

Oxygen exposure and toxicity in recreational technical divers

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Abstract

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Introduction: Central nervous system oxygen toxicity is a recognised risk in recreational open-circuit scuba diving with the use of nitrox (oxygen-enriched air mixtures), but other forms of oxygen toxicity in other diving settings are poorly understood. However, divers using constant partial pressure of oxygen closed-circuit rebreathers (CCRs) for multi-day, multi-dive expeditions could potentially experience cumulative oxygen exposures above the current recommended limits.

Methods: We followed a number of technical recreational diving expeditions using CCRs and recorded the cumulative oxygen exposures of the individual divers. Lung function and visual acuity were recorded at intervals during the expeditions.

Results: Over several 8- to 12-day expeditions, divers either approached or exceeded the recommended maximum repetition excursion oxygen exposure (REPEX) limits. Lung function did not show any significant decrement. Changes in visual acuity changes were reported in several divers but were difficult to quantify. Formal testing of one diver's visual acuity on return home demonstrated a myopic change that resolved over the subsequent eight weeks.

Conclusions: Recreational CCR divers conducting multi-dive expeditions of eight days or more may approach or exceed the REPEX oxygen limits. Despite this, there does not appear to be any significant decrement in lung function. Hyperoxic myopia occurs in some individuals. Changes in acuity appear to resolve spontaneously post exposure. Despite the lack of significant changes in respiratory function, divers should be cautious of such exposures as, should they require recompression therapy for decompression illness, this may result in significant pulmonary oxygen toxicity.

Key words

Technical diving, oxygen, toxicity, pulmonary function, vision, diving research

Introduction

Since the introduction of nitrox (enriched-air nitrogen or EAN), in the early 1990s, the use of oxygen-rich breathing mixtures in recreational diving has become increasingly common.¹ While recreational divers will commonly use nitrox mixtures of 28–36% oxygen (O₂), technical divers use mixtures of up to 100% O₂ in order to accelerate decompression after deep mixed-gas dives. With the introduction of commercially available closed-circuit rebreathers (CCRs) in the late 1990s, technical recreational divers were able to optimize their decompression times further by the use of a constant partial pressure of O₂ (PO₂, usually 1.3–1.4 atm (131–141 kPa))* during technical dives. However, the use of such equipment for deep mixed-gas diving with prolonged decompression times has the potential to result in the diver exceeding the National Oceanographic and Atmospheric Administration's (NOAA) recommended limits of central nervous system (CNS) O₂ exposure.² Where more than one dive per day is conducted over the course of a multi-day expedition, divers may also exceed the NOAA daily limits for pulmonary O₂ toxicity limits or the recommended repetitive excursion (REPEX) exposure limits over the course of the expedition.^{2,3} During a previous observational study of a group of technical rebreather divers,

the authors noticed symptoms suggestive of both pulmonary and ocular O₂ toxicity.⁴

Aims

This study aimed to quantify the O₂ exposure of recreational technical divers using CCR scuba during multi-day expeditions where more than one dive per day was conducted. It also aimed to measure the extent of pulmonary and ocular toxicity by assessing the changes in respiratory spirometry and ocular refraction during such expeditions.

Methods

Ethics approval was given by the Alfred Hospital Ethics Committee (project # 41/07) and the study was conducted in accordance with the Helsinki Declaration. Formal written consent was obtained and divers were given information sheets outlining the nature and aims of the study.

Twenty-nine divers participating in mixed-gas, multi-day, multi-dive diving expeditions were recruited between 2007 and 2010. In some cases, individual divers participated in the study during several of the expeditions over several years. All divers, with the exception of one, used CCRs that maintained

* **Footnote:** The units prevalent in the technical diving community are used for pressure measurements in this article. To use kPa would render the paper largely unintelligible to at least part of its intended readership (the divers themselves) and it would also fail to prepare physicians for the language they will hear technical divers using. The agreed format is: atm for partial pressures or gauge pressures (e.g., the typical PO₂ setpoint of a rebreather is 1.3 atm) and atm abs for ambient pressures at depth.

a constant PO₂ between 1.3 and 1.4 atm during the dives. One diver using open-circuit scuba was recruited to gain insight into typical differences in O₂ exposures between open-circuit (OC) and CCR divers on the same expedition. In some cases decompression was accelerated either by increasing the PO₂ in the CCR manually to 1.5–1.6 atm during the final decompression stop or by using surface-supplied (SS) O₂ at the 6 metres' sea water (msw) decompression stop.

Inclusion criteria included:

- participation in multi-day, multi-dive diving on a suitable dive platform using CCRs such that significant O₂ exposure was likely;
- no known pre-existing lung or ocular disease;
- willingness to participate in the study.

The CCRs maintained a setpoint PO₂ of 1.3–1.4 atm for the majority of the dive. The final decompression was conducted between 1.3 and 1.6 atm on either the CCR or SS O₂ at 6 msw. The average depth of the dives was 69 msw. Divers breathed trimix (helium, oxygen and nitrogen) diluent during all dives.

All dives were logged for depth, duration at setpoint and PO₂ during decompression. O₂ exposures were calculated using the method previously described by others and daily cumulative O₂ exposure stored and plotted using Numbers™ spreadsheet software (Apple® 2009).^{3,5}

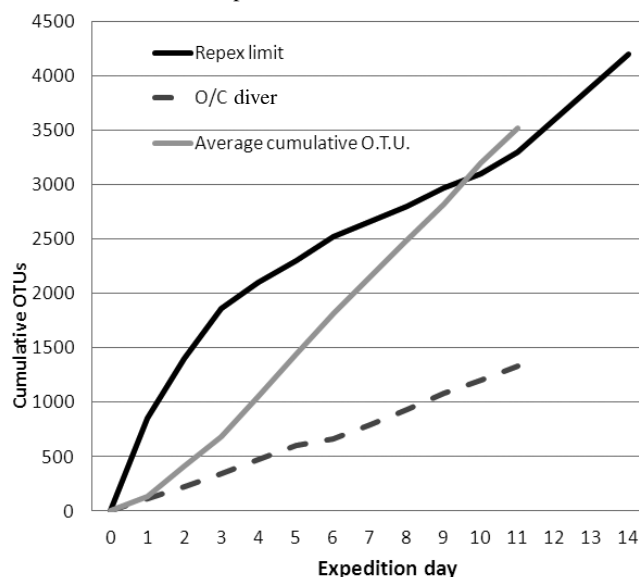
Respiratory function was monitored using an Easy One™ spirometer (Niche Medical®) and stored in the spirometer's proprietary database. This device uses an ultrasonic method of flow detection and does not require calibration either before use or in the field.⁶ Tests were conducted pre-expedition and at regular intervals (usually every third day) during the expedition as well as on the day post expedition. Tests conducted during the expeditions were done below deck at the same time each day (usually 3–4 hours after the last dive) to minimize variability. Parameters recorded were limited to those achievable by the device.⁶

Refractive changes were measured using a full-size Snellen chart at 6 metres' distance and a set of corrective lenses. Assessments were conducted on each eye at the same time of day (on deck) to minimize any possible refractive variations secondary to different lighting. Divers were asked to select the lens which produced the sharpest image of the 6/6 line on the Snellen chart. As with the respiratory tests, refractive measurements were carried out pre- and post-expedition as well as at regular intervals during the expedition.

Divers were questioned as to potential respiratory, ocular and decompression sickness (DCS) symptoms at the time that the respiratory tests were conducted. Divers acted as their own controls, allowing the use of the Student paired t-test. Statistical analysis was performed using the integrated statistical package in the Apple® software Numbers™ spreadsheet. Statistical significance was taken as $P < 0.05$.

Figure 1

Average cumulative oxygen exposures during all diving expeditions in the series



Results

SUBJECTS AND DIVE PROCEDURES

Eight divers who conducted only one dive per day on the expedition in which they were participating, as well as the chief researcher, who was forced to leave the expedition for several days for unrelated reasons, were excluded. After exclusions, 14 male CCR divers (mean age 46-years, range 34–64; BMI 27 kg m⁻², range 23–30) providing 20 data sets over three multi-day diving expeditions and one OC diver were studied. Dives of mean duration 112 minutes (SD 32; range 10–240 min) were conducted twice per day with a surface interval of approximately 4 hours between dives.

PULMONARY OXYGEN EXPOSURE

Cumulative O₂ exposures approached or exceeded the REPEX recommended O₂ limits in most CCR divers by day 8 of the diving expeditions (Figure 1). Where divers elected to take a dive or a day off, this reduced the likelihood of exceeding the limits during the expedition.

The O₂ exposure of the OC scuba diver was substantially different to the CCR divers, despite the OC diver using EAN 50% and 100% O₂ for decompression. Overall, the OC diver conducted shorter dives and had substantially reduced bottom times compared to the CCR divers (OC diver average dive time 44 (SD 14) min versus CCR divers' average dive time 112 (SD 32) min), being constrained by bottom gas duration at the depths experienced (average depth 69 msw).

No significant decrements in forced expiratory volume in 1 sec (FEV₁) and force vital capacity (FVC) were seen, despite almost all the divers approaching or exceeding the REPEX

Table 1

Mean % changes in forced vital capacity (FVC); forced expiratory volume in 1 sec (FEV₁); peak expiratory flow rate (PEFR) and forced expiratory flows at various lung volumes (FEF₂₅, FEF₇₅, FEF₂₅₋₇₅)

	Mean % change	SD	P value
FVC	-0.4%	4.5%	0.42
FEV ₁	-3.0%	5.0%	0.32
PEFR	0.2%	6.8%	0.18
FEF ₂₅	1.5%	10.3%	0.43
FEF ₇₅	-9.0%	20.4%	0.18
FEF ₂₅₋₇₅	-13.0%	13.7%	0.07

O₂ exposure limits (Table 1). Forced expiratory flow 25–75% (FEF_{25-75%}) showed a decrement of 13% which did not reach statistical significance (*P* = 0.07).

Nearly half the divers in the earlier expeditions complained of retrosternal discomfort associated with prolonged O₂ exposure. During the later expeditions in the series where the use of SS O₂ for decompression was less common, these symptoms almost completely disappeared despite total dive times being longer on average. Two-thirds of the divers on the first expedition also complained of a non-productive cough post dive. This symptom was noticeably less common (only one of eight divers) in the last expedition, where divers tended to remain on their CCR, which provided a warm, humidified breathing mixture during decompression.

REFRACTIVE CHANGES

The 14 CCR divers reported subjective difficulty with distance acuity by the end of the expeditions in 18 of the 20 data sets measured. Mean changes in visual acuity are shown in Table 2. Measured refractive changes during the expeditions varied by up to a dioptre from day to day. Measured changes at the end of the expeditions failed to accurately demonstrate the symptomatic decrement in distance acuity described by the divers.

One diver sought formal evaluation post expedition with an ophthalmologist. This demonstrated a 0.75:1.0 dioptre (S:D) myopic change which had resolved to 0.0:0.0 (S:D) eight weeks later. Intraocular pressure was measured as normal at both examinations. Another diver required the use of -1.5 D corrective lenses for six weeks post expedition for driving and distance vision, but did not seek formal evaluation. The oldest diver in the group did not report or experience any myopic refractive changes.

CNS O₂ EXPOSURE

While formal calculation of CNS O₂ exposure was not performed, based on the average dive time per day on 1.3 atm O₂ and the use of increased PO₂ during decompression, the NOAA CNS limits (Table 3) were routinely exceeded.

Table 2

Mean % changes in visual acuity over time of diving expedition (*n* = 20)

Expedition day	Mean change in acuity (dioptres)
1	0.0
6	0.4
9	0.2
12	0.3
13	0.4

Table 3

NOAA CNS allowed oxygen exposure times

Exposure time (min)	PO ₂ (atm)	Daily limit (min)
45	1.6	150
120	1.5	180
150	1.4	180
180	1.3	210
210	1.2	240
240	1.1	270
300	1.0	300
360	0.9	360

No diver during this series complained of or experienced any symptoms suggestive of CNS toxicity while in-water; however, a number of divers did mention that they had begun to experience very vivid dreams at night towards the latter stages of the various expeditions.

Discussion

The history of our understanding of both CNS and pulmonary O₂ toxicity has been described previously.⁷ Studies conducted during the 1940s using Royal Navy volunteers demonstrated that there was considerable inter- and intra-individual variability to O₂ toxicity (see historical article in this issue, p. 105-8).⁸ Whilst, in general, increased pressure reduced the time for the first development of CNS symptoms, convulsions were often the first sign of O₂ toxicity and could occur at any time when the PO₂ exceeded 1.7 atm. In contrast, despite research-based evidence, the US Navy developed a set of recommended O₂ exposures which exceeded this threshold, albeit for short periods of time.⁹ As seen in this study, technical divers using CCRs routinely exceed the NOAA limits for CNS O₂ exposures below 1.6 atm, apparently without issue. This would appear consistent with the earlier Royal Navy studies.

In contrast to the CNS O₂ exposure, until recently it has been uncommon for recreational technical divers to receive enough O₂ during a dive to enter into the realm of potential pulmonary O₂ toxicity. This was due largely to the logistical difficulties of carrying enough open-circuit gas to accumulate sufficient dive time to result in toxic exposure, as evidenced in the OC diver in this study. As

with CNS exposure, pulmonary O₂ toxicity is related to time and pressure. Exposure curves with expected reductions in FVC or FEV₁ are the generally quoted end points, and mathematical descriptions of exposure are commonly used describing the exposure in terms of O₂ toxicity units (OTUs) or units of pulmonary toxicity dose.^{3,5} Once again, considerable inter-individual variability has been observed and most research has focused on single O₂ exposures.⁵

While it has been long understood that low O₂ or air breaks of 5–10 minutes duration delay the onset of pulmonary O₂ toxicity symptoms, the variability in recovery of the lungs once normoxia has been reestablished has meant that an accurate mathematical description of recovery has largely eluded researchers.^{10–12} Instead, the cumulative daily pulmonary O₂ exposure during habitat diving was utilized with an elevated PO₂ by adding the OTUs accrued during excursions to depth and applying an empirical algorithm to determine a daily oxygen exposure limit.³ These became the REPEX limits which are commonly used by technical divers conducting multi-day dives, despite the fact that they were derived in a rather different diving context (excursion dives from saturation).³ Nevertheless, the REPEX limits probably represent the best data currently available to technical divers in order to avoid both pulmonary and whole-body O₂ toxicity, given the scarcity of other data.

Perhaps the best studies available on pulmonary O₂ exposure with constant PO₂ diving have been those conducted by the US Navy.^{13–15} While the total dive times were similar in their divers to those in our group, the exposure was in a single, four-hour dive with a 20–44 hour surface interval, depending on the study. Divers breathed humidified SS O₂ at the bottom of a pool. They were allowed to surface and breath room air (to eat and drink) for no more than 5 minutes per hour. Divers were assessed for pulmonary function including the diffusion capacity of the lung to carbon monoxide (DL_{CO}), as well as ocular refractive and pressure changes. The divers in these studies showed substantial variability in the effects of these exposures, with some individuals showing decrements in both pulmonary and refractive function during the study, which resolved later.

This is consistent with our results, where we also measured changes that seemed to return to baseline or near baseline by the end of our study. In the US Navy studies, the DL_{CO} decreased by 0.6% per day on average while other parameters seemed to show a variable response, with some individuals oscillating about a mean and others showing decrements or occasionally actually improving. Several of the divers also showed significant decrements in their FEF_{25–75}. Unexpectedly, in general, the divers' reported symptoms did not correspond temporally with measured pulmonary changes. The overall conclusions were that this type of exposure caused little in the way of clinically significant changes to pulmonary or ocular refractive function. However, it should be noted that some individual

divers did show significant changes. While we attributed our observed variability in refractive and pulmonary function to the limitations of our equipment (necessitated by the requirement to be portable while operating off-shore) the variability of results also seen in a comprehensive respiratory laboratory suggests that our observed variability is, in fact, not artifact. Indeed, this observed variability and the wide normal range of some of the respiratory parameters (e.g., FEF_{25–75%}) brings into question the value to clinicians of the use of respiratory tests (or indeed symptoms) as a predictive measure for the onset of pulmonary O₂ toxicity.

The fact that one of our divers showed significant refractive changes when formally measured post expedition compared to the final measurement during the expedition does bring into question the sensitivity of refractive testing performed during our study. Regardless of this, significant shifts in refraction can occur in divers exposed to this level of O₂ over several days but may vary on a daily basis. A similar myopic refractive change has been reported in a technical diver.¹⁶ In this case, the diver also had a markedly reduced intra-ocular pressure (IOP). In the diver in our series, the IOP was within the normal range. In both cases, the divers' vision returned to baseline over several weeks.

A progressive 0.25D per week myopic refractive change has been reported in patients undergoing hyperbaric oxygen therapy (HBOT).¹⁷ This change is unrelated to changes in axial length or corneal curvature, implying that it is due to lenticular refractive change alone.¹⁸ The changes observed in our study (with only one week of diving at an average of 1.3 atm, versus most HBOT exposures) would seem rather larger and faster than those described in chamber exposures and may reflect the twice-daily exposure as well as the reduced recovery time between dives with our group. It would be interesting to more formally assess such a group pre- and post-dive expedition to better quantify the changes described. Certainly, the myopic changes observed were enough to cause the divers difficulty in seeing clearly the signs in the airport on the way home and may also be a problem with distance vision for driving. As a side benefit, most divers commented that they found it much easier to read their dive computers as the expeditions progressed!

The original impetus for this study was symptoms suggestive of pulmonary O₂ toxicity during a previous study.⁴ At that time, dives were conducted on the diver's CCR and the final decompression stop used SS O₂ but, by the time of the last expedition in 2010, almost none of the divers used SS O₂, except for short periods of time, and none of the divers complained of the retrosternal burning or other symptoms which had been common previously. It is possible that the original observations may well have been due to the dry nature of the SS gas rather than the O₂ *per se*, though in the US Navy study, up to 16% of divers reported symptoms despite their SS O₂ being humidified.¹⁴

Several divers spontaneously reported experiencing vivid dreams during the latter stages of the last expedition. On close questioning, they stated that this was also common during the latter stages of previous expeditions. It is interesting to speculate whether this might be an effect of the raised levels of cerebral oxygen and an effect on neurotransmitter levels.

During more than 850 dives to an average depth of 64 msw using trimix, there were only four cases of “niggles” (minor type I DCS symptoms, which were self-treated by the divers by in-water recompression on 100% O₂), all during the early expeditions. Given the already substantial pulmonary oxygen exposures, had any of these divers required a more formal oxygen recompression treatment, it may well have resulted in significant pulmonary oxygen toxicity.

Conclusions

Despite substantial O₂ exposures during multi-day, multi-dive expeditions using CCRs, significant measurable changes in pulmonary function were not observed. Despite this lack of observed change in pulmonary function, divers should be aware of the potential for pulmonary toxicity and should keep track of their exposure. Some divers developed myopic refractive changes that took up to eight weeks post expedition to completely resolve. In individuals who are known to experience refractive changes associated with this type of exposure, it may be prudent to carry a pair of -1.0D corrective lenses for the flight home.

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Conflict of interest: nil

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