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ORIGINAL ARTICLE

Phakic and Pseudophakic Eyes in Patients During Hyperbaric Oxygen Therapy

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ABSTRACT

Purpose. To examine the optical components of phakic and pseudophakic eyes during hyperbaric oxygen (HBO) therapy, and to quantify their relative impact on ocular refractive changes.

Methods. HBO therapy was given to 16 phakic and six pseudophakic patients for 90 min daily at a pressure of 240 kPa, 5 d a week for 20 days. An eye examination was performed on the first day of HBO therapy and repeated when the patients had completed 19 days of the treatment. Refractive error, best-corrected visual acuity, corneal power, radius, thickness and volume, anterior chamber depth, axial length, lens opacity, and intraocular pressure were measured in all patients. Serum glucose, glycosylated hemoglobin, serum electrolytes, and protein were measured in the phakic patients.

Results. In the phakic group, a significant myopic shift (≥−0.50 D) occurred in 26 (81%) single eyes during the treatment. The median myopic shift was −0.63 D (min −0.25 D/max −1.88 D) in the OD, and −0.69 D (min −0.38 D/max −2.25 D) in the OS. No myopic shift appeared in the pseudophakic patients; the median refractive changes were +0.06 D (min −0.13 D/max +0.25 D) in the OD and +0.13 D (min 0.00 D/max +0.25 D) in the OS. Intraocular pressure, serum electrolytes, glucose, and glycosylated hemoglobin remained unchanged.

Conclusions. Myopic shifts occurred in phakic but not in pseudophakic eyes during HBO therapy. The myopic shifts must be attributed to changes in the crystalline lens.

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Key Words: hyperbaric oxygen therapy, myopic shift, cataract, pseudophakic, intraocular lenses

Hyperbaric oxygen (HBO) therapy is a medical treatment, which has been shown to stimulate fibroblast proliferation, collagen synthesis, and neovascularization of ischemic tissue. HBO is an adjunct to treatment of selected problem wounds, necrotizing soft tissue infections, gas gangrene, and thermal burns. In this study, the main indication for HBO therapy was radiation-induced cystitis, proctitis, and osteonecrosis of the mandible after previous cancer. The patients inhale 100% oxygen for 90 min daily at a pressure of 200 to 240 kPa for 20 to 30 days. The purpose of breathing oxygen at increased pressure is to increase the pressure gradient for diffusion of oxygen into the tissue in an attempt to revitalize oxygen-dependant synthesis in ischemic tissue. An extensive review of the mechanisms of and indications for HBO treatment is given by the Undersea and Hyperbaric Medical Society.[1]

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Myopic shift in HBO-treated patients was described by Lyne[2] and Anderson and Farmer[3] in 1978. Refractive changes have repeatedly been reported as a potential ocular side effect of multiple exposures to HBO.[2–8] The amount of refractive change is related to the dose and frequency of the HBO sessions.[6] In a previous article, we reported that the myopic shift is more pronounced when the patients receive oxygen by using a hood compared with using an oronasal mask during treatment.[8] The local oxygen tensions of aqueous and lens tissue may produce more rapid and higher elevations when both the corneal surface and arterial blood are exposed to an increased oxygen pressure.[5] After cessation of HBO therapy, the myopic shift tends to resolve and return to pretreatment levels within some weeks.[7,8] In recent years, the occurrence of hypermetropic shifts associated with HBO therapy has also been reported.[9,10] Although the causal relationship remains unclear, the temporary shifts in refractive power are suggested to be related to changes within the crystalline lens.[2,4–6] Previous reports have excluded changes in corneal power,[2,3,5–7] anterior chamber depth (ACD),[2,6,7] axial length (AL) of the eye,[2,5–7] lens thickness,[6,7] and accommodative tonus[2] to explain the degree of ocular refractive changes in HBO-treated patients.

The aim of this study was to examine the individual optical components of the eye, and to quantify their relative impact on the ocular refractive changes seen in patients during HBO therapy. To specifically examine the role of lenticular refractive changes, we included a group of pseudophakic patients with an intraocular lens (IOL) in the posterior chamber after cataract surgery.

MATERIALS AND METHODS

The study sample was recruited consecutively from the population of patients referred to Haukeland University Hospital for HBO therapy. Inclusion criteria were age within 80 years, spectacle prescription within −6.00 D to +6.00 D, and a healthy anterior segment of the eye. Patients with a history of contact lens wear on a regular basis, previous corneal refractive surgery, radiotherapy of the orbit, or HBO therapy during the past 12 months were excluded. Informed consent was obtained from the patients, and the study was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate. The study was conducted according to the recommendations of the Declaration of Helsinki for research involving human subjects.

Sixteen phakic (10 males and 6 females) with a mean age of 60 years (range 47 to 77) and six pseudophakic patients (3 males and 3 females) with a mean age of 66 years (range 48 to 80) met the inclusion criteria. All the patients were white. All the pseudophakic eyes had an in-the-bag implantation after uncomplicated cataract extraction. In all cases, the IOL was well centered in the bag. Results from measurements in which the patients had completed treatments every scheduled day without interruptions were eligible for further processing. HBO treatment was given daily for 20 days, 5 days a week. The patients were compressed in a monoplace hyperbaric chamber with air to a total pressure of 240 kPa within 10 to 15 min. They were then given 100% oxygen (PO_2 240 kPa) in three cycles of 30 min interrupted by breathing air (PO_2 50 kPa) administered by an oronasal mask for two periods of 5 min between each cycle. Thereafter, the patients were decompressed to normal atmospheric pressure in 7 to 10 min.

The eye examination included determination of refractive state, best-corrected visual acuity, corneal power, curvature, thickness and volume, ACD, AL of the eye, lens opacity, and intraocular pressure (IOP). A routine examination with biomicroscope slitlamp and ophthalmoscope was performed. The initial eye examination was performed within the first day of HBO therapy (day 1), and the follow-up assessment was made within the same hour of the day when the patients had completed 19 days of the treatment. Serum glucose, blood glycosylated hemoglobin (HbA1c), serum electrolytes, serum protein, and hemoglobin concentration were measured in the phakic patients because diabetes mellitus and serum osmolarity changes may cause transient refractive changes.[11,12]

The refractive state was first assessed with an auto-refractor/keratometer (Nidek ARK 900), and thereafter by the standard subjective refraction method at distance without the use of cycloplegia. The starting point of the subjective refraction was by adding a plus sphere power (+1.00 D) to the average value of minimum three auto-refractor measures to produce a blur with reduction in visual acuity. Then a standard endpoint of highest plus or least minus accepted for best visual acuity examined with a Snellen chart was used.

IOP was measured with an ICare Rebound tonometer (Tiolat Oy, Helsinki, Finland).[13,14] AL of the eye was determined with the IOLMaster (Carl Zeiss Meditec, Jena, Germany).[15–17] An anterior eye segment analysis system, the Pentacam (Oculus, Lynnwood, WA), was applied to measure corneal power (keratometry), curvature, thickness (pachymetry) and volume, and ACD.[18,19] The system uses a rotating Scheimpflug camera and a mono-chromatic blue (ultraviolet free) slit-light source that rotate together 180° around the optical axis of the eye. The software acquires multiple images of the anterior segment and uses these to generate three-dimensional axial and tangential maps and to calculate the geometric parameters of the eye. The Pentacam defines the "true" ACD as the distance from the posterior surface of the cornea to the anterior surface of the lens. The central corneal thickness was added to the true ACD measurements to obtain the optical ACD values [that were used in crystalline lens power (LP) calculation]. All the Pentacam pictures were taken 30 min after administration of tropicamide 0.5%.

An Interzeag Opacity Lensmeter 701 (Interzeag, Schlieren, Switzerland) was used to quantify lens opacity. The instrument measures by a photometer in which the intensity of the light scattered back from the lens and displays it on a numerical scale from 0 to 100. On this scale, a normal lens ranges from 4 to 25, depending on the age of the patient.[20,21] All the eye examinations and measurements were performed by a single investigator (K.E.). Key baseline data were recorded consecutively for each patient, with no review of the test data before the follow-up examination was issued.

To calculate which impact the individual ocular refractive component had on total ocular refraction, a customary corrected vergence formula was used. The equation is intended for crystalline LP in dioptres at the anterior vertex:

$$LP = n\left(\frac{1}{AL - ACD} - \frac{1}{\frac{n}{K + RE} - ACD}\right)$$

Here n is the refractive index of the aqueous and the vitreous (n = 1.336). Keratometry (K) and refractive error in spherical equivalent are provided in dioptres. The AL of the eye and optical ACD are both specified in meters.

Statistics

Both OD and OS were measured in each subject. In the phakic patients, results from both eyes were considered for statistical analysis. In the group of pseudophakic patients, the results from the four OD and six OS with IOL implants were used. The results obtained by the monocular subjective refractive method (the fogging technique) and the objective refraction (auto-refractor) were converted into spherical equivalents for further analysis. Subjective refraction was chosen as the standard for refractive change comparison. Wilcoxon signed rank test was applied for comparison of ocular biometric parameters and blood biochemical variables taken before (day 1) and after the HBO therapy (day 19). Spearman correlation coefficient was used to verify the correlations between subjective and objective refraction measurements. Changes in astigmatic correction were analyzed within the with-the-rule and against-the-rule concepts.[22] The data were statistically analyzed using the SPSS for Windows 15.0 program (2006). The results are given as median, minimum, and maximum values. $p < 0.05$ was considered to be the level of statistical significance. All variables

used in the analysis have been subjected to checking for distribution, range, and missing values.

RESULTS

Refractive Changes

In the phakic group, a significant myopic shift (≥−0.50 D) occurred in 26 (81%) single eyes during the treatment. The refractive changes in spherical equivalents showed a median myopic shift of −0.63 D (min −0.25 D/max −1.88 D) in the OD, and −0.69 D (min −0.38 D/max −2.25 D) in the OS (Fig. 1). Comparison between subjective and objective refraction revealed high correlations, $R^2 \geq 0.95$ ($p < 0.001$) before and $R^2 \geq 0.95$ ($p < 0.001$) after the treatment in both eyes.

No myopic shift appeared in the pseudophakic eyes. The median refractive changes were +0.06D (min −0.13 D/max +0.25 D) in OD and +0.13D (min 0.00 D/ max +0.25 D) in the OS. Two patients in the pseudophakic group had an IOL implant in their OS only. One of these patients revealed a refractive change of −1.12 D in the phakic eye and +0.12 D in the pseudophakic eye. The second patient showed −0.25 D in the phakic eye and had no refractive change (0.00 D) in the pseudophakic eye. Before cataract extraction during HBO therapy 1 year ago, this patient had been measured with a myopic shift of −0.63 D and −1.00 D in the OD and OS, respectively.

A number of 28 (87%) single eyes in the phakic group and 10 (100%) single eyes in the pseudophakic group showed no significant shifts in cylinder power (≤0.25 D). Changes in total astigmatism (cylinder power) larger than 0.50 D were not found. The calculated polar value (cylinder axis) revealed only minute fluctuations in the balance between the with-the-rule and against-the-rule components for both groups.

Best-corrected visual acuity ranged from 6/18 to 6/4. In one bilateral pseudophakic patient, a change in monocular visual acuity from 6/5 before to 6/4 after HBO therapy was measured. In this patient, drusen in both maculas had been recorded by the surgeon at the preliminary examination before cataract surgery. Other significant changes in visual acuity were not found.

Ocular Biometry

A median increase in ACD appeared in the phakic patients, +0.03 mm (min −0.01 mm/max 0.12 mm) in OD and +0.03 mm (min −0.02 mm/max 0.17 mm) in OS. Median power increase in corneal curvature was +0.08 D (min −0.20 D/max +0.60 D) in the OD and +0.08 D (min −0.30 D/max +0.60 D) in the OS. A median AL reduction occurred, −0.02 mm (min −0.05 mm/max 0.04 mm) and −0.01 mm (max −0.07 mm/max 0.05 mm) in OD and OS, respectively. The other ocular refractive parameters showed no statistically significant changes from the values measured before the HBO therapy. The vergence equation was used to calculate the impact of the total interaction of the changes in ocular refractive components (corneal power, ACD, AL of the eye) in the phakic patients. A median LP increase was found, +1.00 D (min +0.05 D/max +2.49 D) in the OD and +0.96 D in the OS (min +0.30 D/max +2.34 D).

The pseudophakic patients showed somewhat shallower anterior chambers after HBO therapy, median −0.01 mm (min −0.38 mm/

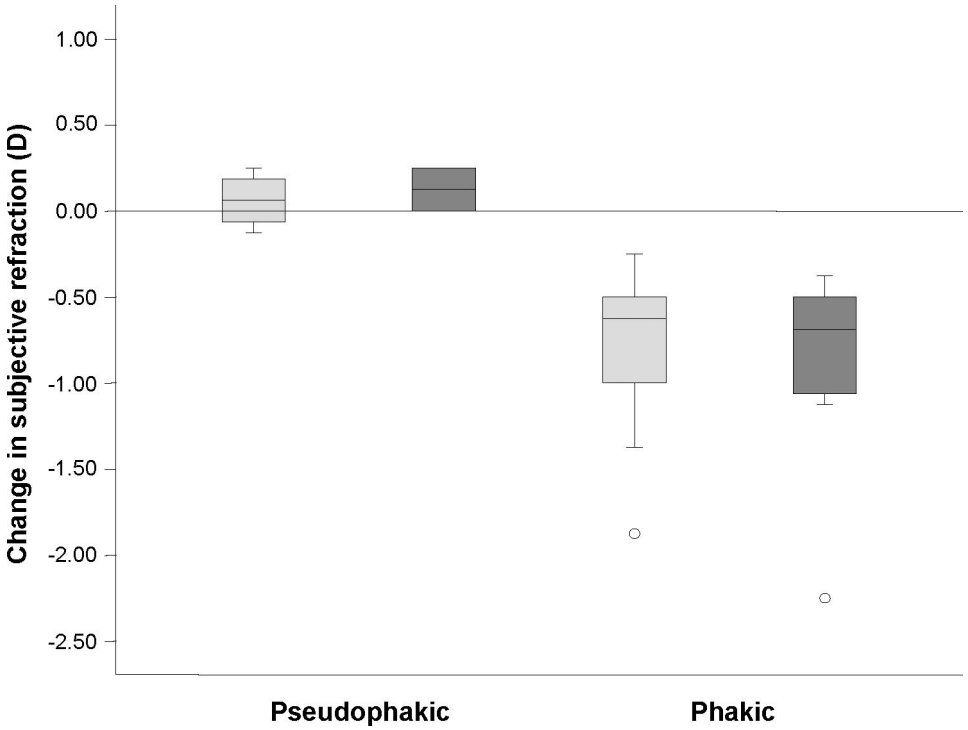

FIGURE 1.

Median subjective refractive changes in 16 phakic and six pseudophakic patients after HBO therapy. Light gray boxes show OD, solid gray boxes show OS, and error bars show maximum and minimum changes. Negative values indicate shift toward myopia, and positive values indicate shift toward hypermetropia.

TABLE 1.
Ocular biometric parameters in 16 phakic (upper panel) and six pseudophakic (lower panel) patients during HBO therapy

			Before HBO therapy (day 1)		After HBO therapy (day 19)		
Ocular biometric parameters	n	Eye	Median	Min/Max	Median	Min/Max	p[a]
Subjective spherical equivalent (D)	16	OD	+0.13	−3.00/+2.50	−0.50	−4.38/+1.50	<0.001
	16	OS	+0.00	−4.38/+2.50	−0.63	−6.63/+1.50	<0.001
Objective spherical equivalent (D)	16	OD	+0.31	−3.25/+3.13	−0.25	−4.38/+2.13	<0.001
	16	OS	+0.13	−4.25/+2.38	−0.38	−6.63/+1.50	<0.001
Keratometry (D)	16	OD	43.05	39.95/45.40	43.13	40.10/45.45	n.s.
	16	OS	43.03	40.00/45.50	43.18	40.15/45.90	0.022
Corneal radius curvature (mm)	16	OD	7.85	7.43/8.46	7.83	7.42/8.43	n.s.
	16	OS	7.83	7.42/8.44	7.82	7.36/8.40	n.s.
Corneal thickness (mm)	16	OD	0.554	0.520/0.593	0.554	0.516/0.584	n.s.
	16	OS	0.552	0.523/0.580	0.560	0.509/0.581	n.s.
Corneal volume (mm^3)	16	OD	58.85	53.40/63.50	58.70	52.30/63.80	n.s.
	16	OS	59.60	54.40/62.50	59.30	54.00/62.80	n.s.
ACD (mm)	16	OD	3.35	2.77/3.96	3.44	2.82/4.07	0.004
	16	OS	3.35	2.70/3.98	3.43	2.77/4.15	0.004
AL (mm)	16	OD	24.02	21.63/25.51	24.00	21.62/25.50	0.022
	16	OS	23.82	21.51/25.55	23.81	21.56/25.58	n.s.
Calculated total LP (D)	16	OD	+17.06	+15.58/+19.66	+17.94	+16.38/+22.15	<0.001
	16	OS	+17.16	+15.51/+20.84	+18.05	+16.29/+23.18	<0.001
IOP (mm Hg)	16	OD	14.67	11.33/21.33	15.00	10.00/20.33	n.s.
	16	OS	14.83	10.67/19.67	14.67	10.33/18.67	n.s.
Lens opacity	16	OD	15.83	11.33/27.00	16.17	12.00/27.00	n.s.
	16	OS	16.67	12.00/36.33	16.50	12.67/34.67	n.s.
Subjective spherical equivalent (D)	4	OD	−0.06	−0.25/+0.13	−0.06	−0.38/+0.25	n.s.
	6	OS	−0.06	−0.50/+0.63	+0.00	−0.25/+0.63	n.s.
Objective spherical equivalent (D)	4	OD	−0.13	−0.25/+0.13	−0.13	−0.38/+0.13	n.s.
	6	OS	+0.06	−0.50/+0.38	+0.00	−0.38/+0.38	n.s.
Keratometry (D)	4	OD	44.23	43.75/45.45	44.43	43.55/45.60	n.s.
	6	OS	43.88	43.00/45.40	43.88	43.05/45.70	n.s.
Corneal radius curvature (mm)	4	OD	7.63	7.42/7.72	7.60	7.40/7.75	n.s.
	6	OS	7.69	7.44/7.84	7.68	7.39/7.85	n.s.
Corneal thickness (mm)	4	OD	0.574	0.509/0.618	0.574	0.516/0.613	n.s.
	6	OS	0.566	0.509/0.611	0.569	0.521/0.615	n.s.
Corneal volume (mm^3)	4	OD	60.35	55.50/65.80	59.85	56.00/66.70	n.s.
	6	OS	62.00	54.80/65.60	60.70	54.00/66.70	n.s.
ACD (mm)	4	OD	5.44	4.46/5.86	5.25	4.44/5.87	n.s.
	6	OS	5.47	4.67/5.83	5.39	4.68/5.82	n.s.
AL (mm)	4	OD	22.92	22.49/23.60	22.91	22.47/23.62	n.s.
	6	OS	23.19	22.48/24.55	23.20	22.48/24.56	n.s.
IOP (mm Hg)	4	OD	12.00	11.67/13.33	11.67	9.67/14.33	n.s.
	6	OS	12.00	10.00/13.00	11.67	11.33/13.33	n.s.
Lens opacity	4	OD	7.67	7.00/8.00	7.50	6.67/8.33	n.s.
	6	OS	9.00	7.00/21.00	8.50	7.00/22.00	n.s.

[a]Wilcoxon signed-rank test.
Min, minimum; max, maximum; n.s., not significant.

TABLE 2.
Blood biochemical variables in 16 phakic patients before and after HBO therapy

Blood biochemical variables	n	Before HBO therapy (day 1)		After HBO therapy (day 19)		p[a]
		Median	Min/Max	Median	Min/Max	
B-hemoglobin ($g \cdot L^{-1}$)	16	149.0	120.0/169.0	140.0	107.0/164.0	0.021
S-natrium ($mmol \cdot L^{-1}$)	16	142.0	136.0/146.0	141.0	138.0/144.0	n.s.
S-kalium ($mmol \cdot L^{-1}$)	15	4.2	3.6/5.1	4.2	3.6/4.7	n.s.
S-chloride ($mmol \cdot L^{-1}$)	16	104.5	97.0/108.0	104.5	102.0/109.0	n.s.
S-calcium ($mmol \cdot L^{-1}$)	16	2.40	2.31/2.53	2.38	2.21/2.53	n.s.
S-phosphate ($mmol \cdot L^{-1}$)	16	1.06	0.69/2.00	1.13	0.78/1.37	n.s.
S-creatinine ($mmol \cdot L^{-1}$)	16	78.5	60.0/99.0	77.5	54.0/104.0	n.s.
S-albumine ($g \cdot L^{-1}$)	16	45.0	38.0/49.0	43.5	37.0/46.0	0.039
S-proteine ($g \cdot L^{-1}$)	14	71.5	57.0/88.0	70.0	58.0/73.0	n.s.
Hb-HbA1c (%)	16	5.7	5.2/6.3	5.6	5.3/6.2	n.s.
S-glucose ($mmol \cdot L^{-1}$), entering the chamber	16	6.2	4.6/8.4	6.1	5.1/10.1	n.s.
S-glucose ($mmol \cdot L^{-1}$), leaving the chamber	16	5.9	4.8/7.2	5.7	4.9/7.9	n.s.

[a]Wilcoxon signed-rank test.
Min, minimum; max, maximum; n.s., not significant; B, blood; S, serum; Hb, haemoglobin.

max 0.01 mm) in OD and −0.01 mm (min −0.19 mm/ max 0.00 mm) in OS, but no statistically significant changes were found in the pseudophakic eyes. There was no change in lens opacity measurements or IOP in neither of the two groups (Table 1).

Blood Samples

The biochemical variables measured in the phakic subjects were all within the normal reference intervals before and after the HBO therapy (Table 2). There were no changes in serum glucose or HbAc1 and no changes in serum electrolytes. Statistically significant differences in serum albumin were measured, but the median change was only 3% ($p = 0.039$). There was a reduction in the concentration of hemoglobin of 6% ($p = 0.021$).

DISCUSSION

Temporary refractive changes toward increased myopia or reduced hypermetropia are frequently reported as a potential ocular side effect in patients after prolonged periods of daily HBO therapy.[2–8] The most significant finding in this study is the lack of refractive changes in the pesudophakic eyes, which compared to the well-known myopic shift in the phakic eyes, strongly indicates that the refractive changes take place in the crystalline lens. All the phakic eyes were measured with refractive changes toward myopia (range, −0.25 to −2.25 D) after HBO therapy. A significant myopic shift (≥0.50 D) occurred in 26 (81%) single phakic eyes, which correspond well with the observation of HBO-treated patients given oxygen by hood in the multiplace chamber.[8] There was no evidence that the myopic shift was related to age, gender, myopic, or hypermetropic refractive error of the eyes before the treatment.

The four most influential ocular biometric parameters to produce a myopic shift are increased corneal refractive power, shorter ACD, longer AL of the eye, or increased crystalline LP. Measuring techniques with high precision were used to detect reliable changes. The Pentacam device collects true curvature data of the entire transparent cornea including the central area. Taking 50 three-dimensional image slices with the same center point, the normal attentive fixation saccadic eye movements do not affect the precision of the result. The IOLMaster measures the AL with a non-contact technique, avoiding the risk of corneal indentation and providing more reproducible results compared with those obtained with ultrasound (A-scan).[15]

There were no significant differences in corneal thickness and volume, and no increase in corneal refractive power large enough to explain the refractive changes. The anterior chamber appeared to be somewhat deeper after the therapy and the AL of the eye tended toward being shorter. The latter changes affect the refraction in the hypermetropic rather than the myopic direction. Sufficient changes in ocular refractive parameters were not measured to verify the amount of refractive changes in phakic patients. Second, the inclusions of pseudophakic eyes, which show no significant refractive changes, strongly indicate that the origin of the myopic shift has to be found in the crystalline lens. HBO exposure may cause changes in the lens, which further lead to an increase in the lens refractive index and a subsequent myopic shift.

With ages between 47 and 77 years, the phakic patients were all presbyopic and required a reading prescription for near-work. The amount of the additional reading power has been shown to stay stable during HBO therapy for presbyopic patients.[10] Probably little or no accommodative power would be left in these patients to cause any pseudomyopia. Despite this, the standard endpoint of highest plus or least minus accepted for the best-corrected visual acuity at distance was performed to avoid an accommodative tonus bias.

Undiagnosed diabetics may complain of transient changes in vision. Presumably, this may be caused by shifts in crystalline lens hydration related to osmotic changes associated with variations in blood glucose levels.[11,12] There was no indication of any changes in glucose metabolism or serum osmolarity in the phakic patients. The reduction in hemoglobin concentration has been demonstrated in other studies as well and has been attributed to a reduction in erythropoietin concentration because of the high partial pressure of oxygen.[23]

The patients recognized no change in vision within the first week of the HBO therapy. After completing 3 weeks of treatment, most of the phakic patients had noticed change in visual quality. Because each daily exposure was of similar oxygen partial pressure and length of time, the visual side effect of HBO exposure is supposed to be a cumulative process. Oxidative stress has been proposed to cause an aggregation of proteins in the lens nucleus with an implication of increased refractive index and a consequent myopic shift.[24,25] Development of cataract has been detected in patients after extended periods of HBO exposure.[4] Cataract formation in a patient after a standard course of HBO therapy has recently been reported.[26] Observations have suggested that the optical properties of the crystalline lens are sensitive to cumulative oxygen exposure,[27] and that changes in lenticular refraction may take place long before any opacity can be seen on gross observation in the lens.[27] An opacification or loss of clarity in the nucleus may follow after a change in the cortical refractive index, which would require only very slight changes in the gradient sufficient to alter the refractive power to a measurable degree.[28]

Myopic shifts occurred in the phakic eyes but not in the pseudophakic eyes during the HBO therapy. This finding, along with no changes in corneal power, ACD, AL, glucose metabolism, or serum osmolarity strongly, suggests that the myopic shift seen in patients treated with HBO is attributed to changes in the crystalline lens.

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