

BRIEF REPORT

The effect of hyperbaric oxygen on intraocular pressure.

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Ersanli D., Akin T., Yildiz S., Akin A., Bilge A.H., Uzun G. The effect of hyperbaric oxygen on intraocular pressure. *Undersea Hyperb Med* 2006; 33(1):1-4. The effects of hyperoxia on intraocular pressure (IOP) have been studied in experiments on human beings and animals. The changes occurring in IOP in patients during routine HBO₂ therapy are unknown. In this study we investigated IOP changes arising during the HBO₂ therapy at 2.5 ATA. Fifty-six patients receiving HBO₂ therapy for various reasons were included in the study. Bilateral IOPs of patients measured with the Tono-pen XL® (Medtronic, Solan, USA) tonometer before, during and after HBO₂ therapy. Average IOPs were 14.85 ± 3.17 mmHg (range, 10-24), 13.00 ± 2.97 mmHg (range, 9-21) and 14.74 ± 3.12 mmHg (range, 10-22), respectively. IOP was reduced significantly during HBO₂ therapy and returned its pre- HBO₂ levels after therapy. Our data indicated a statistically significant decrease in IOPs during therapy at 2.5 ATA. This decrease was of minor physiological significance in these patients whose baseline IOP values were within the normal range.

INTRODUCTION

Hyperbaric oxygen (HBO₂) therapy requires breathing of 100% oxygen at a pressure greater than 1 atmosphere absolute (ATA) (1). At a pressure of 2.5 ATA, a patient is exposed to an inspired PO₂ of 1900 mmHg. Such PO₂ values allow more oxygen to reach the tissues by raising the dissolved oxygen concentration in the plasma (2), which is beneficial for many diseases in which hypoxia is an agent in the physiopathology (3).

In ophthalmology, HBO₂ is used for ischemia of the central retinal artery (3). In studies on the influence of HBO₂ on the eye Bojic reported a positive effect on field of vision in patients with open angle glaucoma (4). The most frequently observed ophthalmological

complication in routine therapy pressures is temporary myopia (3,5-7). Cataract is also reported after chronic long-term HBO₂ therapies(3).

The effects of hyperoxia on intraocular pressure (IOP) have been studied in experiments on human beings and animals (8-11). Normobaric hyperoxia has been shown to cause IOP to fall (9,10). The changes occurring in IOP while patients breathe 100% oxygen during routine HBO₂ therapy are unknown. In this study we investigated IOP changes arising during clinical HBO₂ therapy at 2.5 ATA.

METHODS

Fifty-six patients receiving HBO₂ therapy for various indications at the GMMA Haydarpaşa Training Hospital, Department of

Underwater and Hyperbaric Medicine, were included in the study. The study protocol was approved by the Local Ethics Committee. All subjects consented to participate in the study. Of the 56 patients 30 (53.5%) were men and 26 (46.4%) were women, with an average age of 54.72 ± 5.62 (range 42-62). Those with diabetes, hypertension or cardiovascular complaints were excluded. Corrected visual acuity levels were determined prior to subjects being exposed to hyperbaric environment, and anterior and posterior segment examinations were performed.

A Tono-pen XL[®] (Medtronic, Solan, USA) tonometer was used to measure IOPs following the drop-wise administration of topical proparacaine (Alcaine[®], Alcon Lab). Tono-pen XL[®] calculates a mean and coefficient of variations using micro strain gage technology, and a 1.5mm transducer tip covered with a disposable latex cover. The Tono-pen XL[®] provides accurate IOP readings comparable to the Goldmann applanation tonometer (12). The Tono-pen XL[®] has been used in hypobaric environments and suggested to be unresponsive to changes in ambient pressure (13, 14).

Bilateral IOPs were measured at 1 ATA (room air) before HBO₂ therapy. HBO₂ therapy was carried out in a multiplace hyperbaric chamber (Galeazzi, Italy) in the Department of Underwater and Hyperbaric Medicine. Atmospheric pressure was gradually raised from 1 to 2.5 ATA inside the hyperbaric chamber over approximately 10 minutes, and at depth the patients began breathing 100% oxygen by mask. Twenty minutes after patients donned their masks IOP measurements were performed bilaterally. Bilateral IOP measurements were performed again in all subjects at 1 ATA (room air) after HBO₂ therapy. The three IOP measurements (before, during, and after HBO₂) were compared with each other using one-way analysis of variance (ANOVA) followed by post hoc Bonferroni test, and a significance

level was set at $p < 0.05$.

RESULTS

Table 1 shows IOPs of both eyes measured before, during and after HBO₂. Average IOPs at 1 ATA (room air) in the 112 eyes of the 56 subjects were 14.85 ± 3.17 mmHg (range, 10-24) before HBO₂ therapy . Average IOPs of subjects decreased to 13.00 ± 2.97 mmHg (range, 9-21) after 20 minutes of breathing 100% O₂ at 2.5 ATA. IOP returned to normal levels after HBO₂ therapy ended - 14.74 ± 3.12 mmHg (range, 10-22). Significant IOP changes were observed during HBO₂ therapy ($p < 0.05$) compared with IOP measurements prior to HBO₂ therapy.

Table 1. The intraocular pressures (IOP) of subjects measured before, during and after hyperbaric oxygen (HBO₂) therapy.

	IOP (Mean \pm SD)		
	Right Eye (n=56)	Left Eye (n=56)	Average (n=112)
Before HBO ₂	15.11 \pm 3.41	14.53 \pm 2.95	14.85 \pm 3.17
During HBO ₂	13.05 \pm 3.32	12.87 \pm 2.63	13.00 \pm 2.97*
After HBO ₂	14.55 \pm 3.21	14.33 \pm 2.67	14.47 \pm 2.93

* significant decrease compared to before HBO₂ measurements ($p < 0.05$).

DISCUSSION

Our knowledge of how hyperbaric oxygen alters IOP is limited. Previous studies measured IOPs with Goldmann applanation tonometry (4,9-11) Gallin-Cohen et al. showed a decrease in IOP levels measured using Goldmann applanation tonometry in both subjects breathing air at 3 ATA (PO₂ = 520 mmHg) and subjects breathing 90-95% O₂ at 1

ATA (pO₂ = 722 mmHg) (9). Kalthoff and John reported an average fall of 2-3 mmHg in IOPs of scuba divers at 2 and 4 ATA in a pressure tank (10). Hosking et al. studied IOP changes with induced hypercapnia and hyperoxia. They showed IOP reductions both in glaucoma patients and in normal subjects under hyperoxic conditions (breathing 100% O₂ at 1 ATA for 5 minutes), and also showed IOP changes during hypercapnia. They reported a reduction in ocular blood flow in normal subjects based on vasoconstriction in the ocular veins, and that small diameter veins were more sensitive to hyperoxia than large diameter veins (11).

Bojic et al. stated that HBO₂ (breathing 100% O₂ at 2 ATA) did not influence IOP levels in their study of glaucoma patients (4). Bojic measured IOP after HBO₂ therapy out of the chamber. Patients' IOPs were regulated with medication. We also did not find significant changes between pre- and post- HBO₂ IOP measurements.

The reduction in IOP was proposed to be caused by a fall in choroidal volume occurring as a result of an increase in oxygen partial pressure, a 40% level reduction in intraocular fluid volume or a reduction of 3 mmHg in episcleral pressure, although the actual mechanism is unknown (9). It is thought that increased oxygen concentration (9) with breathing of 100% oxygen by mask during HBO₂ therapy or a reduced carbon dioxide concentration (15) is responsible for the falling IOP, although the effect of arterial pH and carbon dioxide is unknown. Marcus et al. showed that increased CO₂ partial pressure during exercise leads to a fall in blood pH and also in IOP (15). CO₂ and H⁺ ion increase and reduced pH have also been reported among the known effects of HBO₂ therapy (2). The active transport mechanism that plays the major role in the aqueous humor formation may be inhibited by increases in H⁺ ion.

In summary, previous studies

have reported 2-3 mmHg decrease in IOP measurements when patients are exposed to hyperoxia at 522 mmHg, 720 mmHg PO₂ (9) and 760 mmHg PO₂ (11). In our study, we measured IOP at a much higher ambient PO₂ (1900 mmHg) than previous studies but saw the same IOP reduction. This decrease was statistically significant and the values returned to baseline levels after HBO₂.

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