Peripheral Refraction and Ocular Shape in Children

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Purpose. To evaluate the relation between ocular shape and refractive error in children.

Methods. Ocular shape was assessed by measuring relative peripheral refractive error (the difference between the spherical equivalent cycloplegic autorefraction 30° in the nasal visual field and in primary gaze) for the right eye of 822 children aged 5 to 14 years participating in the Orinda Longitudinal Study of Myopia in 1995. Axial ocular dimensions were measured by A-scan ultrasonography, crystalline lens radii of curvature by videophakometry, and corneal power by videokeratography.

Results. Myopic children had greater relative hyperopia in the periphery (+0.80 ± 1.29 D), indicating a prolate ocular shape (longer axial length than equatorial diameter), compared with relative peripheral myopia and an oblate shape (broader equatorial diameter than axial length) for emmetropes (−0.41 ± 0.75 D) and hyperopes (−1.09 ± 1.02 D). Relative peripheral hyperopia was associated with myopic ocular component characteristics: deeper anterior and vitreous chambers, flatter crystalline lenses that were smaller in volume, and steeper corneas. Lens thickness had a more complex association. Relative peripheral hyperopia was associated with thinner lenses between refractive error groups but changed in sign to become associated with thicker lenses when analyzed within each refractive error group. Receiver operator characteristics analysis of the ocular components indicated that vitreous chamber depth was the most important ocular component for characterizing the myopic eye, but that peripheral refraction made a significant independent contribution.

Conclusions. The eyes of myopic children were both elongated and distorted into a prolate shape. Thinner crystalline lenses were associated with more hyperopic relative peripheral refractions across refractive error groups, but failure of the lens to thin may account for the association between thicker lenses and more hyperopic relative peripheral refractions within a given refractive group. Increased ciliary–choroidal tension is proposed as a potential cause of ocular distortion in myopic eyes. (*Invest Ophthalmol Vis Sci.* 2000;41:1022–1030)

Myopia occurs when the length of the eye, particularly the vitreous chamber, exceeds the focal length of the optical components that contribute to the refracting power of the eye. The cause of this excessive ocular growth has been the subject of much research and debate.[1] Ocular growth in infancy tends to produce beneficial changes in refractive error—namely, emmetropization or a reduction in the amount and variability of the hyperopia typically seen in infants.[2–4] The eye continues to grow between infancy and childhood, but the majority of children remain either emmetropic or exhibit low amounts of hyperopia. By age 6 years, only 2% of children are myopic.[5] Later in childhood, ocular growth may become excessive and produce myopia, so that 15% of children become myopic by age 15 years.[5] To maintain emmetropia there must be corresponding changes in the optical components (the cornea and/or crystalline lens) during the growth of the eye. Although the cornea is the most powerful dioptric component of the eye, it completes its relatively small amount of change early in life. Therefore, it has limited potential to produce and maintain emmetropia over time. It decreases in power by only 3 to 5 D to nearly adult values by the age of 2 years,[6–9] then becomes relatively stable throughout childhood.[10,11] In contrast, the crystalline lens equivalent power decreases by some 20 D by the age of 6 years,[12,13] with changes in lens radii continuing throughout childhood[10,13,14] and decreases in equivalent power up to age 10 years.[13,15] Mechanisms for how ocular growth and crystalline lens development might interact to produce emmetropia or myopia have not been elucidated. Active emmetropization models of refractive development generally do not include a role for the crystalline lens, but rather postulate that the growth of the eye is modulated by a visual feedback response to the defocus produced by the eye's refractive state.[16,17] An alternative theory of passive emmetropization proposes that compensatory changes in lens power may occur as the eye grows and expands through the anatomic connection between the globe and the crystalline lens.[18–22]

We recently reported that the ability of the lens to respond to growth in the equatorial plane of the globe may be a source of interaction with both the overall size and shape of the developing eyes of children.[13] Before ages 9 to 10 years, when the prevalence of myopia is 5% or less, the crystalline lens displays optical and structural changes that are consistent

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with its role in compensating for ocular growth. It flattens in curvature, thins axially, decreases in equivalent refractive index, and decreases in equivalent power. As the eye continues to grow after the age of 10 years, the crystalline lens continues to flatten, but it no longer displays the previous pattern of compensation for growth. It stops thinning, the equivalent refractive index stops decreasing and begins to increase, and the average equivalent power stays constant. In our sample, the prevalence of myopia also increases at this age to reach 21% by age 14 years. We proposed that ocular growth in the equatorial plane stretches the crystalline lens in early childhood to produce thinning, flattening, and compensatory changes in lens power to maintain emmetropia within the growing eye, whereas restriction of equatorial expansion in later childhood interferes with the lens thinning and power decreases compensating for ocular growth. As an added myopigenic factor, equatorial restriction of the growing eye has the potential to accentuate axial elongation.[13]

The shape of the eye may therefore be an important indicator of this equatorial restriction. Ocular shape is associated with refractive error in adult eyes, with myopic eyes tending to be prolate (having a longer axial length than equatorial diameter), and hyperopic eyes tending to be oblate (having a broader equatorial diameter than axial length). This shape difference has been measured both physically and optically. Deller et al.[23] measured refractive error and the axial, transverse, and vertical diameters of 45 eyes by radiograph. Departure from a spherical shape was defined as an "index" different from 1:

$$\text{index} = (\text{transverse diameter} \cdot \text{equatorial diameter}) / (\text{axial length}^2).$$

Myopes had a value less than one, indicating more elongation in the axial than in the equatorial direction (0.90 ± 0.08). Shapes for emmetropes and hyperopes were nearly spherical at 1.00 ± 0.09 and 1.01 ± 0.04, respectively. Data from computed tomography in 131 subjects yield similar ratios for the horizontal transverse–anteroposterior axis (myopes 0.94, emmetropes 1.01, hyperopes 1.04).[24]

Differences in ocular shape can also be measured optically as the relative change in the spherical equivalent refractive error with movement from an axial to a peripheral angle of measurement. This was used by Feree et al.,[25] who identified three patterns for peripheral refraction in 21 eyes of adults using a refractometer (Carl Zeiss, Oberkochen, Germany): type B, when both sagittal and tangential foci become more hyperopic in the periphery; type C, when there is asymmetry between the refraction in the temporal and nasal halves of the visual field; and type A, when the sagittal focus becomes more hyperopic and the tangential focus more myopic in the periphery. Rempt et al.[26] called these patterns I, III, and IV, respectively, and added two more classes from their investigation of 442 adults by retinoscopy. Coining the term "skiagram" for these patterns, they described the type II skiagram in which the sagittal focus becomes more hyperopic in the periphery, whereas the tangential remains the same, and the type V in which the inverse occurs: The sagittal focus remains the same, whereas the tangential focus becomes more myopic.[26] Retinoscopic studies have shown an association between peripheral refraction and refractive error consistent with radiographic and tomographic studies. Myopes tend to have relative peripheral hyperopia (a larger axial length compared with equatorial diameter), whereas hyperopes either have relative peripheral myopia (a larger equatorial diameter compared with axial length) or resemble emmetropes who most often show little difference between the central and peripheral spherical equivalent refractive error (a nearly spherical eye).[26,27]

The validity of peripheral refraction as a measure of ocular shape has been evaluated in computer simulations, with spherical equivalent peripheral refractive error yielding valid retinal coordinates (within 0.50 D or 0.20 mm) for field angles up to 40°. It appears to be robust to variation in gradient index profile, lens shape, corneal toricity, and lens tilt.[28] Logan et al.[29] have evaluated retinal contour in a case of myopic anisometropia using instrumentation similar to that used in the present study, finding that measurement of the subject's prolate retinal contour using an autorefractor (R-1; Canon, Lake Success, NY; no longer manufactured) was repeatable.[29] Because our model suggests that equatorial restriction and distortion may be involved in the development of myopia, we evaluated the importance of ocular shape measured by peripheral refraction as a characteristic of refractive error in children.

Methods

Subjects were 827 children participating in 1995 in the Orinda Longitudinal Study of Myopia (OLSM), a cohort study of ocular component development and risk factors for the onset of myopia. Peripheral refraction data were obtained from 822 of these children due to a temporary mechanical problem with the autorefractor. Data from 820 children were used in logistic regression analyses, because ultrasonography could not be performed in an additional two subjects. Parents gave consent for their children's participation after all study procedures were explained in accordance with the Declaration of Helsinki. This sample was 47.9% female and predominantly white (87.3%, with 11.2% Asian-American, 1% African-American, and 0.5% Hispanic). The participation rate for girls varied ran-

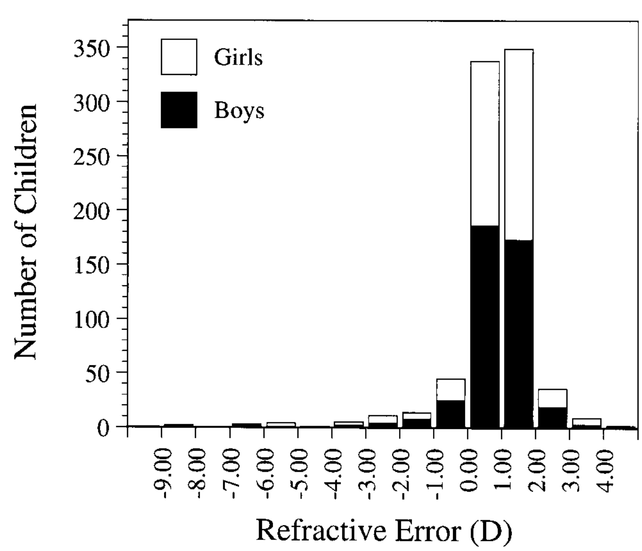

Figure 1. Distribution of refractive errors in the vertical meridian by gender for children in the study.

Table 1. Number of Subjects According to Age

	Age at Last Birthday							
	≤6	7	8	9	10	11	12	≥13
Subjects (*n*)	115	122	117	126	102	90	68	87
Percentage of total	13.9	14.8	14.1	15.2	12.3	10.9	8.2	10.5

Age in years. $N = 827$.

domly by age group, with a maximum of 57.4% at age 6 years and a minimum of 35.6% at age 11 years.

Refractive error in the vertical meridian ranged from −9.62 D to +4.10 D within a leptokurtic distribution skewed toward myopia typical for children of this age (Fig. 1). Age at last birthday ranged from 5 to 14 years (first through eighth grade). Children in the eighth grade were recruited into the study in the third grade in 1990. Children in all other grades were recruited initially as first graders. The number of subjects by age at last birthday is shown in Table 1, Myopes in this report had at least −0.75 D or more myopia in each principal meridian (7.1%) and hyperopes at least +1.00 D or more hyperopia in each principal meridian (9.4%). Emmetropes represent the remainder of the sample (83.6%).

Peripheral refraction was added to the OLSM protocol in 1995. This report presents this first year's cross-sectional results. Peripheral refraction was measured on the right eye of subjects by the R-1. Subjects were tested under cycloplegia 30 minutes after 1 drop proparacaine 0.5% and 2 drops tropicamide 1%. The R-1 was reported to need a minimum pupil size of 2.90 mm.[30] At 30° this requires an entrance pupil of 3.35 mm (2.90 mm/cos 30°). Mydriasis was adequate in all cases for measurement of peripheral refraction. Subjects first fixated a reduced Snellen target through a +4.00-D Badal lens in primary gaze. The target was placed on a track allowing for the adjustment of target distance to relax accommodation yet provide maximum clarity to hyperopic and myopic subjects. Ten autorefractor measurements were made according to our standard protocol for cycloplegic autorefraction.[31] Immediately after measurement in primary gaze, the track holding the Snellen target was rotated 30° and placed before a front surface mirror on the patient's right (Fig. 2). Subjects were instructed to turn their eyes and to look into the mirror to find the backward letters. Once the subject responded that the letters were backward, and the eye was observed to be steady in the television monitor of the R-1, five autorefraction measurements were recorded. Ten readings in primary gaze may be an excessive number, because the average spherical equivalent of the first five and last five readings differ by only 0.016 D. We therefore chose to take only five readings in peripheral gaze, but we continued with 10 primary gaze readings because we were reluctant to change our long-standing protocol for cycloplegic autorefraction.

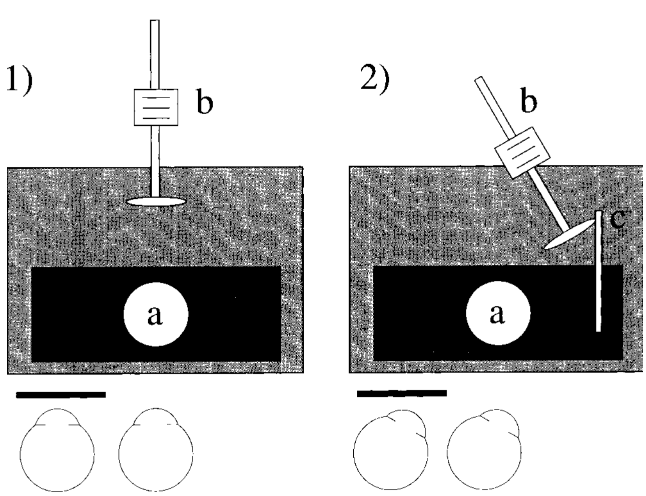

Figure 2. Diagram of the experimental apparatus viewed from above. 1a and 2a are the objective lens of the Canon R-1 autorefractor; 1b and 2b are the Badal track with letter target; 2c is the front surface mirror.

All measurements were later averaged by the method of Harris.[32] Although this method is not necessary to obtain an average spherical equivalent, it provides a valid average sphere and cylinder. In this report, peripheral refraction refers to the refractive error at 30°, without reference to refractive error in primary gaze. Ocular shape is inferred from the relative peripheral refractive error, where the spherical equivalent of the average refraction in primary gaze is subtracted from the spherical equivalent of the average refractive error in 30° temporal gaze (i.e., the autorefractor axis directed 30° in the nasal visual field of the subject's right eye).

Ocular components were measured according to previously published OLSM protocols.[31] Corneal topography was assessed using a Computed Anatomy TMS-1 (Tomey, Waltham, MA) before cycloplegia.[11] Lens radii of curvature were measured by videophakometry as described in detail elsewhere.[33] Axial ocular dimensions were measured by A-scan ultrasonography (Model 820, Humphrey Instruments, San Leandro, CA), consisting of five readings using a handheld probe in semiautomatic mode. Statistical analyses were performed using commercial software and general linear model (GLM) procedures (SAS Institute, Cary, NC). This statistical package computes simple and multiple regression, as well as analysis of variance, depending on the type of variables in the analysis. S-Plus (MathSoft, Seattle, WA) was used for logistic models.[34]

Results

On average, subjects with myopia measured in primary gaze also had myopic peripheral spherical values, whereas subjects who were hyperopic centrally were hyperopic in the periphery. Emmetropes were also hyperopic in the periphery, but less so than hyperopes (Table 2; GLM least-squares means comparison; all differences significant, $P < 0.0001$). Because of the difference between peripheral sphere values, peripheral spherical equivalents were also significantly more myopic among myopes and more hyperopic among hyperopes; em-

Table 2. Refractive Errors According to Refractive Group

Refractive Category	Primary Gaze Spherical Equivalent	Peripheral Sphere	Peripheral Cylinder	Peripheral Spherical Equivalent
Myopes	−2.84 ± 2.09	−0.61 ± 1.88*	−2.81 ± 0.82	−2.04 ± 1.82*
Emmetropes	+0.44 ± 0.45	+1.47 ± 0.64*	−2.84 ± 0.85	+0.03 ± 0.79*
Hyperopes	+1.81 ± 0.74	+2.24 ± 0.93*	−2.97 ± 0.98	+0.72 ± 1.09*

Data are mean diopters ± SD measured in primary gaze and 30° in the nasal visual field. Differences between peripheral cylinders are not significant ($P \leq 0.76$).

* Differences are significant ($P < 0.0001$).

metropes maintained an emmetropic absolute spherical equivalent in the periphery (GLM least-squares means comparison; all differences significant, $P < 0.0001$). The SDs for the central and peripheral spherical equivalent in Table 2 were higher among myopes, most likely because myopia encompasses a broader range of refractive errors than the other disorders. We found no significant differences in the amount of cylinder at 30° as a function of refractive group (GLM least-squares means comparison, $P \leq 0.76$), consistent with results in one study,[27] but contrary to previous findings of lower amounts of peripheral cylinder in myopic adults.[26]

Regarding relative peripheral refraction, the raw, unadjusted averages (±SD) by refractive category indicated that myopic children had relative hyperopia in the periphery (+0.80 ± 1.29 D) compared with emmetropes (−0.41 ± 0.75 D), indicating a prolate ocular shape in myopia (Fig. 3). Hyperopes had greater relative peripheral myopia (−1.09 ± 1.02 D) and therefore a more oblate ocular shape (Tukey's Studentized range test; all comparisons significantly different, $P < 0.05$). Variability in relative peripheral refraction also differed by refractive group with myopes having the highest SD, hyperopes an intermediate value, and emmetropes the lowest SD (F test for equal variance; all comparisons significantly different, $P < 0.028$). Unlike the absolute peripheral spherical equivalents in Table 2, this difference in variability is not a simple function of range.

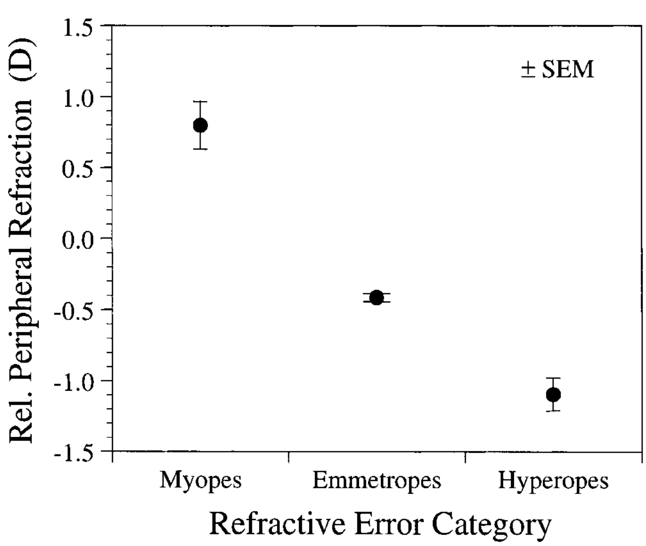

Figure 3. Raw, unadjusted means (±SEM) for relative peripheral refraction by refractive error category.

The raw, unadjusted average (±SD) values for relative peripheral refraction by age at last birthday showed the youngest subjects at age 6 years had the most relative peripheral myopia, which became less myopic with age (Fig. 4A). Multiple comparisons indicated that the relative peripheral refractions at ages 8 through 13 or more years were significantly less myopic than that at age 6 years or less (Tukey's Studentized range test, $P < 0.05$,). Because relative peripheral refraction was related to refractive error and refractive error changed with age (Fig. 5), we adjusted values for relative peripheral refraction for age and refractive category. Age-adjusted values for relative peripheral refraction were nearly unchanged and still significantly different from each other (+0.78 D, −0.42 D, and −1.06 D for myopes, emmetropes, and hyperopes respectively; GLM least-squares means comparison, $P < 0.0001$). Adjustment for refractive category disturbed the previous pattern of change with age (Fig. 4B). Relative peripheral refraction at age 8 years was significantly less myopic than at age 6 years or less, 7 years, and 11 years (GLM least-squares means comparison, $P < 0.015$). Perhaps as expected, most ages had refractive error-adjusted values similar to those for emmetropes. Age-related changes in the unadjusted relative peripheral refractions appeared to be due in large part to age-related changes in refractive error.

Girls had a less myopic relative peripheral refraction than boys, after adjustment for age, refractive error category, and ocular component values (−0.19 D compared with −0.39 D for boys, GLM least-squares means comparison, $P < 0.0004$). This may indicate the tendency of girls to become myopic earlier than boys and therefore to have a higher prevalence and average amount of myopia. Girls had the higher prevalence of myopia in five of seven age groups (none had myopia at age 6 years) and the higher average refractive error among myopes in six of seven. For example, at age 12 years, 21% of girls had myopia averaging −3.37 D compared with 12% of boys averaging −3.10 D.

To explore the relation between peripheral refraction and the ocular component values that comprise refractive error, we modeled relative peripheral refraction as a function of vitreous chamber depth, anterior chamber depth, lens thickness, lens spherical volume, Gullstrand lens power, and corneal power as independent variables in a model adjusted for age at last birthday, gender, and refractive error category (Table 3). Variables such as crystalline lens equivalent power and equivalent refractive index, which are mathematically derived from refractive error and ocular components, were excluded because of their potentially artificially inflated correlations with the ocular components.

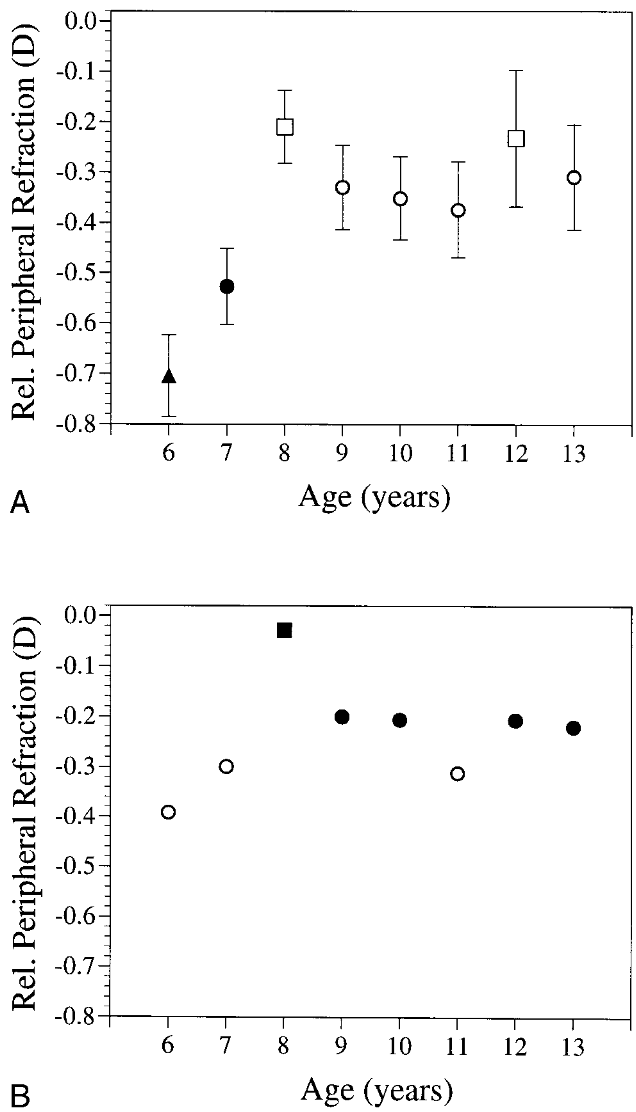

FIGURE 4. (**A**) Raw, unadjusted mean values (±SEM) for relative peripheral refraction by age at last birthday. Ages indicated by *open symbols* are all significantly different from the value at age 6 years or less (▲), and ages indicated by *open squares* (8 and 12 years) are both significantly different from the value at age 7 (●). (**B**) Least-squares mean values for relative peripheral refraction by age at last birthday adjusted for refractive error category. Ages indicated by *open circles* are all significantly different from the value at age 8 (■). In (**A**) and (**B**) ages indicated by *filled symbols* are not significantly different from each other, and ages indicated by *circles* are not significantly different from each other.

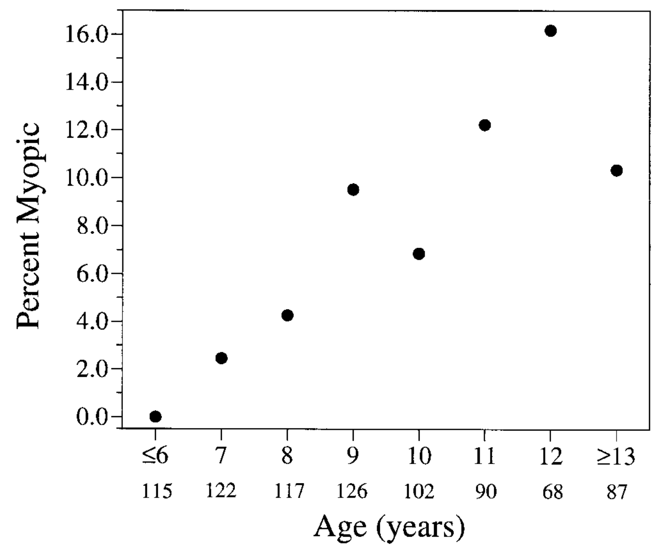

FIGURE 5. The proportion of myopic children as a function of age for subjects in the study. The number of observations per age group is given beneath subject age.

Each of the ocular components measured was significantly correlated with relative peripheral refraction in the multiple regression model (Table 3). The ocular components displayed correlations with peripheral refraction that are consistent with the association between more hyperopic peripheral refractions and more myopic refractive errors. Myopia is associated with longer anterior and vitreous chambers,[35,36] flatter lens shapes,[37] steeper corneas,[35,38] and relative peripheral hyperopia. More hyperopic peripheral refractions also occurred when the lens had a smaller spherical volume, a finding that is consistent with our proposed model that an insufficient amount of lens material may contribute to its inability to stretch equatorially as the eye grows. The positive sign for lens thickness indicates that thicker, rather than thinner, lenses were associated with more hyperopic peripheral refractions *within* each refractive group. This is in contrast to the association between thinner crystalline lenses and more myopic refractive errors *between* refractive groups.[39] This same pattern can be seen in relative peripheral refraction. Increasingly, hyperopic relative peripheral refractions were associated with thinner lenses *between* refractive groups. This is indicated by the significant negative coefficient in a univariate regression of peripheral refraction as a function of lens thickness (−0.65, GLM, $P < 0.0009$) when refractive group is left out of the model. There was no significant interaction between lens thickness and refractive group in this model.

To rank peripheral refraction and the ocular components for their importance as characteristics of the myopic eye, we conducted a series of logistic regressions of the log odds of being myopic as a function of these ocular components. It should be noted that these data were obtained from prevalent myopes and are presented for the purpose of characterizing the myopic eye. Model sensitivities and specificities should not be interpreted as the performance of these variables as risk factors for predicting the onset of

TABLE 3. Multiple Regression Coefficients for Relative Peripheral Refraction

Ocular Component	Multiple Regression Coefficient	*P*
Vitreous chamber depth (mm)	0.32	0.0001
Anterior chamber depth (mm)	1.46	0.0001
Lens thickness (mm)	4.82	0.0006
Lens spherical volume (mm^3)	−0.055	0.0029
Gullstrand lens power (D)	−0.41	0.0054
Corneal power (D)	0.19	0.0001

Model contains age, gender, refractive group, and the ocular components listed.

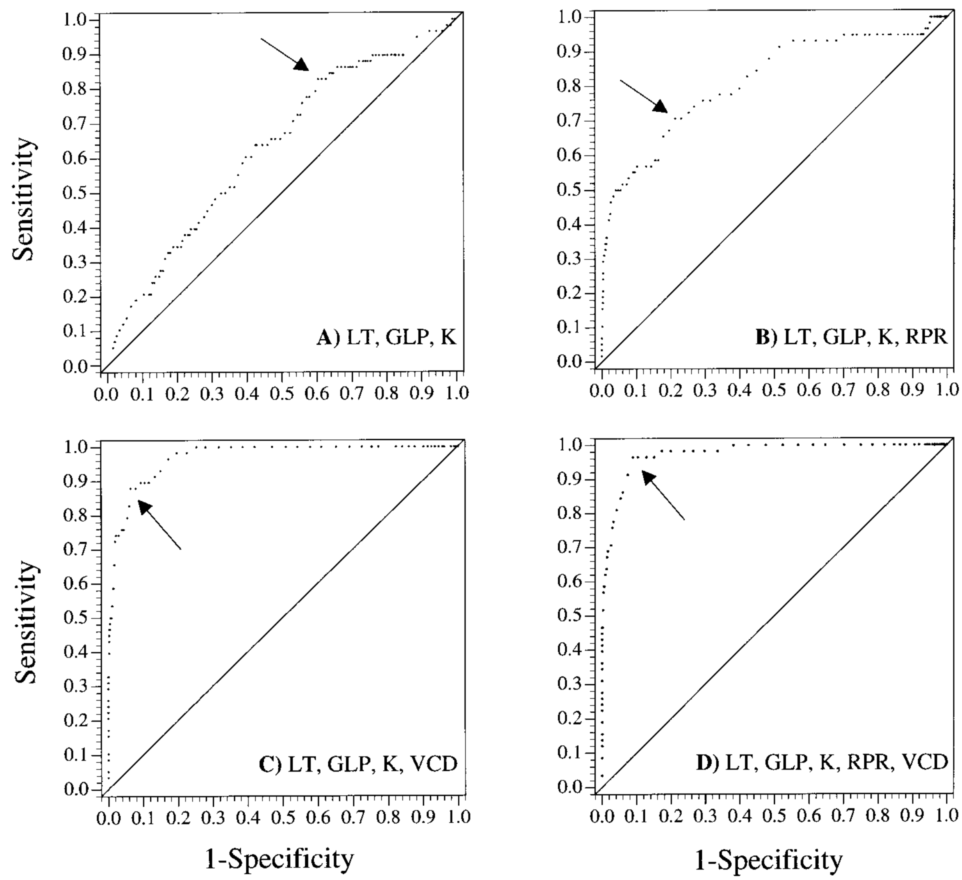

Figure 6. Receiver operator characteristics (ROC) curves for four models using ocular components to characterize myopic eyes. **(A)** Contains lens thickness, Gullstrand lens power, and corneal power; **(B)** contains components listed for **(A)** plus relative peripheral refraction; **(C)** contains components listed in **(A)** plus vitreous chamber depth; and **(D)** contains components listed in **(A)** plus relative peripheral refraction and vitreous chamber depth. *Arrows* indicate the point of maximum discrimination. Data shown in Table 4.

myopia. Such an analysis would require prospective, longitudinal data. Age, gender, anterior chamber depth, and lens spherical volume were not significant parameters in this logistic model. Vitreous chamber depth (considered the gold standard characteristic of the myopic eye), relative peripheral refraction, lens thickness, Gullstrand lens power, and corneal power remained significant. A logistic model containing lens thickness, Gullstrand lens power, and corneal power was then used as a baseline from which to judge the relative contributions of vitreous chamber depth and relative peripheral refraction.

Receiver operator characteristics (ROC) analysis applies the logistic model to individual subject data and plots the sensitivity and 1 − specificity for classifying subjects as myopic or not across the range of values for the model. The ROC curve associated with this baseline model is presented in Figure 6A. At the point of maximum discrimination, these features were only moderately successful in distinguishing the myope from the nonmyope, displaying good sensitivity but poor specificity (0.83 and 0.40, respectively; Table 4, model A). The addition of relative peripheral refraction to the baseline logistic model was a significant improvement ($\chi^2 = 90.5$, $df = 1$, $P < 0.0001$). The ROC curve associated with that model is presented in Figure 6B. The addition of relative peripheral refraction decreased the sensitivity but increased the specificity compared with the baseline model (0.71 and 0.79, respectively; Table 4, model B). The addition of vitreous chamber depth to the baseline logistic model was also a significant improvement ($\chi^2 = 235.8$, $df = 1$, $P < 0.0001$). The ROC curve associated with that model is presented in Figure 6C. Vitreous chamber depth added to the sensitivity and the specificity of the baseline model (0.88 and 0.94 respectively; Table 4, model C). Despite the association between vitreous chamber depth and peripheral refraction (Table 3), the best model included the baseline components and both vitreous chamber depth and relative peripheral refraction (comparing full model to baseline plus vitreous chamber depth model yields $\chi^2 = 26.0$, $df = 1$, $P < 0.0001$; comparing full model to baseline plus relative peripheral refraction model yields $\chi^2 = 171.2$, $df = 1$, $P < 0.0001$). The ROC curve associated with that model is presented in Figure 6D. Sensitivity and specificity were the highest with both vitreous chamber depth and relative peripheral refraction in the model (0.97 and 0.91 respectively; Table 4, model D). An enlarged vitreous chamber depth appeared to be the strongest characteristic of the myopic eye, but both shape (prolate) and size (elongated) were needed to best characterize the myopic eye.

Table 4. Sensitivity and Specificity at the Point of Maximum Discrimination for Four Models Characterizing Myopic Eyes by Ocular Component

	Measured as Myopic	Measured as Nonmyopic
Model A		
Myopic by logistic model (n)	48	461
Nonmyopic by logistic model (n)	10	301
Sensitivity (95% CI)	0.83 (0.71–0.91)	
Specificity (95% CI)		0.40 (0.36–0.43)
Model B		
Myopic by logistic model (n)	41	162
Nonmyopic by logistic model (n)	17	600
Sensitivity (95% CI)	0.71 (0.57–0.82)	
Specificity (95% CI)		0.79 (0.76–0.82)
Model C		
Myopic by logistic model (n)	51	49
Nonmyopic by logistic model (n)	7	713
Sensitivity (95% CI)	0.88 (0.77–0.95)	
Specificity (95% CI)		0.94 (0.92–0.95)
Model D		
Myopic by logistic model (n)	56	68
Nonmyopic by logistic model (n)	2	694
Sensitivity (95% CI)	0.97 (0.88–1.00)	
Specificity (95% CI)		0.91 (0.89–0.93)

Model A contains lens thickness, Gullstrand lens power, and corneal power. Models B, C, and D contain the same components plus relative peripheral refraction (model B), vitreous chamber depth (model C), and relative peripheral refraction plus vitreous chamber depth (model D). Data coincide with arrows in Figure 6A, 6B, 6C, and 6D, respectively.

Discussion

The shape of the eye determined by peripheral refraction is an important factor associated with refractive error in children. The relative peripheral myopia in hyperopic eyes indicated an oblate shape, with a longer equatorial than axial diameter. Emmetropic eyes were closer to spherical and slightly oblate in shape. The relative hyperopia in myopic eyes indicated a prolate shape with a longer axial than equatorial diameter. Along with an elongated vitreous chamber depth, a prolate ocular shape made an independent contribution to the characterization of the myopic eye.

One potential limitation of this study is that only one point is sampled in the periphery. We feel justified in making inferences about ocular shape using only one point based on two previous studies in which multiple points at various field angles, both nasal and temporal, were measured.[26,27] Both studies found that the difference in relative refractive error increases monotonically with field angle from the fovea and that there was a high degree of symmetry between the two hemifields for most eyes. Specifically, Rempt et al.[26] found that only 3.2% of 442 subjects had a significant nasal–temporal asymmetry in their peripheral refractions (type III). Millodot[27] noted an asymmetry in the amount of peripheral astigmatism, with the nasal visual field (temporal retina) having the larger amount. This difference was only significant, however, for field angles beyond 30°. Nasal–temporal asymmetry has been reported in 3 of 18 subjects with anisomyopia,[29] but this is a rare refractive condition. Therefore, distortion in ocular shape should be detectable at the one angle used in the present study.

The power to detect change should be greatest at the most extreme field angles. Our choice of 30° represents a compromise between a field angle that is feasible to measure with the Canon R-1 autorefractor and an angle sufficiently large to find significant differences if they exist. Sampling larger field angles approaching 60° would have the advantage of measuring the eye near the equator. However, a previous study[27] and subsequent analysis[40] have suggested that the absolute peripheral spherical equivalent of different refractive groups tends to converge at extreme field angles, implying that the equator of the eye has a similar diameter across refractive error groups. As shown in Table 2, the absolute peripheral spherical equivalents at 30° indicate that myopes have the largest, emmetropes the intermediate, and hyperopes the smallest eyes at the less extreme angle used in the present study.

Ocular component relations with relative peripheral refraction were consistent with previous reports of component relations with myopia: longer vitreous chambers, deeper anterior chambers,[35,36] flatter crystalline lenses,[37] and steeper corneas.[35,38] The positive relation between relative peripheral refraction and lens thickness deserves special attention, because it indicates that thicker, rather than thinner, lenses were associated with more hyperopic relative peripheral refractions, and therefore more myopic refractive errors. This was a counterintuitive finding, in that we have reported that thin lenses are associated with myopia in children.[39] A more complete analysis indicated that thin lenses were indeed associated with more hyperopic peripheral refractions but only *between* refractive error groups, not *within* a given refractive error group.

Having a thick lens may simply be associated with a more prolate shape within a refractive group as a normal characteristic. The disconnection between the behavior of lens thickness and Gullstrand lens power (lens shape) in their relation with relative peripheral refraction suggests that something more complex may be occurring. Gullstrand lens power has a negative association with relative peripheral refraction, whether refractive group is in the model (Table 3) or not (regression coefficient = -0.09, univariate GLM, $P < 0.0001$). This indicates that the lens continually flattens as the eye grows and assumes a prolate shape. If thinning, flattening, and decreasing power of the crystalline lens were the simple response to the expanding equator, then all three should continue changing as the eye grows and expands. This may occur for lens shape, but lens thickness and power do not behave in this manner. After age 10 years, lens flattening disconnects from thinning and power changes; the lens stops thinning and decreasing in power even as the eye continues to grow and the lens to flatten.[13] We propose that failure of the lens to thin may be causing the eye to distort and that this accounts for the positive association between greater lens thickness and more hyperopic relative peripheral refractions *within* refractive groups. Determining whether distortion is due to the initial thickness of the lens or to its having thinned to a point that exceeds a critical limit will require longitudinal data. It is interesting to note that lens thickness in third grade (mean age, 8.6 years) is not a significant predictor of future myopia, indicating that initial thickness may not be an important determinant of final refractive error.[37] Such analyses of how ocular component development interacts with ocular shape may also help to explain the greater variability in ocular shape among myopes. Perhaps various subtypes of myopia may be found: those associated with distortion and those in which shape is maintained. Again, longitudinal analyses are needed to evaluate crystalline lens development and changes in relative peripheral refraction more fully as risk factors for myopia.

Other mechanisms may explain why the eye may be distorted into a prolate shape in myopia. Slow equatorial expansion may be due to forces external to the eye, such as the extraocular muscles, as suggested by the rabbit model of myopia.[41] The bony orbit itself may limit the equatorial size of the eye. There may be a difference in the maturation rate of the equatorial sclera compared with the axial sclera. If the equatorial sclera of the human eye stopped growing before the axial sclera, then processes related to equatorial growth may also stop before the end of axial growth. These could include lens stretch and stretch-related changes in lens parameters. Thicker lenses and more prolate shapes within refractive error groups would merely indicate that the eye has stopped growing equatorially due to one of these external causes. However, if the source of the equatorial restriction is internal and is due to the failure of the crystalline lens to thin, then the increased tension on the lens might be expected to have an impact on accommodation. It should become more difficult, creating an increase in accommodative lag and the accommodative convergence/accommodation (AC/A) ratio. This is consistent with the deficient accommodative responses of myopes[42] and recent analyses showing an increased AC/A ratio in juvenile myopia.[43,44] It is possible that the distortion of the globe may be an independent anatomic consequence of the larger eye sizes that produce myopia, yielding spurious relations with other consequences of enlargement such as lenticular tension. However, it seems unlikely that the two physical events of lenticular tension and ocular distortion would occur at the same time by mere coincidence.

Tension in an expanding sphere does not always imply a distorted shape. For example, a soap bubble remains a sphere as it expands. If lenticular resistance is important, it must be transmitted to the equator to create distortion. The ciliary muscle connects to sclera at its origin at the scleral spur, but this connection is located some 13 to 15 mm anterior to the equator.[45] The ciliary muscle also has an epichoroidal insertion near the ora serrata—closer to the equator, yet still several millimeters anterior to it. Suprachoroidal connective tissue is bound to the lamina fusca of the sclera,[45] but this connection is considered weak[18] and may not be sufficient to distort the globe.

Microscopic analysis shows that ciliary muscle elastic tendons make extensive connections with the elastic layer of Bruch's membrane.[46] This connection should be sufficient to transmit tension from the crystalline lens through the ciliary muscle to the choroid, evidenced by the impact of accommodation on the choroid. Psychophysical studies and direct observation of the choroid under scleral dissections demonstrate that the retina and underlying choroid stretch, even at the posterior pole, due to tension induced by accommodation.[18,47–50] This choroidal stretch from accommodation creates a negative pressure in the suprachoroidal space of up to 3 mm Hg.[18,50] Perhaps the surrounding band of extraocular muscles relieves this negative pressure to create distortion at the equator. Choroidal tension from lenticular resistance to stretching would be substantially less than that from maximal accommodation. An additional distorting factor may be an inherent anisotropy in the choroid. Intact choroid denuded of sclera expands more in the anteroposterior direction than in the equatorial direction when inflated and stretched.[51] One important perspective is that the distortion reported here is small. If 2.5 D corresponds to 1 mm of length in the periphery as it does centrally, the 1.86-D difference in relative peripheral refraction between myopes and hyperopes corresponds to only 0.74 mm.

Although the precise mechanism creating ocular distortion may be unknown, associations between a prolate ocular shape and the ocular components suggest that crystalline lens–induced tension within the choroid may initiate this process. This model deserves further consideration as investigators attempt to understand the structural and functional consequences of juvenile myopia.

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