated operational limits for its divers of 172 kPa for pure oxygen and to a PPO_2 of 203 kPa (2.0 Ata) for nitrox mixtures.[10]

Post World War Two

The USN also conducted a series of trials in the period immediately after World War Two. There was criticism of the RN studies based on the belief that the type of rebreather equipment used would have allowed accumulation of CO_2 and this would have reduced the RN divers' oxygen tolerance.

Table 1
First reported symptoms of CNS oxygen toxicity (modified from Donald K[9])

Symptoms	Number of cases	Percentage
Convulsions	46	9.2
Twitching lips	303	60.6
Vertigo	44	8.8
Nausea	43	8.6
Respiratory disturbance	19	3.8
Dyspnoea	8	
Cough	6	
Other	5	
Twitching, other than lips	16	3.2
Generalised jactitations	7	
Other	9	
Sensations of abnormality	16	3.2
Drowsiness	7	
Numbness	3	
Other	6	
Visual disturbances	5	1
Acoustic hallucinations	3	0.6
Paraesthesia	2	0.4

Extraordinarily, it was also suggested that there was some question as to the 'quality' of the RN dives and the divers conducting them.[11] Donald countered by pointing out that subsequent analysis of the equipment had not shown CO_2 accumulation, and that a substantial number of his subjects had gone on to win a Victoria Cross and other awards for bravery![10] The initial trials conducted by the USN produced higher oxygen tolerances but in hindsight it would appear that inadequate oxygen purging of their rebreather units may have allowed retention of nitrogen. This may explain the apparently higher oxygen tolerances reported.

Subsequent USN experimentation using open-circuit equipment resulted in progressive revisions to the USN oxygen limits. However, the data used to determine these limits were in general taken from very small numbers of trials and, in some cases, the exposures accepted (depth-time combination) in the tables seem at odds with the experimental data. In some cases, symptoms were observed at or before the time limits finally recommended.[11] As late as 1986, USN oxygen tolerance tables allowed up to 10 minutes at 253 kPa (2.5 Ata) and 240 minutes at 162 kPa. In contrast, the nitrox tables used by the USN were far more conservative, allowing only 30 minutes at 162 kPa PPO_2.

Recreational diving

Recreational oxygen exposure limits are generally based on the NOAA tables for oxygen exposure limits (Table 2).[1] These were reputedly derived from the USN nitrox tables but the actual experimental basis for them remains elusive. Hamilton has stated that these recommendations "*represent an operation decision, not research results*".[12] The NOAA recommendations limit the maximum oxygen exposure to 162 kPa for a maximum of 45 minutes at that pressure. They allow a maximum of 720 minutes at 61 kPa (0.6 Ata). As limits of tolerance, these recommendations seem at odds with the published CNS exposure data where definite symptoms are used as an endpoint. However, in recent large studies of Israeli military divers, softer or more subjective signs of possible oxygen toxicity were accepted as end points.[13] If these symptoms are accepted as endpoints of CNS oxygen toxicity, limits lower than those originally proposed by Donald or the USN may have some justification especially in the recreational setting.

For recreational diving the recommended maximum inspired PPO_2 at depth is usually limited to 143 kPa and for decompression to 162 kPa. Based on the available evidence, it would seem that acute CNS toxicity would be unlikely to occur in divers using these limits provided that there is strict adherence to prescribed gas composition and depth of use.

Table 2
NOAA oxygen exposure limits[11]
with permission (1 Ata = 101.3 kPa)

PPO_2	Single exposure	Daily limit
Ata	mins	mins
1.6	45	150
1.5	120	180
1.4	150	180
1.3	180	210
1.2	210	240
1.1	240	270
1.0	300	300
0.9	360	360
0.8	450	450
0.7	570	570
0.6	720	720

Pulmonary oxygen toxicity

In contrast to CNS oxygen toxicity, pulmonary oxygen toxicity has received little attention as a risk for recreational diving. However, modern trends in technical diving have seen dive times exceeding six hours with a considerable proportion of that time spent at partial pressures of oxygen in excess of 142 kPa to accelerate decompression. As a result, pulmonary effects of oxygen also now need to be considered in this setting. The symptoms of pulmonary oxygen toxicity are relatively consistent in contrast to acute CNS toxicity. When fully developed they resemble those of a viral upper respiratory tract illness with a dry hacking cough and retro-sternal chest discomfort. Both the pain and the coughing are markedly aggravated by deep inspiration. While initial changes are easily reversible (though individuals show marked variability in recovery time), severe toxicity may result in permanent lung damage. As with CNS oxygen toxicity, there appear to be considerable variations between individuals as to their pulmonary oxygen tolerance.

Pulmonary tolerance to high levels of oxygen has been shown by several researchers to be increased by the inclusion of low oxygen breathing periods.[10,11] Lambertsen et al demonstrated that if oxygen breathing was interrupted by five-minute periods of normoxia after each 20-minute high PPO_2 exposure this more than doubled the tolerable oxygen breathing time.[14]

Unit of pulmonary toxicity dose (UPTD)

As a medical therapy oxygen at partial pressures up to 51 kPa (0.5 Ata) is generally well tolerated for continuous periods of many days. Above this level oxygen toxicity results in a gradual reduction in vital capacity (VC) with increasing exposure. The oxygen exposure that risks a chosen reduction in VC is described by a hyperbolic relationship between PPO_2 and duration of exposure. Bardin and Lambertsen described this relationship mathematically.[15] They defined what they termed a 'unit of pulmonary toxicity dose' (UPTD) as the degree of pulmonary oxygen toxicity incurred from breathing 100% oxygen for one minute.

Mathematically, UPTD is defined as

$$UPTD = t((PPO_2 - 0.5)/0.5)^{0.83}$$

Where t is time in minutes, and PPO_2 is the partial pressure of oxygen.[15]

Using this methodology, their exposure data indicated that dives incurring 615 UPTD would be expected to sustain a 2% reduction in VC and dives incurring 1425 UPTD would be expected to sustain a 10% reduction in VC. The former is considered the limit for routine diving and the latter for therapy of life-threatening decompression illness.[10] These limits were based upon a small sample of subjects and there is significant variability in what actually occurs in any particular diver. Nevertheless, the UPTD tables provide a well-tried formula for limiting pulmonary risk.

Using the UPTD system to assess typical recreational rebreather diving reveals there should be minimal risk of developing pulmonary toxicity for dives of average duration. As an example, a closed-circuit rebreather (CCR) dive to a maximum depth of 67 msw for 30 minutes' bottom time requires 71 minutes at a PPO_2 of 131 kPa (1.3 Ata) and then 34 minutes at 162 kPa during decompression. This will incur 105 UPTD + 65 UPTD = 170 UPTD for the dive (CNS = 116% of NOAA limit). If two such dives were performed in a day, this would equate to 340 UPTD per day. However, should the diver require a therapeutic recompression using a Royal Navy Treatment Table 62, a further 645 UPTD would be incurred resulting in a total exposure of 985 UPDT, still within the acceptable limits. If 1425 UPTD is accepted as the daily maximum, then it would seen prudent to limit the daily diving exposure to somewhat less than 780 UPTD.

Maximal oxygen exposure will be associated with multi-day programmes of multiple, long-duration dives per day. The applicability of UPTDs to such scenarios is uncertain as no allowance is made for 'surface intervals' or cumulative exposure. In the late 1980s, Hamilton et al working with NOAA investigated the effects of prolonged exposure to raised PPO_2 in sea-floor habitats (generally shallow saturation diving with intermittent deeper excursions).[12] Due to the long exposures to elevated levels of oxygen, this work seems better constructed for application to recreational technical expeditions. Using the same calculation method as Bardin et al, UPTDs were renamed as oxygen tolerance units (OTUs) and were assumed to reflect total body oxygen toxicity. It was proposed that if exposures were kept below the described 'REPEX' (REPetitive air EXcursion) limits and a maximum PPO_2 of 152 kPa, then CNS toxicity issues would take care of themselves. OTUs are calculated on a cumulative basis, e.g., if planning an eight-day expedition, the REPEX table would allow a total of 2,800 OTUs over the eight-day period. This would allow 350 OTUs per day or 175 OTUs per dive if two dives per day were planned.

With respect to eye toxicity, it has been traditionally thought that there is little risk when diving within limits designed to avoid both CNS and pulmonary toxicity. Lambertsen et al demonstrated, visual field contraction, thought to be due to retinal vasospasm, after 2.5–3 hours of exposure to 304 kPa (3.0 Ata).[11] Likewise, the hyperbaric oxygen exposures associated with myopic change to the lens usually involve a PPO_2 ranging from 203 to 284 kPa (2.0–2.8 Ata) for 1.5 to 2 hours per session, daily for several weeks.[3]

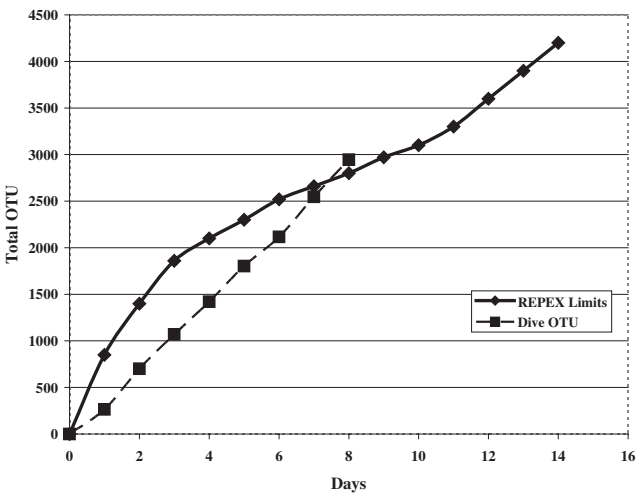
It would seem, therefore, that there should be little risk of pulmonary or eye toxicity with normal recreational technical diving. However, during a recent study of technical divers, the author noted symptoms suggestive of pulmonary oxygen toxicity in 50% of the divers while well below the REPEX exposure limits.[16] While these symptoms developed before the REPEX limits were approached, the cumulative OTUs in this group of recreational technical divers did approach the REPEX limits towards the end of the expedition indicating that cumulative oxygen exposures may become significant in this type of diving (Figure 2). In addition, a number of divers reported transient visual symptoms suggestive of a change in refraction. Such changes in refractive index in a recreational CCR diver have been reported previously.[17]

Summary

There would appear to be little supporting evidence of significant symptoms of CNS toxicity developing below a threshold of 162 kPa PPO_2 despite substantial exposures exceeding the NOAA limits. Exposures beyond PPO_2 of 172 kPa may result in convulsions at any time and without warning.

With the increasing popularity of technical diving using CCRs that maintain a relatively high PPO_2 throughout the

Figure 2
Allowable cumulative oxygen dose: REPEX limits and cumulative oxygen dose (OTU) for expedition divers (see text for explanation)



dive (commonly 131 kPa), the REPEX limits may be more relevant than the NOAA limits. Divers conducting such dives should be mindful of the potential for cumulative effects of multi-day diving on the lungs and the potential need for a therapeutic recompression when calculating daily exposure allowances and plan their dives accordingly.

For the diver conducting a single prolonged dive where pulmonary oxygen toxicity may become an issue, there may be a case for switching to a low PPO_2 for five-minute periods during the latter stages of long decompression profiles where high-oxygen mixes are used. Any such reduced oxygen periods would need to be taken into account in decompression calculations and gas mixtures should be selected with the aim of avoiding potential counter-diffusion problems where helium-based diluents are used.

There is a case for further studies that objectively assess changes in vision and lung function in divers undergoing repetitive, high-oxygen exposure, recreational diving.

References

1 Joiner JT, editor. *NOAA Diving Manual*. 4th ed. Flagstaff AZ: Best Publishing; 2001. p. 1-16.
2 Clark J, Thom S. Oxygen under pressure. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th ed. Edinburgh: Saunders; 2003. p. 358-408.
3 Evanger K, Haugen OH, Irgens A, Aanderud L, Thorsen E. Ocular refractive changes in patients receiving hyperbaric oxygen administered by oronasal mask or hood. *Acta Ophthalmol Scand*. 2004; 82: 449-53.
4 Bert P. *La pression barametrique: researches de physiologie experimentale*. 1878. Translated from the French by Hitchcock MA, Hitchcock FA. Published as: Barometric pressure researches in experimental physiology. In: *Key documents of the biomedical aspects of deep sea diving selected from the world's literature 1608–1982*. Durham, NC: Undersea and Hyperbaric Medical Society; 1983. CD-ROM. p. 2508-60.
5 Lorrain Smith J. The pathological effects due to increase of oxygen tension in the air breathed. *Journal of Physiology (London)*.1899; 24: 19-35.
6 Boycott D, Damant G, Haldane J. The prevention of compressed-air illness. *J Hyg*. 1908; (8): 342-443.
7 Behnke AR. USS Squalus: medical aspects of the rescue and salvage operations and the use of oxygen in deep sea diving. *US Navy Medical Bulletin*. 1939. In: *Key documents*, Undersea and Hyperbaric Medical Society; 1983. CD-ROM. p. 860-9.
8 Behnke AR, Shaw LA, Messer AC, Thomson RA, Motley EP. The circulatory and respiratory disturbances of acute compressed air illness and the administration of oxygen as a therapeutic measure. *Am J Physiol*. 1936; 114: 525-33.
9 Donald KW. Oxygen poisoning in man. *BMJ*. 1947; (May 17th): 667-717.
10 Donald K. *Oxygen and the diver*. Hanley Swan, Worcs: The SPA Ltd; 1992.
11 Lambertsen CJ, Clark JM, Gelfand R, Pisarello JB, Cobbs WH. Definition of tolerance to continuous hyperoxia in man: An abstract report of predictive studies V. In: Bove AA, Bachrach AJ, Greenbaum LJ, editors. *Underwater and hyperbaric physiology IX*. Bethesda, Ma: Undersea and Hyperbaric Medical Society; 1987. p. 717-49.
12 Hamilton RW, Kenyon DJ, Peterson RE, Butler GJ, Beers DM. *REPEX: development of repetitive excursions, surfacing techniques, and oxygen procedures for habitat diving*. NURP Technical Report 88-1A. Rockville, MD: NOAA, US Department of Commerce; 1988.
13 Arieli R, Shochat T, Adir Y. CNS toxicity in closed-circuit oxygen diving: symptoms reported from 2,527 dives. *Aviat Space Environ Med*. 2006; 77: 526-32.
14 Lambertsen CJ. Extension of oxygen tolerance in man: philosophy and significance. *Exp Lung Res*. 1988; 14(Suppl): 1035-58.
15 Bardin H, Lambertsen CJ. *A quantitative method for calculating pulmonary oxygen toxicity. Use of the "Unit of Pulmonary Toxicity Dose" (UPTD)*. Environmental Medicine Report. Philadelphia: University of Pennsylvania; 1970.
16 Fock AW. Health status and diving practices of a technical diving expedition. *Diving and Hyperbaric Medicine*. 2007; 36: 179-85.
17 Butler FK Jr, White E, Twa M. Hyperoxic myopia in a closed-circuit mixed-gas scuba diver. *Undersea Hyperb Med*. 199; 26: 41-5.

Dr Andrew Fock, FANZCA, Dip DHM, is a Senior Staff Specialist, and
Ian Millar, FAFOM, Dip DHM, is Medical Director in the Hyperbaric Services, The Alfred Hospital, Melbourne

Address for correspondence:
Dr Andrew Fock
The Alfred Hospital, Commercial Road, Prahan
Melbourne, Australia 3004
Phone: *+61-(0)3-9276-2269*
Fax: *+61-(0)3-9076-3052*
E-mail: *<a.fock@alfred.org.au>*

This review article is based on a presentation by Dr Fock at the SPUMS ASM, New Zealand, 2007.