Original articles

The myopic shift associated with hyperbaric oxygen administration is reduced when using a mask delivery system compared to a hood – a randomised controlled trial

Michael H Bennett[1,2], Cheng FB Hui[1,2], Hooi G See[3], Kwan L Au-Yeung[4], Christopher Tan[5], Stephanie Watson[1,6]

[1] *Prince of Wales Clinical School, University of New South Wales, Sydney, Australia*
[2] *Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Sydney, Australia*
[3] *Division of Anaesthesiology and Perioperative Medicine, Singapore General Hospital, Singapore*
[4] *Department of Emergency Medicine, Princess Elizabeth Hospital, Hong Kong*
[5] *Department of Anaesthesia, The Wollongong Hospital, Wollongong, Australia*
[6] *Save Sight Institute, Discipline of Ophthalmology, The University of Sydney, Sydney, Australia*

Corresponding author: *Professor MH Bennett, Prince of Wales Clinical School and Academic Director, Wales Anaesthesia, Ground Floor, East Wing Edmund Blackett Building, Prince of Wales Hospital, Barker Street, Randwick, NSW 2031, Australia*
m.bennett@unsw.edu.au

Key words
Side effects; Hyperbaric oxygen; Ophthalmology; Myopia; Vision

Abstract
(Bennett MH, Hui CF, See HG, Au-Yeung KL, Tan C, Watson S. The myopic shift associated with hyperbaric oxygen administration is reduced when using a mask delivery system compared to a hood – a randomised controlled trial. Diving and Hyperbaric Medicine. 2019 December 20;49(4):245–252. doi: 10.28920/dhm49.4.245-252. PMID: 31828742.)
Introduction: A temporary myopic shift is a well-recognized complication of hyperbaric oxygen treatment (HBOT). Oxidation of proteins in the crystalline lens is the likely cause. Direct exposure of the eye to hyperbaric oxygen may exacerbate the effect. Our aim was to measure the magnitude of the myopic shift over a course of HBOT when using two different methods of oxygen delivery.
Methods: We conducted a randomised trial of oxygen delivery via hood versus oronasal mask during a course of 20 and 30 HBOT sessions. Subjective refraction was performed at baseline and after 20 and 30 sessions. We repeated these measurements at four and 12 weeks after completion of the course in those available for assessment.
Results: We enrolled 120 patients (mean age 57.6 (SD 11.2) years; 81% male). The myopic shift was significantly greater after both 20 and 30 sessions in those patients using the hood. At 20 treatments: refractory change was -0.92 D with hood versus -0.52 D with mask, difference 0.40 D (95% CI 0.22 to 0.57, $P < 0.0001$); at 30 treatments: -1.25 D with hood versus -0.63 with mask, difference 0.62 D (95% CI 0.39 to 0.84, $P < 0.0001$). Recovery was slower and less complete in the hood group at both four and 12 weeks.
Conclusions: Myopic shift is common following HBOT and more pronounced using a hood system than an oronasal mask. Recovery may be slower and less complete using a hood. Our data support the use of an oronasal mask in an air environment when possible.

Introduction

Hyperbaric oxygen treatment (HBOT) is used for the treatment of both decompression illness (DCI) following compressed gas breathing and a range of other indications where the administration of high oxygen pressures has been shown to improve outcome.[1,2] While HBOT is generally considered safe, as with most medical procedures, adverse effects can occur.[3] Although not always documented, the most common adverse effect following HBOT is the development of a temporary myopic shift (a negative change in refraction on formal assessment). Previous reports suggest the expected refractive change in phakic eyes (natural lens present) is about -0.5 to -0.74 diopters (D) over a typical course of treatment, with about 75% of individuals experiencing a measurable shift in at least one eye.[4,5]

It is our experience that a substantial proportion of patients are significantly impacted by this change, being unable to easily view the television or safely drive a motor vehicle. In most cases this refractory change is temporary but most reports suggest this may take several weeks to resolve.[6]

HBOT can be administered in a multiplace chamber via a hood or an oronasal mask, the choice of which may influence the degree of myopic shift in the patient.[6] The aim of this

Figure 1
Hood (A) and mask (B) delivery systems. This oronasal circuit is assembled on-site from components manufactured by Hudson RCI®, NC, USA

trial was to compare the development of refractive changes in patients allocated randomly to receive HBOT via oronasal mask or hood over a treatment course of at least 20 sessions, and to document the rate of recovery in those who returned for review.

Methods

Following local ethics committee approval (South Eastern Sydney Area Health Service 10/128), we conducted an open (unmasked) randomised controlled trial comparing a course of HBOT using a hood administration system (Amron Oxygen Treatment Hood, Amron International, Vista, CA) versus an oronasal mask system assembled in our treatment centre (see Figure 1). This trial is registered on the Australian New Zealand Clinical Trials Registry (Trial ID: ACTRN12609000619246).

We included any patient where the treating hyperbaric physician planned a course of HBOT between 20 and 30 treatment sessions at 243 kPa (2.43 atmospheres absolute (atm abs)) for 90 minutes administered once daily, Monday to Friday over a four or six-week period. In our facility, some indications routinely receive 20 and others 30 sessions. Exclusion criteria were the inability to comfortably and effectively use either delivery system, a corrected visual acuity of less than 6/12 on initial assessment for treatment or the presence of non-native lenses in both eyes following intra-ocular implant surgery.

Following informed consent, each patient was assessed for visual acuity using a standard Snellen Chart, then auto-refraction and keratometry (measuring the curvature of the anterior surface of the cornea and calculating the refractory power of the cornea) were performed (Zeiss VISUREF 100, Carl Zeiss Pty Ltd, North Ryde). One of the investigators then formally assessed subjective refraction of both eyes using standard techniques with a trial frame, Jackson Cross Cylinder Lens and both cylinder and spherical lenses.[7] The auto-refraction settings were used as the starting point for this procedure. The final best subjective refraction was recorded for all phakic eyes.

Prior to the first therapeutic compression, one of the investigators (MB) consulted a computer-generated randomisation schedule to determine group allocation.[8] All treatments were conducted on a standard treatment protocol at 243 kPa for a total of 90 minutes breathing oxygen in a multiplace chamber (Fink Engineering, Warana, Queensland). On reaching treatment pressure, each patient was assisted to correctly apply the randomised delivery system for the duration of the treatment.

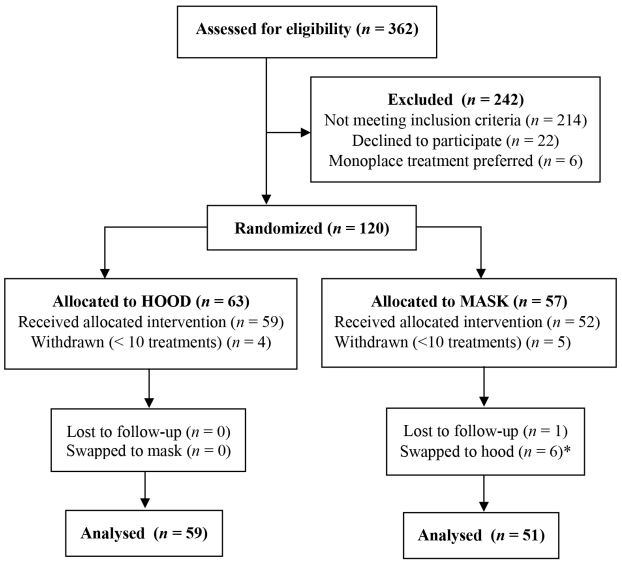
The primary outcome was the comparative change in the mean subjective refraction (myopic shift) of all eyes between groups at completion of 20 and 30 HBOT sessions according to the allocation to group (intention to treat). Secondary outcomes were myopic shift using a per protocol received approach, the proportion of eyes with a deterioration in subjective refraction of ≥ one diopter (D), changes in keratometry, changes in eye-related symptoms using a five-point scale (0-none to 4-very severe) for each of six dimensions (blurred vision, discomfort, difficulty with street signs/shop names, daytime driving, night driving and reading)[9] and any oxygen-related adverse effects of treatment. In addition, we estimated the rate of recovery of subjective refraction in those subjects available for examination at four and 12 weeks following the final treatment session.

STATISTICAL METHODS AND SAMPLE SIZE

Sample size was estimated based on the results of Evanger 2004 and on defining a myopic shift of ≥ 0.5 D as clinically significant.[6] We estimated 60% and 83% of eyes in the mask and hood group respectively would develop a myopic shift

Figure 2

PRISMA flow diagram for the study. Patients were formally withdrawn from the study if they received fewer than ten treatment sessions. *Patients swapping from mask to hood during the course of treatment were analysed as allocated for intention to treat and moved to the hood group for a secondary per protocol analysis



of at least 0.5 D, and power calculations suggested that for an 80% chance of finding a difference of 20% between the groups at significance level of $P < 0.05$, a sample size of 120 subjects would be required (60 in each group). After plotting all continuous variables to visually examine distributions, we analyzed any differences between groups using Student's *t*-test where data were approximately normally distributed and the Mann-Whitney U test (MWUT) when the normal assumption was not sustainable. One-way ANOVA was used to compare multiple groups where appropriate. Results are presented as mean with standard deviation (SD) or 95% confidence intervals (CI), or as median and inter-quartile range (IQR) as appropriate. One-way ANOVA results were presented with the F-test and *P*-value. Differences in proportions were analyzed using Chi-squared testing and presented with 95% CI. All statistical calculations were made using StatsDirect software package 3.1.22 (StatsDirect Ltd, UK).

Results

One-hundred-and-twenty patients met all inclusion criteria and none of the exclusion criteria (63 allocated to oxygen administration via the hood and 57 via the mask). Nine patients were withdrawn from the study as they received fewer than ten HBOT sessions (four in the hood and five in the mask group). Six patients elected to change from using a mask to using a hood for oxygen delivery during their course of treatment. These patients were included in the primary intention to treat analysis as allocated, but swapped groups for the 'per protocol' secondary analysis. Figure 2 details the patient flow through the study.

Table 1

Demographic data and co-morbidities in each group. Data are *n* (%) other than age reported as mean (SD)

Characteristic	Hood (*n* = 59)	Mask (*n* = 51)
Mean age in years (SD)	58.9 (11.1)	57.9 (11.5)
Sex female	17 (28.8)	22 (43)
> 4 pack year smoker	25 (42)	19 (37)
Diabetes mellitus	8 (14)	4 (8)
Hypertension	26 (44)	19 (37)
Hypercholesterolaemia	19 (32)	13 (26)
Radiotherapy in past	49 (83)	43 (84)
Eye pathology	3 (5)	2 (4)
Indication for treatment:		
Soft tissue radiation injury	29 (48)	26 (51)
Osteoradionecrosis	20 (33)	17 (33)
Problem wound	7 (12)	5 (10)
Other	4 (7)	3 (6)

In total, data from 210 eyes of 104 patients are included in our analysis after 20 treatments (55 patients in the hood group versus 49 in the mask group) and 80 patients after 30 treatments (43 in the hood group and 37 in the mask group) contributing data from 155 eyes.

The patient characteristics in each group are presented in Table 1. There were no clearly important differences between groups in demographics, comorbidities or indication for hyperbaric treatment with the exception of the sex ratio (29% female in the hood group versus 43% in the mask group).

For the primary outcome, a myopic shift was confirmed in both groups after both 20 and 30 treatments and this change was statistically significantly greater in those patients using the hood versus the mask (at 20 treatments: mean subjective refractory change -0.92 D with the hood versus -0.52 D with the mask, difference between groups 0.40 D (95% CI 0.22 to 0.57, $P < 0.0001$); at 30 treatments: -1.25 D with the hood versus -0.63 with the mask, difference 0.62 D (95% CI 0.39 to 0.84), $P < 0.0001$). The per-protocol analysis produced similar results. The intention to treat and per-protocol results are shown in detail in Table 2. The mean myopic shift was not statistically different between males and females (mean in males -1.3 D versus -0.9 D. $P = 0.2$ after 30 treatments).

Patients using the hood system were significantly more likely to have a clinically important change in refraction over the course of treatment of ≥ 1.0 D in either one or both eyes (for example, after 30 treatments: hood group proportion with both eyes affected 24/45 (53%), mask group 5/33 (15%), relative risk 3.5 (95% CI 1.6 to 8.3), $P = 0.0006$). The results are detailed in Table 3.

A high proportion of individuals in both groups had an eye-related symptom score of zero at baseline, indicating

Table 2
Mean refractory change with hood and mask oxygen delivery systems at completion of both 20 and 30 hyperbaric treatments. Both the primary (intention to treat) and per protocol analyses are shown; D = dioptres

Comparison (*n* eyes)	Hood mean (D) (95% CI)	Mask mean (D) (95% CI)	Difference mean (D) (95% CI)	*P*-value
Intention to treat analysis				
Baseline to 20 treatments (117 hood / mask 93)	-0.92 (-1.05 to -0.78)	-0.52 (-0.63 to -0.41)	0.40 (0.22 to 0.57)	0.0001
Baseline to 30 treatments (84 hood / mask 71)	-1.25 (-1.41 to -1.08)	-0.63 (-0.78 to -0.48)	0.62 (0.39 to 0.84)	0.0001
Per protocol analysis				
Baseline to 20 treatments (108 hood / mask 85)	-0.93 (-1.06 to -0.80)	-0.42 (-0.53 to -0.32)	0.51 (0.35 to 0.68)	0.0001
Baseline to 30 treatments (hood 90 / mask 65)	-1.22 (-1.38 to -1.06)	-0.60 (-0.74 to -0.46)	0.62 (0.40 to 0.82)	0.0001

Table 3
Proportions of eyes with at least one D myopic shift after 20 and 30 treatments, between group comparison and relative risk in hood group compared to mask

Comparison	Eyes with ≥ one D myopic shift	*n* / denominator (%)		Chi^2 and *P*-value	Relative risk (95% CI)
		Hood	Mask		
After 20 treatments	One or both eyes	40/59 (68)	9/43 (21)	21.9 $P < 0.0001$	3.2 (1.9 to 6.1)
	Both eyes	18/59 (31)	3/43 (9)	8.4 $P = 0.004$	4.4 (1.5 to 13.4)
After 30 treatments	One or both eyes	37/45 (82)	13/33 (39)	15.2 $P < 0.0001$	2.1 (1.4 to 3.4)
	Both eyes	24/45 (53)	5/33 (15)	11.9 $P = 0.0006$	3.5 (1.6 to 8.3)

Table 4
Keratometry in both groups at baseline, 20 treatments and 30 treatments. CRC = corneal radius of curvature in millimetres; RPC = refractive power of the cornea in Dioptres

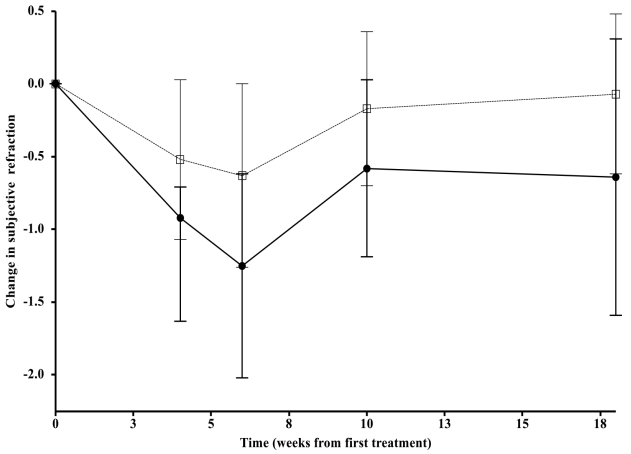
Group	CRC; baseline Mean (95% CI)	CRC; 20 treatments Mean (95% CI)	CRC; 30 treatments Mean (95% CI)	One-way ANOVA and *P*-value
Hood	7.82 (7.76 to 7.87)	7.81 (7.75 to 7.86)	7.83 (7.76 to 7.91)	F = 0.19, $P = 0.83$
Mask	7.69 (7.54 to 7.84)	7.70 (7.60 to 7.80)	8.02 (7.55 to 8.49)	F = 2.04, $P = 0.13$
	RPC; baseline Mean (95% CI)	**RPC; 20 treatments Mean (95% CI)**	**RPC; 30 treatments Mean (95% CI)**	
Hood	43.3 (43.0 to 43.6)	43.3 (43.0 to 43.6)	43.2 (42.7 to 43.6)	F = 0.10, $P = 0.90$
Mask	43.2 (42.4 to 44.0)	43.4 (42.9 to 43.9)	43.5 (43.2 to 43.9)	F = 0.14, $P = 0.90$

no problematic issues with vision (hood 50/63 (79%), mask 38/55 (69%). There was evidence of a deterioration in scores in the hood group after both 20 and 30 treatments: baseline median 0 (IQR 0 to 0); after 20 treatments median 0 (IQR 0 to 4), MWUT using exact probabilities $P < 0.0001$; and after 30 treatments median 2 (IQR 0 to 2), MWUT $P < 0.0001$. There was a difference in median scores of 2 (95% CI 0 to 4). There was no statistically significant deterioration in scores at either time in the mask group: baseline median 0 (95%CI 0 to 1); after 20 treatments 0 (0 to 2), MWUT $P = 0.18$; after 30 treatments 0 (0 to 1.5), MWUT $P = 0.75$.

There were no important changes in keratometry over the course of treatment in either group. For example, the mean corneal radii of curvature in the hood group at baseline, 20 and 30 treatments were 7.82 (95% CI 7.76 to 7.87), 7.81 (7.75 to 7.87) and 7.83 (7.76 to 7.91) respectively, and one-way analysis of variance suggested no statistically significant differences (F = 0.19, $P = 0.83$; Table 4).

In order to obtain some idea of the rate of improvement in myopic shift after cessation of treatment, we re-examined those patients who returned for review at four and 12 weeks (Figure 3). At four weeks the hood group ($n = 24$) had improved from values obtained after both 20 and 30 treatments to a mean shift of -0.58 D (95% CI -0.74 to -0.42) while at 12 weeks ($n = 14$) there was little further change (mean shift -0.64 D, 95% CI -1.02 to -0.26). For the mask group, there was an improvement at four weeks to a mean shift of -0.17 D (95% CI -0.31 to -0.02, $n = 23$) and at 12 weeks the mean shift was -0.07 D (95% CI -0.33 to 0.19, $n = 10$).

Figure 3
Mean myopic shift over time in mask and hood groups. Open squares = mask group; Closed circles = hood group. Error bars show standard deviation



Discussion

This study demonstrated myopic shift is more pronounced when using a hood system to deliver hyperbaric oxygen compared to using a mask during a standard 90 minute HBOT protocol at 243 kPa daily, Monday to Friday. The shift is about twice the magnitude with a hood after both 20 and 30 treatments and the risk of developing a shift of ≥ 1 D in at least one eye was about three times higher using a hood than a mask. The available data at four and 12 weeks after completion of treatment suggest recovery may be considerably slower and perhaps less complete after using a hood system.

Individuals with myopic shift experience poorer distance vision. This may impact considerably on many activities of daily living such as driving, playing sports, reading street signs, and watching television.[9] It is our practice to recommend correction of any myopia by the purchase of widely available cheap, temporary non-prescription spectacles. There are also adjustable-power spectacles available as an alternative. Even in hyperopic individuals, where a myopic shift can technically correct the refractive error to some degree, the refractive change can cause their prescription glasses to be incompatible with good vision for the duration of the myopic shift. Further, as the myopic shift is generally temporary and variable, it is impractical for patients to obtain new prescription glasses as required.

HBOT induced myopia was first reported in 1978 in a series of ten patients who had received 40 sessions of HBOT at 203 kPa (2 atm abs) for two hours daily, Monday to Friday.[10] The mean myopic shift was -1.6 D (95%CI -0.5 to -2.5). Since then, a succession of studies have published similar results that together suggest: masks produce smaller myopic shifts than either hoods or monoplace treatments in 100% oxygen-filled chambers; that this problem is evident in phakic eyes rather than those with intra-ocular lens replacements; and that the degree of myopic shift is positively correlated with the number of treatment sessions (Table 5).

On this basis, myopia is widely acknowledged as a common side effect of HBOT. In our facility, patients are routinely advised of this possibility and we suggest the myopic shift will resolve over a period of weeks, although after extreme exposures Palmquist 1978 reported that some patients never return to baseline and a small number develop formal cataracts.[12] The authors of a large, prospective cohort of elderly individuals have independently postulated a myopic shift itself can predispose to the development of cataracts.[15]

The exact mechanism by which HBOT causes myopia is not known with certainty. Several models have been hypothesized to explain the anatomical basis of refractive change, but many of these have little support from the evidence available. To date, no changes in corneal curvature (confirmed in the present study), anterior chamber depth, axial eye length or lens diameter have been identified in patients with HBOT induced myopia.[4,13,16]

By elimination, these findings are highly suggestive of a lenticular etiology for refractive change and there is corroborating evidence available to support this proposition. Both Khan 2003 and Evanger 2011 demonstrated a much higher incidence of myopia in phakic eyes over pseudophakic eyes following HBOT; indeed none of the pseudophakic eyes developed significant myopia.[5,14] The evidence, confirmed in our study, that hoods produce greater myopic shifts than the same oxygen dose delivered by oronasal mask suggests the exposure of the eye directly to 100% oxygen at pressure is the primary causative factor. During a typical hyperbaric treatment, unlike hoods or 100% oxygen-filled monoplace treatments where the eye is exposed to 203 to 243 kPa of oxygen, wearing an oronasal mask only exposes the eye to air at pressure – approximately 40 to 50 kPa of oxygen.

While hyperbaric practitioners are quick to invoke high arterial oxygen tensions as the primary driver of therapeutic mechanisms for HBOT, there are anatomical and physiological reasons to suggest it is direct exposure of the anterior chamber of the eye to high oxygen tensions that produces the observed effects on refraction. The cornea and lens are avascular structures and receive a significant proportion of oxygen by passive diffusion from the air. Oxygen diffuses down a gradient from the pre-corneal tear film, through the cornea, into the anterior chamber and thence to the lens.[17] There is also some diffusion to the rear of the lens from the retinal artery through the vitreous humour as well as a component from the choroidal circulation. Increased oxygen tension in the aqueous humour has been reported in studies when rabbit corneal surfaces were exposed to HBO while maintaining normal respiration with ambient air.[18]

Structural changes in the lens after HBO exposure have also been reported, including altered phospholipid composition in lens epithelial cells, decreased protein sulfhydryl groups

Table 5
Summary of studies examining the myopic shift associated with hyperbaric oxygen. RCT = randomised controlled trial; * = duration of hyperbaric treatments not specified

Study	Type and exposure	Delivery system	Myopic shift
Anderson and Farmer 1978[10]	Case series *n* = 10 40 x 203 kPa x 120min	Hood	Mean -1.6 D (95% CI -0.5 to -2.5)
Lyne 1978[11]	Case series *n* = 26 20–260 x 243 kPa x 120min	Monoplace 100% oxygen environment	Range -0.5 D to -5.5 D
Palmquist et al. 1978[12]	Non randomised cohorts *n* = 25: 75–425 x 203–243 kPa x 120min *n* = 19: 'control' on waitlist	Monoplace 100% oxygen environment	24/25 treated pts ≥ 1 D vs none in control pts Mean -3.0 D (no CI or SD given) in treated pts
Ross et al. 1996[13]	Case series *n* = 8 20 x 253 kPa x 120min	Monoplace 100% oxygen environment	2/8 had shift of ≥ -0.5 D
Fledelius et al. 2002[4]	Case series *n* = 17 20 x 253 kPa x 95min	Mask	Mean -0.49 (no CI or SD given)
Khan et al. 2003[5]	Cohort *n* = 43 (75 phakic eyes versus 11 pseudophakic eyes) 30-40 x 203–243 kPa x ?*	Monoplace 100% oxygen environment	Means: Phakic: -0.74 D (SE 0.12) Pseudophakic: -0.03 D (SE 0.05)
Evanger et al. 2004[6]	RCT *n* = 32 21 x 243 kPa x 90min	Hood *n* =12 Mask *n* = 20	Mean -1.08 D (SD 0.54) Mean -0.54 D (SD 0.41)
Evanger et al. 2011[14]	Cohort *n* = 22 (32 phakic eyes versus 12 pseudophakic eyes) 20 x 243 kPa x 90min	Monoplace 100% oxygen environment	Phakic: median -0.63D (Range -0.25 D to -1.88 D) Pseudophakic: median 0.06 D (Range -0.13 to 0.25 D)

and increased disulfide formation, and loss of cytoskeleton proteins.[19–21] The refractive index of the lens is highly dependent on the protein concentration within the lens and changes resulting from oxidative damage to lens proteins are likely to have direct effects on the refractive power of the lens.[22]

These effects suggest an increased oxidative stress induced by prolonged exposure to elevated oxygen tension. Interestingly, there are some similarities with changes described in the lens during nuclear cataract formation and it may be that both these processes are due to oxidative changes over different time courses.

Overall, the prevailing evidence strongly implies a lenticular etiology for HBOT-induced myopic shift, with the effects of oxidative stress being the primary mechanism. The current study supports a lenticular etiology.

Our study does have limitations. Several patients elected to switch from the mask to the hood, particularly during the early part of the recruitment phase. This was most likely due to the lower familiarity with the mask system at that time, along with the potential observation by the patient that most others were using the hood. Our per-protocol and intention to treat analyses were very similar and we do not believe these decisions affected our overall conclusions. A second potential problem was the open nature of the intervention. This introduced the potential for bias into the trial, although it is hard to envision how the subjects could influence the subjective refraction estimation in a systematic way. More important is the potential for bias due to the investigator performing the subjective refraction being aware of allocation. However, the investigators were asked not to discuss the method of oxygen delivery with the patient and were generally not otherwise involved in patient treatment. The exceptions were some evaluations performed by the first three authors when no other trained individual was available.

A higher proportion of females were enrolled in the mask group than the hood group (43% and 29% respectively). Although not statistically significantly different, the myopic shift in females was less than in males (mean -0.9 D versus -1.3 D respectively). While any systematic bias due to gender difference would tend to exaggerate the difference between oxygen delivery groups, it is also possible the observed difference between the sexes is the result of that allocation. No sex differences in myopic shift have been previously observed and we believe confounding by gender is unlikely.

It is also possible the observed changes were reflective of nothing more than a lower dose of oxygen in those using an oronasal mask. It has long been observed that entrainment of air while using an oronasal mask results in a lower effective inspired fraction of oxygen compared to the use of a hood.[23] Stephenson showed the oxygen concentration in a hood when using flows of 30 to 50 litres per minute approaches 100%, and individuals using a hood are more likely to achieve an oxygen fraction of > 0.8 within the hood than when using an oronasal mask, even when the latter are supervised by a trained nurse. These authors noted the fraction of oxygen measured in the dead space of the mask

could not be regarded as an accurate measure of the inspired fraction, influenced as it would be by the exhaled fraction. On the other hand, Sheffield had previously demonstrated that a well-fitting oronasal mask delivered a mean end-inspiratory of oxygen of 97.8 % (range 96 to 99) and this was considered satisfactory oxygen delivery.[24] Given many centres employing monoplace chambers routinely treat at 203 kPa, the equivalent oxygen dose at 243 kPa can be achieved with an inspired fraction of about 0.83. While we are confident therapeutic oxygen doses are delivered with our current oronasal mask, we are nevertheless currently evaluating the effective dose delivered to confirm this.

While it is likely recovery will take longer in those using the hood because of the greater magnitude of change, the suggestion this group achieves incomplete recovery of refraction should be interpreted with caution. These patients were re-examined at four and 12 weeks in an opportunistic way and it is quite possible those with persisting myopic shift were more likely to present at these times.

Conclusion

The use of a hood system to deliver hyperbaric oxygen results in a more profound myopic shift than when using an oronasal mask. The mean refractory change is approximately twice the magnitude with the hood and there is some indication from follow-up that the shift resolves after treatment more slowly and perhaps less completely when using the hood. The changes support a lenticular etiology to explain myopic shift associated with HBO exposure. Consideration should be given to discussing these implications with individual patients during the consent process prior to commencing HBOT and to selecting an oronasal mask as the default delivery system.

References

1 Weaver LK, editor. Hyperbaric oxygen therapy indications. 13th ed. North Palm Beach (FL): Best Publishing Company; 2014. ISBN: 978-1930536-73-9.

2 Mathieu D, Marroni A, Kot J. Tenth European consensus conference on hyperbaric medicine: recommendations for accepted and non-accepted clincial indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017;47:24–32. doi: 10.28920/dhm47.1.24-32. PMID: 28357821. PMCID: PMC6147240.

3 Heyboer M 3rd, Sharma D, Santiago W, McCulloch N. Hyperbaric oxygen therapy: side effects defined and quantified. Adv Wound Care (New Rochelle). 2017;6:210–24. doi: 10.1089/wound.2016.0718. PMID: 28616361. PMCID: PMC5467109.

4 Fledelius HC, Jansen EC, Thorn J. Refractive change during hyperbaric oxygen therapy. A clinical trial including ultrasound oculometry. Acta Ophthalmol Scand. 2002;80:188–90. doi: 10.1034/j.1600-0420.2002.800213.x. PMID: 11952487.

5 Khan B, Evans AW. Refractive changes in patients undergoing hyperbaric oxygen therapy: a prospective study. Undersea Hyperb Med. 2003;30:215. Available from: http://archive.rubicon-foundation.org/xmlui/handle/123456789/1361. [cited 2019 December 04].

6 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. doi: 10.1111/j.1395-3907.2004.00290.x. PMID: 15291940.

7 Elliott M, Simpson T, Richter D, Fonn D. Repeatability and accuracy of automated refraction: a comparison of the Nikon NRK-8000, the Nidek AR-1000, and subjective refraction. Optom Vis Sci. 1997;74:434–8. doi: 10.1097/00006324-199706000-00028. PMID: 9255823.

8 Urbaniak GC, Plous S Lastik M. Research Randomizer. Available from: https://www.randomizer.org/. [cited 2019 March 28].

9 Rose K, Harper R, Tromans C, Waterman C, Goldberg D, Haggerty C, et al. Quality of life in myopia. Br J Ophthalmol. 2000;84:1031–4. doi: 10.1007/978-0-387-78665-0_6171. PMID: 10966960. PMCID: PMC1723631.

10 Anderson B Jr, Farmer JC Jr. Hyperoxic myopia. Trans Am Ophthalmol Soc. 1978;76:116–24. doi: 10.1111/cxo.12839. PMID: 754368. PMCID: PMC1311617.

11 Lyne A. Ocular effects of hyperbaric oxygen. Trans Ophthalmol Soc UK. 1978;98:66–8. doi: 10.1016/b978-1-4160-3406-3.50029-9. PMID: 285513.

12 Palmquist BM, Philipson B, Barr PO. Nuclear cataract and myopia during hyperbaric oxygen therapy. Br J Ophthalmol. 1984;68:113–7. doi: 10.1136/bjo.68.2.113. PMID: 6691953. PMCID: PMC1040267.

13 Ross ME, Yolton DP, Yolton RL, Hyde KD. Myopia associated with hyperbaric oxygen therapy. Optom Vis Sci. 1996;73:487–94. doi: 10.1097/00006324-199607000-00006. PMID: 8843129.

14 Evanger K, Vaagbø G, Thorsen E, Haugen OH. Phakic and pseudophakic eyes in patients during hyperbaric oxygen therapy. Optom Vis Sci. 2011;88:691–6. doi: 10.1097/opx.0b013e318213f001. PMID: 21399553.

15 Kanthan GL, Mitchell P, Rochtchina E, Cumming RG, Wang JJ. Myopia and the long-term incidence of cataract and cataract surgery: the Blue Mountains Eye Study. Clin Exp Ophthalmol. 2014;42:347–53. doi: 10.1111/ceo.12206. PMID: 24024555.

16 Evanger K, Pierscionek BK, Vaagbø G, Thorsen E, Haugen OH. Myopic shift during hyperbaric oxygenation attributed to lens index changes. Optom Vis Sci. 2015;92:1076–84. doi: 10.1097/opx.0000000000000705. PMID: 26414557.

17 Kwan M, Ninikoski J, Hunt TK. In vivo measurements of oxygen tension in the cornea, aqueous humor, and anterior lens of the open eye. Invest Ophthalmol. 1972;11:108–14. PMID: 5009103.

18 Jampol LM. Oxygen therapy and intraocular oxygenation. Trans Am Ophthalmol Soc. 1987;85:407–37. PMID: 3447339. PMCID: PMC1298787.

19 Huang L, Estrada R, Yappert MC, Borchman D. Oxidation-induced changes in human lens epithelial cells. 1. Phospholipids. Free Radic Biol Med. 2006;41:1425–32. doi: 10.1016/j.freeradbiomed.2006.07.022. PMID: 17023269.

20 Bantseev V, Oriowo OM, Giblin FJ. Leverenz VR, Trevithick JR, Sivak JG. Effect of hyperbaric oxygen on guinea pig lens optical quality and on the refractive state of the eye. Exp Eye Res. 2004;78:925–31. doi: 10.1016/j.exer.2004.01.002. PMID: 15051474.

21 Padgaonkar V, Lin AR, Leverenz VR, Rinke A, Reddy VN, Giblin FJ. Hyperbaric oxygen in vivo accelerates the loss of cytoskeletal proteins and MIP26 in guinea pig lens nucleus. Exp Eye Res. 1999;68:493–504. doi: 10.1006/exer.1998.0630.

PMID: 10192807.
22 Giblin FJ, Padgaonkar VA, Leverenz VR, Lin LR, Lou MF, Unakar NJ, et al. Nuclear light scattering, disulfide formation and membrane damage in lenses of older guinea pigs treated with hyperbaric oxygen. Exp Eye Res. 1995;60:219–35. doi: 10.1016/s0014-4835(05)80105-8. PMID: 7789403.
23 Stephenson RN, Mackenzie I, Watt SJ, Ross JA. Measurement of oxygen concentration in delivery systems used for hyperbaric oxygen therapy. Undersea Hyperb Med. 1996;23:185–8. PMID: 8931286.
24 Sheffield PJ, Stork RL, Morgan TR. Efficient oxygen mask for patients undergoing hyperbaric oxygen therapy. Aviat Space Environ Med. 1977;48:132–7. PMID: 871282.

Acknowledgements

The authors thank the staff of the Departments of Diving and Hyperbaric Medicine and Ophthalmology for their assistance with this trial. Stephanie Watson is a Sydney Medical School Foundation Fellow.

Conflicts of interest and funding: nil

Submitted: 10 April 2019
Accepted after revision: 17 May 2019