1040-5488/13/9001-0009/0 VOL. 90, NO. 1, PP. 9–15
OPTOMETRY AND VISION SCIENCE

ORIGINAL ARTICLE

Peripheral Refraction and Retinal Contour in Stable and Progressive Myopia

Miguel Faria-Ribeiro*, António Queirós†, Daniela Lopes-Ferreira*, Jorge Jorge†, and José Manuel González-Méijome†

ABSTRACT

Purpose. To compare the patterns of relative peripheral astigmatic refraction (tangential and sagittal power errors) and eccentric eye length between progressing and stable young-adult myopes.

Methods. Sixty-two right eyes of 62 white patients participated in the study, of which 30 were nonprogressing myopes (NP group) for the last 2 years and 32 were progressing myopes (P group). Groups were matched for mean spherical refraction, axial length, and age. Peripheral refraction and eye length were measured along the horizontal meridian up to 35 and 30 degrees of eccentricity, respectively.

Results. There were statistically significant differences between groups ($p < 0.001$) in the nasal retina for the astigmatic components of peripheral refraction. The P group presented a hyperopic relative sagittal focus at 35 degrees in the nasal retina of $+1.00 \pm 0.83$ diopters, as per comparison with a myopic relative sagittal focus of -0.10 ± 0.98 diopters observed in the NP group ($p < 0.001$). Retinal contour in the P group had a steeper shape in the nasal region than that in the NP group (*t* test, $p = 0.001$). An inverse correlation was found ($r = -0.775$; $p < 0.001$) between retinal contour and peripheral refraction. Thus, steeper retinas presented a more hyperopic trend in the periphery.

Conclusions. Stable and progressing myopes of matched age, axial length, and central refraction showed significantly different characteristics in their peripheral retinal shape and astigmatic components of tangential and sagittal power errors. The present findings may help explain the mechanisms that regulate ocular growth in humans.

(Optom Vis Sci 2013;90:9–15)

Key Words: myopia progression, peripheral refraction, axial length, retinal shape, oblique astigmatism

Myopia progression is a serious public health concern. Beyond the limitations caused by refractive error, moderate to high myopia is associated with an increased risk of serious ophthalmic diseases like primary open-angle glaucoma, retinal detachment, or macular degeneration.[1]

Clinical evidence indicates that the peripheral refraction pattern plays an important role in the regulation of the growth of the human eye, as first reported by Hoogerheide et al.,[2] who found that, in a group of 214 young pilots entering the Danish Army, those who showed greater myopic progression over time also developed more hyperopic peripheral defocus. Another example is the lower progression rates in children wearing orthokeratology (OK)[3] lenses when compared with those wearing glasses. To date, the only justification for this behavior lies in the significant myopization effect induced by the OK treatment beyond the foveal area.[4,5] This has led to the development of soft contact lenses attempting to reproduce similar refractive patterns of peripheral myopic defocus. These have already proved effective in slowing myopia progression.[6]

Furthermore, animal studies have confirmed that locally induced hyperopic defocus causes a local increase in the axial length (AL) in chicks,[7–9] and that central vision is not essential for guiding the emmetropization mechanism, whereas the peripheral retina seems to be more relevant in this respect. This was also demonstrated in the studies by Smith et al.,[10–12] who reported that myopia could be induced even after laser photoablation of the fovea in rhesus monkeys.

As far as the human eye is concerned, the evidence that the posterior retinal contour of myopic eyes is steeper[13,14] (relatively more prolate or relatively less oblate) than that of emmetropic and hyperopic eyes fits well into the trends of more hyperopic peripheral retinal defocus in myopic patients. Although the development of peripheral hyperopia might be considered as a consequence

*OD, MSc
†OD, PhD
Clinical and Experimental Optometry Research Lab, Center of Physics, University of Minho, Braga, Portugal.

in addition to a cause of myopia progression,[15,16] if the peripheral retinal shape and/or relative peripheral refractive error guide the growth stimulus of axial elongation in myopic patients, it can be expected that stable and progressing myopes present differences regarding these parameters.

The purpose of the present study is to compare retinal steepness and the pattern of peripheral astigmatic refraction between stable and progressing myopes of similar age, axial refractive error, and AL.

METHODS

Subjects and Inclusion Criteria

Each patient signed a consent form before enrollment after the nature of the study was explained. The preliminary examinations for the inclusion criteria consisted of evaluating general health, family history, and corneal topography. Patients with any type of ocular pathology likely to create artifacts in data acquisition were excluded from this study. The distribution of the patients, among the two groups, was carried out according to their refractive history (last two prescriptions) and by comparing current subjective noncycloplegic refraction with the patient's habitual refraction. "Stable myopes" were defined as those who had been wearing the same prescription, according to the result of our subjective noncycloplegic refraction, for at least the last 2 years, within a range of ±0.25 diopters (D) in spherical equivalent. "Progressive myopes" were defined as those who had experienced a myopic shift in refraction of at least −0.50 D in the last year.

Other subjects whose current refractive status could not be confirmed or when the cycloplegic and noncycloplegic refraction differed by more than 0.50 D were excluded from this study.

Peripheral Refraction and Axial Length

Tropicamide 1% (Tropicil; Laboratórios Edol, Portugal) was used to establish control of pupil size and accommodation. After administration of two drops of tropicamide 1%, the waiting period required for establishing cycloplegia and mydriasis was about 30 minutes. Intraocular pressure was measured before and 30 minutes after the instillation of tropicamide, as well as at the end of the data collection and before the patient left the clinic, with the noncontact tonometer Ocular Response Analyzer (ORA Technologies Reichert, Depew, NY) to monitor the potential acute increase in intraocular pressure caused by iridocorneal angle closure during mydriasis. Measurement of central and peripheral refraction without correction was carried out with the open-field Autorefractometer/Keratometer Grand Seiko WAM-5500 (Grand Seiko Co., Ltd., Hiroshima, Japan)[17,18] attached to a portable computer with data acquisition software (DRRE; CEORLab, University of Minho, Braga, Portugal), allowing for all measurements to be automatically exported to an Excel spreadsheet, thus avoiding errors in data collection, for later statistical analysis with the appropriate software.

The fixation targets were placed at 2.5 m from the patient's eye and consisted of 15 light-emitting diodes (LEDs) in the horizontal direction: one central, seven to the right side, and seven to the left side. The LEDs were separated from each other by an angle of 5 degrees. The subject was seated with the head stabilized in a chin rest so that the right eye was aligned with the central LED. The left eye was occluded while patients kept their head stationary and rotated their right eyes to view a series of fixation targets. This procedure was first described by Radhakrishnan and Charman,[19] whose results, obtained under similar conditions, failed to find significant differences in peripheral refraction when measurements where made with eye turn rather than head turn. For the right eye, the fixation of an object positioned on the right side of the central point corresponds to the temporal retina measures. Five readings were averaged at each position. The axis of the autorefractometer was aligned with the center of the entrance pupil during all measurements.

The IOLMaster (Carl Zeiss, Germany) optical coherent biometer with an apparatus attached to the headrest, designed to accurately control the gaze position to the desired eccentricities, as previously described by Mallen and Kashyap[20] in 2007, was used to determine central and peripheral eye length (EL). The system is composed of a beam splitter placed at an angle of 45 degrees to the infrared laser beam, a fixation target with the Maltese cross printed in black, and a circular goniometer mounted on a bracket attached to the headrest. The goniometer was placed above the center of rotation of the eye, approximately 15 mm behind the apex of the cornea.[21] Three measures were averaged in the central, 10-degree, 20-degree, and 30-degree positions (temporal and nasal). The values were exported to a Microsoft Excel 2007 (Microsoft, USA) database for further processing.

Decomposition of the Astigmatic Off-Axis Image

When an off-axis beam of light is refracted at the cornea, it becomes astigmatic (oblique astigmatism). The image is formed in two separate focal lines called tangential and sagittal.[22] As with astigmatic refraction in general, the focal lines are each perpendicular to the associated principal meridian.

The relative peripheral refraction or refractive error, which can be interpreted as the patient's off-axis refraction when compensating for his/her central refractive error, was obtained by subtracting the central refraction from the peripheral refraction. Because of inherent problems when analyzing cylinder power in its conventional form central and peripheral refraction were converted into power vector components (M, J_0, J_{45}), using Fourier analysis, as described by Thibos et al.[23]:

$$M = Sph + \frac{Cyl}{2}$$

$$J_0 = \frac{-Cyl \cdot \cos(2\Theta)}{2}$$

$$J_{45} = \frac{Cyl \cdot \sin(2\Theta)}{2}.$$

Central M was subtracted from the off-axis M to obtain the relative peripheral spherical equivalent refraction (RPSER). The two relative peripheral astigmatic components, tangential (F_T') and sagittal (F_S') power errors, were calculated using the following equations:

$$F_T' = M + J_0$$
$$F_S' = M - J_0$$

It is important to note that relative refractive error is a rough approximation to the actual peripheral defocus the patient might

TABLE 1.
Average ± SD AL, *M*, and age in both groups

	NP group	P group	Difference P-NP	p	Test
AL, mm	24.58 ± 0.83	24.63 ± 0.87	0.05	0.821	T
M, D	−2.96 ± 1.63	−2.68 ± 1.27	0.27	0.657	U
Age, yr	21.94 ± 1.72	22.10 ± 1.81	0.16	0.591	U

T, *t* test for two independent samples; U, Mann-Whitney *U* test.

habitually experience, as it does not account for the off-axis optical effects induced by the different types of correction options.[24–26]

Statistical Analysis

The SPSS software package version 18 (SPSS Inc., Chicago, IL) was used for statistical analysis. The Kolmogorov-Smirnov test was applied to evaluate the normality of data distributions. When normality could not be assumed, the Mann-Whitney *U* test and Spearman correlation coefficient were used as alternatives to the *t* test for two unpaired samples and to the Pearson coefficient. A value of $p < 0.05$ was considered statistically significant.

RESULTS

Using a myopia criterion of at least −0.50 D of central equivalent refraction, measurements were carried out on 62 myopic right eyes, 32 with a known stable or nonprogressing central refraction (NP group), of which 25 (78.13%) were females, and 30 with a myopic refraction still in progression (P group), of which 24 (80%) were females. No statistically significant differences were found between groups in terms of AL, central spherical equivalent refraction (*M*), and age, as shown in Table 1. Regarding the patients' habitual corrections, those were similarly distributed between groups, with 14 of 32 contact lens (CL) wearers in the NP group and 16 of 32 in the P group. Those wearing CLs had a quite recent history of CL wear (less than 3 years). Furthermore, the average refractive error was neither different when compared for P versus NP groups for CL wearers and for spectacle wearers separately.

Relative Peripheral Spherical Equivalent Refraction

Fig. 1 represents the RPSER profile along the horizontal meridian for both groups, fitted with third-order polynomials. Statistically significant differences were found between the two groups. Indeed, a more hyperopic peripheral refraction in the nasal hemifield was observed in the progressing group. In the P group, peripheral focus was more myopic for almost all the eccentricities assessed than in the NP group, with differences being statistically significant in the nasal retina ($p \leq 0.002$). In the P group, the RPSER assumes values close to emmetropia for all visual field eccentricities under evaluation.

Off-Axis Relative Astigmatic Tangential and Sagittal Foci

Panels A and B of Fig. 2 represent the profiles of tangential and sagittal power errors at each of the eccentricities assessed fitted

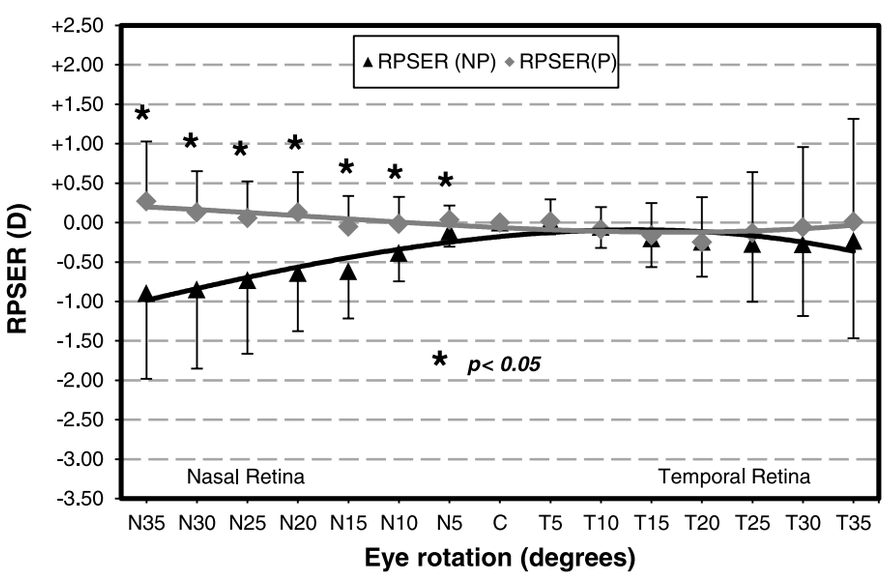

FIGURE 1.
Relative peripheral spherical equivalent refraction as a function of eye rotation angle for the nonprogressing (NP) and progressing (P) groups (*$p < 0.05$).

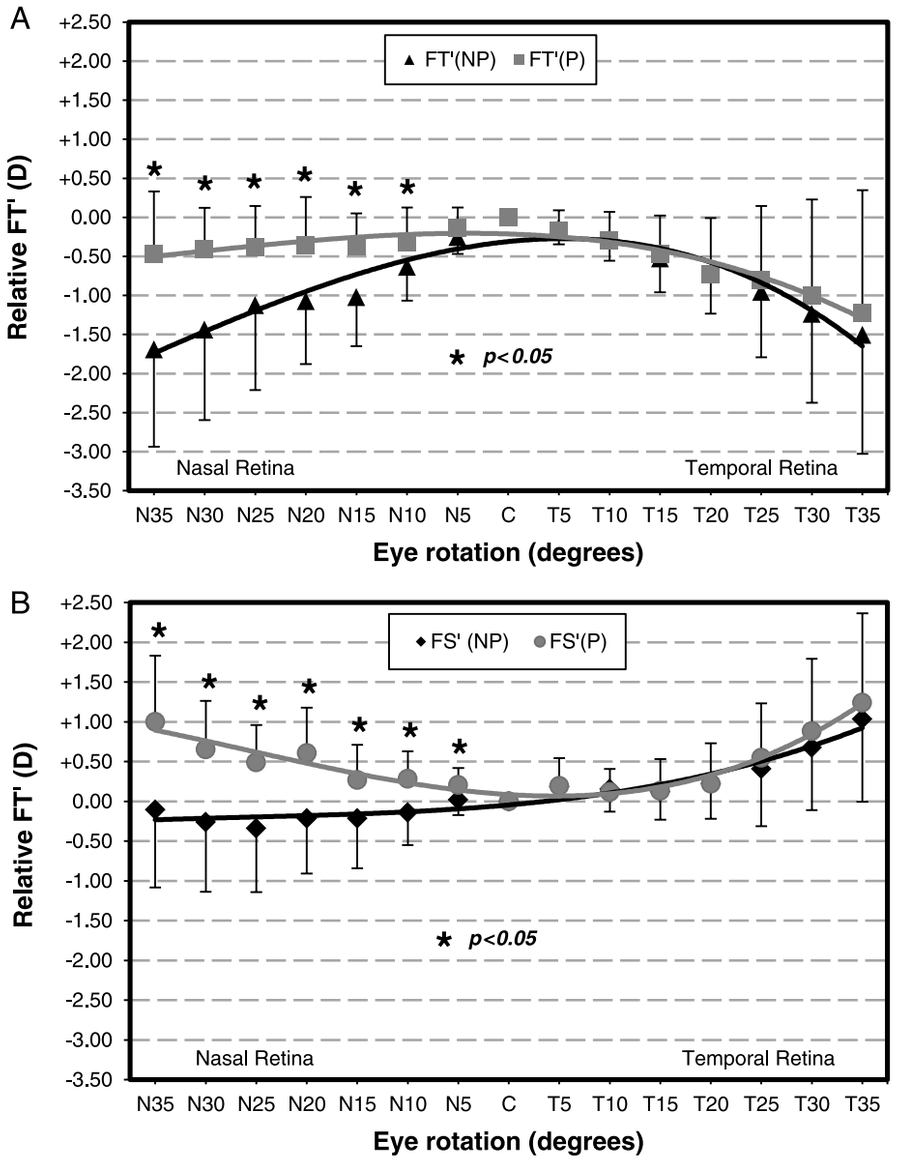

FIGURE 2.

A, Relative tangential (F_T') and (B) relative sagittal (F_S') power errors as a function of eye rotation angle for the nonprogressing (NP) and progressing (P) groups (*p < 0.05).

with third-order polynomials. The results show statistically significant differences between both groups in the nasal retina, with F_S' and F_T' values being more hyperopic or less myopic in the NP group, with exception for F_T' values at the 5-degree nasal eccentricity (p = 0.069). The F_S' image shell in progressing group will be formed "behind" the retina (hyperopic), whereas the F_T' shell is located "closer" to the retina (less myopic). As for the nonprogressing group, both astigmatic image shells remain in front of the nasal retina (myopic), with the F_S' becoming hyperopic in the temporal retina hemifield.

Retinal Steepness

To establish a comparison of retinal steepness along the evaluated eccentricities between the two groups, the variables related to the variation of EL, for nasal and temporal hemifields were calculated (Fig. 3). Although the EL-plotted shell does not represent real retinal contour, as the IOLMaster off-axis measurements fail to account for refraction within the eye and ignore differences in refractive index along the optical path, particularly along oblique directions inside the crystalline lens,[27] any potential source of error will be similarly distributed in both groups, allowing us to compare them. The relative peripheral EL (nasal or temporal RPEL) was obtained by subtracting the AL from the eccentric EL. The RPEL variable is intended to be a quantitative parameter of retinal steepness. Thus, the more negative the RPEL, the shorter the EL along that direction in comparison with the AL in primary gaze fixation (ie, negative and positive RPEL values represent relatively steeper and flatter retinas, respectively).

Regarding the shape differences in the posterior pole between both groups, the results show a steeper shape in the P group, which is statistically significant in the nasal region (*t* test, p = 0.001).

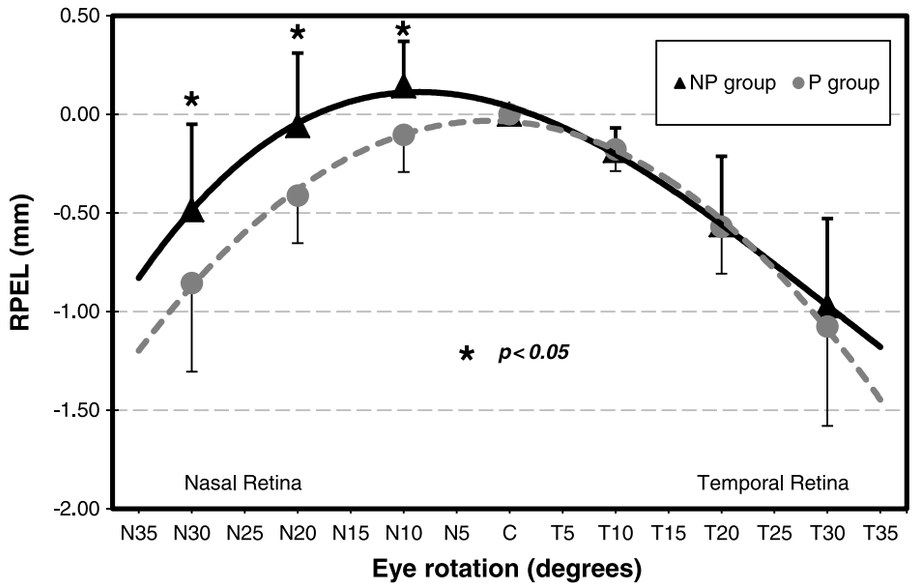

FIGURE 3.

Relative peripheral eye length (RPLE) as a function of eye rotation angle for the nonprogressing (NP) and progressing (P) groups (*p < 0.05).

To establish a relationship between ocular shape and peripheral refraction, correlation coefficients were determined between RPEL and each of the relative peripheral refraction components, RPSER, F_T', and F_S'. The shape of the posterior pole (RPEL) shows a strong inverse correlation with peripheral refraction (Table 2), which suggests that eyes with a steeper shape, whether P or NP, have a more hyperopic relative peripheral refraction.

DISCUSSION

We found statistically significant differences between both groups ($p < 0.001$) in the nasal retina for the peripheral refractive astigmatic components. Although no comparison between progressing and stable myopia has been previously conducted, other studies[2,28,29] found a trend towards a more hyperopic relative peripheral refraction in myopes than in emmetropes or hyperopes. This peripheral hyperopic refraction is believed to be responsible for myopia development, as the eye's visuallyguided growth mechanism tries to compensate with further elongation for the imposed peripheral defocus even in the presence of an optimal central correction and a perfectly focused central image.

According to our results, the NP group presents a myopic RPSER. If in fact peripheral error seems to regulate ocular growth, as the animal models clearly suggest,[10–12,30,31] the reason for myopia stabilization might be related with the same mechanism. One might speculate that, if for any reason during eye growth, the peripheral refraction image shell falls in front of the retina the visually guided stimulus will change and the eye will no longer elongate trying to "catch" a new focal plane behind the actual location of the retina.

In the P group, the results show RPSER values close to emmetropia for all eccentricities, suggesting that a circle of least confusion in focus with the retina (RPSER = 0) does not seem to be sufficient to halt the continuous increase in AL in this group. Analyzing the question in other terms, one might also hypothesize that the analysis of peripheral refractive error in terms of spherical equivalent might be insufficient to identify the visual feedback mechanism that guides ocular growth. This strengthens the relevance considering other aspects related to peripheral

TABLE 2.

Correlation between retinal steepness (RPEL) and refraction (RPSE, F_T', F_S') at 30 degrees eccentricity nasal and temporal

				NP group			
RPEL	r*	RPSER	p	F_T'	p	F_S'	p
	N30	−0.606	<0.001	−0.505	<0.001	−0.730	<0.001
	T30	−0.788	<0.001	−0.744	<0.001	−0.775	<0.001
				P group			
RPEL	r**	RPSER	p	F_T'	p	F_S'	p
	N30	−0.657	<0.001	−0.546	0.020	−0.678	<0.001
	T30	−0.541	0.002	−0.472	0.008	−0.595	0.001

*Spearman coefficient.
**Pearson coefficient.

refractive error other than spherical equivalent refraction, for example, the position of the astigmatic foci (F_T' and F_S'), when assessing peripheral refraction.

However, the hypothesis that astigmatic focus plays a role in ocular growth needs further discussion. It has been previously suggested that the peripheral retina has neurons tuned for different orientations[32,33] and that it makes use of the two astigmatic foci to recognize the defocus signal to regulate ocular growth. Bearing this in mind, the peripheral retinal neuron circuits might have distinct levels of sensitivity for the tangential and sagittal foci inputs. A similar process is used in some optical devices such as compact discs players that use an astigmatic lens to optimize the focusing mechanism. When one axis is better focused than the other, dotlike features on the disc are projected into elliptical shapes. The orientation of the major and minor elliptical axes indicates which axis is better focused and, hence, in which direction the lens needs to move to compensate for it. In a similar fashion, it could be hypothesized that the ocular growth mechanism in the peripheral retina might also use similar orientation cues to assess the two astigmatic image shell "positions" and thus compensate for peripheral hyperopic defocus when the relative peripheral sagittal focal line "stands behind" the retina, as previously suggested by Howland.[34]

The data from this study suggest that a myopic sagittal focal image shell, as seen in the nasal retina of the NP group, may result in a neural peripheral retinal input that inhibits ocular growth. Conversely, in the P group, the results show that a hyperopic sagittal relative focal in the nasal hemifield might activate the ocular growth mechanism and accelerate axial growth to compensate for peripheral defocus at the expense of a central increase in myopia. This hypothesis is consistent with the experiments described in US patent 7,025,460 by Smith et al.,[35] who reported a trend for the eye, in the presence of mixed astigmatism, to grow to reposition the retina with the most "posteriorly positioned" astigmatic focal line (F_S'). This process may then start over when new lenses are prescribed to compensate for the increase in central myopia; furthermore, as the eyeball elongates, the retina becomes steeper, thus increasing the hyperopic trend in the periphery. The potential impact in other directions, including vertical and oblique directions, might also be investigated.

The present hypothesis based on previous studies and on the data obtained in this study lacks a baseline and a longitudinal follow-up to confirm it, but even if one considers that a more hyperopic relative peripheral refraction might be a consequence rather than a cause of myopia progression, it is still intriguing to notice the statistically significant differences regarding nasal retina shape between both groups. Such differences might have been caused by an asymmetric ocular elongation during myopia progression in the NP or were already there before the onset. Either way, one cannot exclude the possibility that the NP eyes have grown and altered their shape to compensate for a hyperopic sagittal error as previously described by Smith el al.[35] The asymmetry observed between the temporal and nasal retina hemifields has also been found in at least one other study[36] where retinal shape was obtained by A-scan ultrasonography and peripheral refraction in white and Chinese patients. In this study, Logan et al.[36] found that the nasal-temporal asymmetry only presented itself in the white group, with greater enlargement of the nasal retinal sector, but the reason for such asymmetry remained unclear.

Our results suggest that the nasal half of the retina might be more important in terms of mechanism of ocular growth control. Nevertheless, considering that the animal retina can respond asymmetrically and locally to deprived stimuli,[37] we might argue that the nasal retina, being exposed to a wider visual field experience, might also be more sensitive to peripheral astigmatic defocus. This would help explain why myopia progression stops in the NP group when the nasal retina no longer receives a hyperopic defocus signal from the sagittal foci even when the temporal retina still receives it.

In conclusion, the myopic patients in the P group showed a more hyperopic relative astigmatic defocus than the NP group. Even when RPSER assumes values close to zero or slightly myopic, it still seems that the hyperopic stimulus provided by the sagittal foci can be sufficient to induce axial growth in the P group. These results seem be in agreement with previous theories that point to a visually guided grown mechanism and provide new outcomes to understand the process behind it. The strong correlation found between eye shape and refraction, along with the high differences in shape and refraction between both groups in the nasal retina, may be indicative of a distinct sensitivity "weight" between the two retina hemifields.[38]

ACKNOWLEDGMENTS

None of the authors have any proprietary or financial interest in any of the materials mentioned in this article.

Received May 3,2012; accepted September 17, 2012.

REFERENCES

1. Saw SM, Gazzard G, Shih-Yen EC, Chua WH. Myopia and associated pathological complications. Ophthalmic Physiol Opt 2005; 25:381–91.
2. Hoogerheide J, Rempt F, Hoogenboom WP. Acquired myopia in young pilots. Ophthalmologica 1971;163:209–15.
3. Cho P, Cheung SW, Edwards M. The longitudinal orthokeratology research in children (LORIC) in Hong Kong: a pilot study on refractive changes and myopic control. Curr Eye Res 2005;30:71–80.
4. Charman WN, Mountford J, Atchison DA, Markwell EL. Peripheral refraction in orthokeratology patients. Optom Vis Sci 2006;83: 641–8.
5. Queirós A, Gonzalez-Meijome JM, Jorge J, Villa-Collar C, Gutierrez AR. Peripheral refraction in myopic patients after orthokeratology. Optom Vis Sci 2010;87:323–9.
6. Sankaridurg P, Holden B, Smith E, 3rd, Naduvilath T, Chen X, de la Jara PL, Martinez A, Kwan J, Ho A, Frick K, Ge J. Decrease in rate of myopia progression with a contact lens designed to reduce relative peripheral hyperopia: one-year results. Invest Ophthalmol Vis Sci 2011;52:9362–7.
7. Irving EL, Callender MG, Sivak JG. Inducing myopia, hyperopia, and astigmatism in chicks. Optom Vis Sci 1991;68:364–8.
8. Wallman J, Adams JI. Developmental aspects of experimental myopia in chicks: susceptibility, recovery and relation to emmetropization. Vision Res 1987;27:1139–63.

9. Wildsoet CF, Schmid KL. Emmetropization in chicks uses optical vergence and relative distance cues to decode defocus. Vision Res 2001;41:3197–204.
10. Smith EL, 3rd, Ramamirtham R, Qiao-Grider Y, Hung LF, Huang J, Kee CS, Coats D, Paysse E. Effects of foveal ablation on emmetropization and form-deprivation myopia. Invest Ophthalmol Vis Sci 2007;48:3914–22.
11. Smith EL, 3rd, Hung LF, Huang J, Blasdel TL, Humbird TL, Bockhorst KH. Effects of optical defocus on refractive development in monkeys: evidence for local, regionally selective mechanisms. Invest Ophthalmol Vis Sci 2010;51:3864–73.
12. Smith EL, 3rd, Huang J, Hung LF, Blasdel TL, Humbird TL, Bockhorst KH. Hemiretinal form deprivation: evidence for local control of eye growth and refractive development in infant monkeys. Invest Ophthalmol Vis Sci 2009;50:5057–69.
13. Atchison DA, Pritchard N, Schmid KL, Scott DH, Jones CE, Pope JM. Shape of the retinal surface in emmetropia and myopia. Invest Ophthalmol Vis Sci 2005;46:2698–707.
14. Schmid GF. Association between retinal steepness and central myopic shift in children. Optom Vis Sci 2011;88:684–90.
15. Sng CC, Lin XY, Gazzard G, Chang B, Dirani M, Lim L, Selvaraj P, Ian K, Drobe B, Wong TY, Saw SM. Change in peripheral refraction over time in Singapore Chinese children. Invest Ophthalmol Vis Sci 2011;52:7880–7.
16. Mutti DO, Sinnott LT, Mitchell GL, Jones-Jordan LA, Moeschberger ML, Cotter SA, Kleinstein RN, Manny RE, Twelker JD, Zadnik K. Relative peripheral refractive error and the risk of onset and progression of myopia in children. Invest Ophthalmol Vis Sci 2011;52:199–205.
17. Queirós A, Gonzalez-Meijome J, Jorge J. Influence of fogging lenses and cycloplegia on open-field automatic refraction. Ophthalmic Physiol Opt 2008;28:387–92.
18. Queirós A, Jorge J, Gonzalez-Meijome JM. Influence of fogging lenses and cycloplegia on peripheral refraction. J Optom 2009;2:83–9.
19. Radhakrishnan H, Charman WN. Peripheral refraction measurement: does it matter if one turns the eye or the head? Ophthalmic Physiol Opt 2008;28:73–82.
20. Mallen EA, Kashyap P. Technical note: measurement of retinal contour and supine axial length using the Zeiss IOLMaster. Ophthalmic Physiol Opt 2007;27:404–11.
21. Fry GA, Hill WW. The center of rotation of the eye. Am J Optom Arch Am Acad Optom 1962;39:581–95.
22. Bennett AG, Rabbetts RB. Bennett & Rabbetts' Clinical Visual Optics, 3rd ed. Boston, MA: Butterworth-Heinemann; 1998.
23. Thibos LN, Wheeler W, Horner D. Power vectors: an application of Fourier analysis to the description and statistical analysis of refractive error. Optom Vis Sci 1997;74:367–75.
24. Bakaraju RC, Ehrmann K, Papas E, Ho A. Do peripheral refraction and aberrations profiles vary with the type of myopia? An illustration using a ray-tracing approach. J Optom 2009;2:29–38.
25. Bakaraju RC, Ehrmann K, Ho A, Papas EB. Pantoscopic tilt in spectacle-corrected myopia and its effect on peripheral refraction. Ophthalmic Physiol Opt 2008;28:538–49.
26. Lin Z, Martinez A, Chen X, Li L, Sankaridurg P, Holden BA, Ge J. Peripheral defocus with single-vision spectacle lenses in myopic children. Optom Vis Sci 2010;87:4–9.
27. Atchison DA, Charman WN. Can partial coherence interferometry be used to determine retinal shape? Optom Vis Sci 2011;88:601–7.
28. Tabernero J, Schaeffel F. More irregular eye shape in low myopia than in emmetropia. Invest Ophthalmol Vis Sci 2009;50:4516–22.
29. Mutti DO, Sholtz RI, Friedman NE, Zadnik K. Peripheral refraction and ocular shape in children. Invest Ophthalmol Vis Sci 2000;41:1022–30.
30. Smith EL, 3rd, Kee CS, Ramamirtham R, Qiao-Grider Y, Hung LF. Peripheral vision can influence eye growth and refractive development in infant monkeys. Invest Ophthalmol Vis Sci 2005;46:3965–72.
31. Smith EL, 3rd, Hung LF, Harwerth RS. Effects of optically induced blur on the refractive status of young monkeys. Vision Res 1994;34:293–301.
32. Schall JD, Perry VH, Leventhal AG. Retinal ganglion cell dendritic fields in old-world monkeys are oriented radially. Brain Res 1986;368:18–23.
33. Passaglia CL, Troy JB, Ruttiger L, Lee BB. Orientation sensitivity of ganglion cells in primate retina. Vision Res 2002;42:683–94.
34. Howland HC. A possible role for peripheral astigmatism in the emmetropization of the eye: Symposium 17, abstract 3. In: Tarutta E, Chua WH, Young T, Goldschmidt E, Saw S-M, Rose KA, Smith EL, 3rd, Mutti DO, Ashby R, Stone RA, Wildsoet C, Howland HC, Fischer AJ, Stell WK, Reichenbach A, Frost M, Gentle A, Zhu X, Summers-Rada Jody, Barathi V, Jiang L, McFadden S, Guggenheim JA, Hammond C, Schippert R, To CH, Gwiazda J, Marcos S, Collins M, Charman WN, Artal P, Tabernero J, Atchison DA, Seidemann A, Uttenweiler D, Troilo D, Norton TT, Wallman J, eds. Myopia: Why Study the Mechanisms of Myopia? Novel Approaches to Risk Factors Signaling Eye Growth—How Could Basic Biology Be Translated into Clinical Insights? Where Are Genetic and Proteomic Approaches Leading? How Does Visual Function Contribute to and Interact with Ametropia? Does Eye Shape Matter? Why Ametropia at All? Optom Vis Sci 2011;88:447.
35. Smith E, III, Greeman N, Jr., Ho A, Holden BA. Methods and Apparatuses for Altering Relative Curvature of Field and Positions of Peripheral, Off-Axis Focal Positions. US Patent 7,025,460 B2. April 11, 2006.
36. Logan NS, Gilmartin B, Wildsoet CF, Dunne MC. Posterior retinal contour in adult human anisomyopia. Invest Ophthalmol Vis Sci 2004;45:2152–62.
37. Raviola E, Wiesel TN. An animal model of myopia. N Engl J Med 1985;312:1609–15.
38. Charman WN. Myopia, posture and the visual environment. Ophthalmic Physiol Opt 2011;31:494–501.

Miguel Faria-Ribeiro
Clinical and Experimental Optometry Research Lab
Center of Physics (Optometry)
University of Minho
4710-057 Braga
Portugal
e-mail: mig.afr@gmail.com