CASE REPORT

Hyperoxic myopia in a closed-circuit mixed-gas scuba diver

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Butler FK Jr, White E, Twa M. Hyperbaric myopia in a closed-circuit mixed-gas scuba diver. Undersea Hyper Med 1999; 26(1):41–45.—A myopic shift occurred in a closed-circuit mixed-gas scuba diver using a 1.3 atm abs constant partial pressure of oxygen in a nitrogen–oxygen mix. This change was noticed after approximately 18 days of diving with a mean dive time of 4.04 h each day. The observed myopic shift was due to hyperoxic myopia, one sign of lenticular oxygen toxicity, and resolved over a 1 mo. period after diving was completed. On a subsequent dive trip, a myopic shift was found in both the index diver as well as two other divers breathing the same gasmix on similar profiles. Diving communities should be aware of the risk of both lenticular and pulmonary oxygen toxicity when conducting intensive diving at oxygen partial pressures in the 1.3–1.6 atm abs range.

oxygen toxicity, lenticular oxygen toxicity, hyperbaric oxygen, closed-circuit scuba, scuba diving, hyperoxic myopia, eye, lens

Repetitive oxygen exposures during hyperbaric oxygen (HBO2) therapy have been reported to cause myopic refractive shifts. This phenomenon has not to the authors' knowledge been previously reported in scuba divers.

CASE REPORT

A 48-yr-old white male, closed-circuit mixed-gas scuba diver called the Diver's Alert Network (DAN) because of a progressive decrease in distant visual acuity which he noted on a diving trip. He had no history of previous eye problems, was in good general health, and was taking no medications except for antioxidant vitamins and an occasional Rolaid tablet for gastroesophageal reflux disease. He was diving with a Biomarine Mark 155 closed-circuit mixed-gas underwater breathing apparatus (UBA) adjusted to provide a constant partial pressure of 1.3 atm abs oxygen in a nitrogen–oxygen mix. During the course of an underwater film-making project, he was diving at an average depth of 21.3 m (70 ft) with a mean daily dive time of 4.04 h. There were typically three dives performed per day, with approximately half of those requiring short decompression stops. Diving began on 23 January 1998 and was completed on 13 February 1998. He performed 21 days of mixed-gas diving with a total of 84.8 h accumulated on the Mark 155 at 1.3 atm abs O2. There were four additional dives on which open-circuit air was used. After approximately 18 days of diving, he began to notice blurred vision at distance with intact near visual acuity. This decrease in distance acuity became progressively worse over the last 3 days of diving and continued to worsen for 2 days after diving was completed. The diver experienced no signs or symptoms of decompression sickness during the course of the dive trip.

The diver had been examined by his optometrist on 20 October 1997. At that time, he had 20/20 distance vision in both eyes with the following refraction:

OD: -0.50 – 1.00 × 168
OS: Plano –2.50 × 175

He was examined again by his optometrist on 18 February 1998 after returning to the United States. He was once more found to have 20/20 distance acuity OU but now required the myopic refraction noted below to achieve this acuity:

OD: -2.00 – 1.00 × 170
OS: -1.50 – 2.50 × 175

His examination was otherwise unremarkable except for a marked decrease in intraocular pressure from 14 mmHg OU on 20 October 1997 to pressures of 6 mmHg OD and 8 mmHg OS on 18 February 1998. Gonioscopy revealed open grade IV angles OU. Pupils were equal and reactive to light and accommodation. Dicon autoperimetry found no visual field defects in either eye. His funduscopic exam revealed a cup to disk ratio of 0.1 and sharp disk margins in both eyes with normal macula and peripheral retina OU.

Because of this individual's recent history of diving, consultation with the Diver's Alert Network (DAN) was
obtained. After a review of the above history and ocular findings, the diver was advised that his presentation was most consistent with a diagnosis of hyperoxic myopia and that his refraction should return to normal over a period of several weeks to several months. A fasting blood glucose was obtained to rule out diabetes mellitus, and was normal.

The diver’s myopia slowly decreased over the ensuing month. By 6 April 1998, his distance visual acuity remained at 20/20 OU and his refraction was as follows:

**OD:** Plano −1.00 × 170  
**OS:** +0.25 −2.50 × 175

A chronological summary of the refractive data and intraocular pressure measurements from his eye exams between 20 October 1997 and 6 April 1998 is provided in Table 1.

**DISCUSSION**

The differential diagnosis of an acute myopic shift includes osmotic changes in the lens of the eye caused by the hyperosmolar state found in untreated diabetes mellitus, systemic medications (especially diuretics), miotic eye medications, ciliary spasm, and hyperoxic myopia.

Oxygen, at a high enough partial pressure, can result in initial functional impairment and the ultimate chemical destruction of any living cell (1,2). The damage caused by HBO₂ is caused primarily not by the direct actions of the superoxide radical and hydrogen peroxide, but by the secondary generation of more reactive intermediates such as the hydroxide radical and the products of lipid peroxidation (1). The two most commonly encountered types of oxygen toxicity associated with hyperbaric exposures are central nervous system oxygen toxicity and pulmonary oxygen toxicity, but as early as 1935, Behnke et al. (3) reported a reversible decrease in peripheral vision after O₂ breathing at 3.0 atm abs (3). Lambertsod and Clark and their colleagues (1,2,4) also observed a progressive decrease in peripheral vision associated with hyperoxic exposures that probably represents a form of retinal O₂ toxicity. A decrease in peripheral vision was noted after approximately 2.5 h of O₂ breathing at 3.0 atm abs in a dry chamber. This decrease was progressive until O₂ breathing was stopped at 3.5 h. The average decrement in visual field area was 50%. Recovery was complete in all subjects after 45 min of air breathing (1,4). A decrease in ERG amplitude was noted as well, but did not correlate directly with the size of the visual field defect and returned to normal more slowly after the termination of the hyperoxic exposure (1,4). Visual acuity and visual cortical evoked responses remained normal in all subjects.

**Myopic changes in hyperbaric oxygen therapy:** As noted in the introduction, progressive myopic changes as a result of hyperoxic exposures have previously been reported in patients undergoing HBO₂ therapy (1,5–9). The rate of myopic change has been reported to be approximately 0.25 diopters per week and this change was progressive.

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**Table 1: Chronology of Ocular Findings**

<table>
<thead>
<tr>
<th>Date</th>
<th>Eye</th>
<th>Refraction</th>
<th>Visual Acuity</th>
<th>Keratometry</th>
<th>Axial Length</th>
<th>IOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 October</td>
<td>OD</td>
<td>−0.50 − 1.00 × 168</td>
<td>20/20</td>
<td>43.75/45.12 × 090</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>plano −2.50 × 174</td>
<td>20/20</td>
<td>43.25/45.75 × 087</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>18 February</td>
<td>OD</td>
<td>−2.00 − 1.00 × 170</td>
<td>20/20</td>
<td>43.87/45.50 × 082</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>−1.50 − 2.50 × 175</td>
<td>20/20</td>
<td>43.25/46.25 × 088</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>19 February</td>
<td>OD</td>
<td>−2.00 − 1.25 × 173</td>
<td>20/20</td>
<td>44.00/45.75 × 085</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>−1.50 − 2.75 × 172</td>
<td>20/20</td>
<td>44.00/45.75 × 085</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>20 February</td>
<td>OD</td>
<td>−1.75 − 1.25 × 175</td>
<td>20/20</td>
<td>43.75/45.25 × 086</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>−1.50 − 2.25 × 175</td>
<td>20/20</td>
<td>43.25/45.87 × 086</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>26 February</td>
<td>OD</td>
<td>−1.00 − 1.25 × 175</td>
<td>20/20</td>
<td>43.87/45.50 × 083</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>−0.75 − 2.50 × 175</td>
<td>20/20</td>
<td>42.12/46.37 × 086</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>3 March</td>
<td>OD</td>
<td>−1.00 − 1.25 × 175</td>
<td>20/20</td>
<td>43.87/45.37 × 087</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>−0.75 − 2.50 × 175</td>
<td>20/20</td>
<td>43.87/45.75 × 087</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>11 March</td>
<td>OD</td>
<td>−0.50 − 1.00 × 168</td>
<td>20/20</td>
<td>43.87/45.37 × 087</td>
<td>14</td>
<td></td>
</tr>
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<td>plano −2.50 × 174</td>
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<td>14</td>
<td></td>
</tr>
<tr>
<td>24 March</td>
<td>OD</td>
<td>plano −2.00 × 170</td>
<td>20/20</td>
<td>43.00/45.25 × 082</td>
<td>13</td>
<td></td>
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<tr>
<td></td>
<td>OS</td>
<td>+0.25 − 2.50 × 175</td>
<td>20/20</td>
<td>43.00/45.75 × 091</td>
<td>10</td>
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<tr>
<td>26 March</td>
<td>OD</td>
<td>Plano −1.00 × 170</td>
<td>20/20</td>
<td>43.75/45.37 × 087</td>
<td>14</td>
<td></td>
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<tr>
<td></td>
<td>OS</td>
<td>+0.25 − 2.50 × 175</td>
<td>20/20</td>
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<td>10</td>
<td></td>
</tr>
<tr>
<td>6 April</td>
<td>OD</td>
<td>Plano −1.00 × 170</td>
<td>20/20</td>
<td>43.75/45.37 × 087</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OS</td>
<td>+0.25 − 2.50 × 175</td>
<td>20/20</td>
<td>43.12/45.75 × 085</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>
HYPEROXIC MYOPIA IN SCUBA DIVER

throughout the course of HBO₂ therapy (5). Hyperoxic myopia is generally attributed to an increase in the refractive power of the lens, since Anderson and Shelton (6) have shown that axial length and keratometry readings did not reveal a corneal or axial length basis for the myopic shift. The ocular findings in this diver were consistent with this presumed etiology. Reversal of the myopic shift after discontinuation of the HBO₂ therapy usually occurs within 3–6 wk, but may take as long as 6–12 mo. (8). The PO₂ in these exposures typically varies from 2.0 to 3.0 atm abs depending on the treatment protocol used.

Cataract formation has also been reported by Palmquist and co-authors (7) in patients undergoing a prolonged course of daily HBO₂ therapy at 2.0–2.5 atm abs. Seven of 15 patients with clear lenses at the start of therapy developed cataracts during their course of treatment. Fourteen of these 15 patients received a total HBO₂ treatment time of between 300 and 850 h. The lens opacities noted were not completely reversible after HBO₂ therapy was discontinued. Hyperoxic myopia and subsequent cataract formation may therefore be considered to represent two levels of severity of lenticular O₂ toxicity.

Similarities to age-related cataracts: The manifestations of lenticular O₂ toxicity are similar to the ocular findings encountered in the development of age-related nuclear sclerotic cataracts except for a much-accelerated time course. The natural progression of age-related nuclear sclerotic cataracts is a gradual increase in the density of the lens, which initially results in a myopic shift, but eventually reduces the transparency of the lens and the patient's vision. The parallels between age-related nuclear sclerotic cataracts and lenticular O₂ toxicity are intriguing in light of a recent longitudinal study which found that the incidence of nuclear sclerotic cataracts was decreased by one-third in individuals taking multivitamin supplements and by one-half in those taking vitamin E supplements (10). It is interesting to note that the refractive changes observed in this diver occurred despite his taking both a multivitamin supplement and an additional antioxidant vitamin supplement. This suggests that either there is not a protective effect from antioxidant supplements on lenticular oxygen toxicity or that the doses used were not sufficient to provide a protective effect under the hyperoxic conditions described.

Relevance to technical and military divers: This is the first report known to the authors of hyperoxic myopia resulting from closed-circuit scuba diving. A MEDLINE search and a search of the Undersea and Hyperbaric Medical Society database revealed no previous reports of this phenomenon in scuba divers. Although the PO₂ experienced by this diver was lower than that described in the reports of hyperoxic myopia caused by HBO₂ therapy, immersion and exercise are well known to potentiate the toxic effects of O₂ on the central nervous system (11–15). It is possible that these factors may cause a similar potentiation in ocular tissues. No other etiology for the diver’s myopic shift was found, and the steady return to his pre-exposure refraction after diving was completed strongly suggests hyperoxic myopia.

This observation is an important one for the technical diving and military diving communities as they consider what PO₂ to use in their closed-circuit, mixed-gas UBAs. Cave divers, salvage divers, Naval Special Warfare divers conducting SEAL Delivery Vehicle (SDV) operations, and other specialized types of divers may dive frequently and long enough to make hyperoxic myopia a possibility at a PO₂ of 1.3 atm abs. Most recreational divers do not perform diving of the sustained intensity conducted by this diver and would be unlikely to experience hyperoxic myopia during the course of their diving, although this possibility cannot be completely dismissed. Associated decrease in intraocular pressure: Also of interest is the observed transient decrease in intraocular pressure. A decrease in intraocular pressure from 14 to 6 mmHg is seldom seen clinically in the absence of ocular trauma, ocular inflammation, or intraocular pressure-lowering medications. The refractive changes in the tissues of the lens would not be expected to result in a decrease in intraocular pressure. Intraocular pressure is determined by the balance between aqueous formation by the ciliary processes and the outflow facility of the eye through the trabecular meshwork and the uveoscleral channels. The pressure decreases noted may represent a toxic effect of O₂ on the ciliary processes, resulting in decreased aqueous formation or, alternatively, may represent an increase in outflow facility. Nichols and Lambertsen (2) describe a number of ocular changes in humans and animals resulting from hyperoxia, including retrolental fibroplasia, retinal vasoconstriction, visual field constriction, retinal detachment, retinal microinfarcts, changes in dark adaptation, photoreceptor damage, and a decrease in amplitude of the electroretinogram. These authors did not report any observations of a decrease in intraocular pressure. A more recent review article on diving and hyperbaric ophthalmology also made no mention of reports of decreases in intraocular pressure resulting from hyperoxic exposures (9).

Recurrent of myopic changes on subsequent dive trips: The diver sought advice concerning a possible recurrence of his hyperoxic myopia during a subsequent dive trip which was planned for the near future and which was anticipated to require the same depths and bottom times as the previous trip. He was advised that further diving with
the same gas mix on similar depth/time profiles would have a high probability of resulting in a recurrence of his visual problems. Two potential strategies were discussed to avoid a recurrence of his symptoms. The first was to recalibrate his UBA to function at a lower \( \text{PO}_2 \). This would, of course, result in a decrease in the allowed no-decompression time or an increase in required decompression stop time. The other option was to shorten the dive times and increase the periods of normoxia at the surface while the film in the camera was changed. Longer periods of normoxia might prolong the time required for the presumed oxidative changes to occur in the lens of the eye. Researchers at the US Navy Submarine Development Group One and the University of Pennsylvania have demonstrated that brief periods of normoxia during \( \text{O}_2 \) breathing at 2 atm abs resulted in a marked delay in the onset of pulmonary oxygen toxicity (16). This observation resulted in the short air breaks now present during HBO2 breathing during US Navy treatment tables (17). Pulmonary oxygen toxicity has recently been reported to be the limiting factor in closed-circuit \( \text{O}_2 \) diving at a \( \text{PO}_2 \) of approximately 1.6 atm abs (18).

The diver used both of the strategies mentioned above and, in addition, broke up the diving in the second trip by doing 6 days of surface photography and filming during the middle of the trip. His next dive trip began on 11 April 1998. There were 18 days of diving with daily dive times ranging from 1.75 to 5.75 h (mean 3.6 h). All dives were on the Biomarine Mark 155 with the \( \text{O}_2 \) set point reduced to 1.2 atm abs. The benefit from this strategy may have been reduced by the fact that bottom times were not decreased from the previous dive series and the lower \( \text{PO}_2 \) required more decompression time. Decompression stops were performed at 9.2 and 4.6 m (30 and 15 ft) stop depths, with the UBA purged with \( \text{O}_2 \) to maintain a \( \text{PO}_2 \) of 1.1–1.2 atm abs. Dive times for the second trip typically ranged from 60 to 90 min on the bottom followed by a 15–20 min air break while the film and camera batteries were being changed. This cycle was repeated 2–3 times per day for each of the dive days. The diver monitored his distance visual acuity using a Snellen eye chart, which was brought along on the dive trip for this purpose. Despite these preventive measures, the diver began to notice a decrease in his distance acuity after 9 days of diving. At this point, he refrained from diving for 4 days in the middle of the trip in an attempt to minimize further myopic changes. Post-trip optometric examination revealed the following refraction:

\[
\begin{align*}
\text{OD: } -0.75 & -1.00 \times 170 \\
\text{OS: } -0.50 & -2.25 \times 175 
\end{align*}
\]

His corrected visual acuity remained at 20/20 OU throughout all of the above examinations. The smaller myopic shift noted after the second trip also slowly resolved over the ensuing weeks.

On a third dive trip, all three divers monitored their distance visual acuity with a Snellen eye chart and, with hand-held trial lenses, attempted to estimate any myopic shifts that they encountered. This was done under consistent lighting conditions and wearing whatever refractive correction was typically used by each diver. After 15 days and approximately 45 h each of diving (with their UBAs set at a 1.3 atm abs \( \text{PO}_2 \)), all three divers noted a myopic shift of 0.5–1.5 diopters. Although these refractive findings are only approximations at best, they suggest that the myopic shift in the index diver may not be an uncommon occurrence at the level of hyperoxic stress these divers encountered.

CONCLUSIONS

- A myopic shift occurred in a closed-circuit scuba diver using a 1.3 atm abs constant \( \text{PO}_2 \) in a nitrogen–oxygen mix. This change was due to hyperoxic myopia, one component of lenticular \( \text{O}_2 \) toxicity.
- The myopic shift noted above resolved over a 1-mo. period after completion of the hyperoxic exposures.
- A transient decrease in intraocular pressure was noted in association with the above finding.
- Lenticular \( \text{O}_2 \) toxicity may result in both a myopic shift and subsequent lens opacification. These findings are identical to the changes seen in age-related nuclear sclerotic cataracts except for a much-accelerated time course.
- Although the use of antioxidant supplements has recently been reported to decrease the incidence of age-related nuclear sclerotic cataracts, a commercially available antioxidant vitamin supplement did not prevent hyperoxic myopia from developing in this diver.
- Lowering the \( \text{PO}_2 \) to 1.2 atm abs and increasing the normoxic period between hyperoxic exposures did not prevent a recurrence of hyperoxic myopia on a subsequent dive trip.
- On a third dive trip, a myopic shift was found in both the index diver as well as his two diving partners using a Snellen eye chart and self-refraction with hand-held lenses.
- Diving communities should be aware of the risk of lenticular and pulmonary \( \text{O}_2 \) toxicity when conducting intensive diving at a \( \text{PO}_2 \) in the 1.3–1.6 atm abs range.
- Research into optimized doses of antioxidants or other measures designed to prevent both lenticular and pulmonary \( \text{O}_2 \) toxicity should be undertaken as diving communities anticipate the use of greater levels of hyperoxia in their UBAs.

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HYPEROXIC MYOPIA IN SCUBA DIVER

REFERENCES
