0093-7002/87/6409-0657$02.00/0
AMERICAN JOURNAL OF OPTOMETRY & PHYSIOLOGICAL OPTICS

Vol. 64, No. 9, pp. 657–663
Printed in U.S.A.

Reduction in Axial Length with Age: An Emmetropizing Mechanism for the Adult Eye?

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ABSTRACT

Mechanisms accounting for the fact that the eye tends to become and remain emmetropic have been proposed for the developing eye, but no such mechanisms have been proposed for the adult eye. In the study reported here, refractive component data published by Sorsby and his co-workers were reanalyzed in terms of the variations in these components with age. Included in the analysis are component data for one eye of each of 271 subjects from age 4 to age 70 years, whose ocular refraction was between plano and +2.00 D. The results of this analysis show that the axial length of the eye appears to decrease during the adult years of life, concurrently with a decrease in anterior chamber depth and an increase in the refractive power of both the cornea and the lens. It is proposed that a reduction in the axial length of the adult eye serves as an emmetropizing mechanism, occurring in harmony with the increase in the refracting power of the eye, which would otherwise cause the refraction of the eye to move in the myopic direction.

Key Words: emmetropization, mechanisms of emmetropization, axial length, presbyopic eye

When the refractive error distribution of a large number of people is plotted, the invariable result is a narrow, leptokurtic distribution, skewed in the myopic direction, rather than the broader, normal distribution found for other biological variables. The leptokurtosis has been explained by the fact that the components of ocular refraction (corneal refracting power, anterior chamber depth, lens refracting power, and axial length of the eye) are interdependent, rather than independent, variables: as the eye grows, the values of all the refractive components tend to vary in harmony, with the result that the eye tends to become, and to remain, emmetropic.

This tendency for the eye to become and remain emmetropic has been termed emmetropization, and has been defined as "a process presumed to be operative in producing a greater frequency of emmetropia and near-emmetropia than would be expected in terms of chance distribution. . . ."[1] Mechanisms that may be responsible for emmetropization have been postulated by many authors, including Hirsch and Weymouth,[2] Sorsby et al.,[3] Van Alphen,[4] and Hofstetter.[5] The proposed mechanisms have been applied almost exclusively to the developing eye. Even though a large proportion of human eyes do not develop significant ametropia during the entire life span, little or no consideration has been given to the possibility that emmetropizing mechanisms may exist in the mature eye.

The purpose of this paper is to consider the possibility that a gradual reduction in the axial length may play a role in the emmetropization of the adult eye.

MECHANISMS OF EMMETROPIZATION IN THE DEVELOPING EYE

During the first half of the present century it was believed that all children were hyperopic at birth. However, studies reported by Cook and Glasscock[6] and by Goldschmidt[7] have shown a wide distribution of refractive error at birth, extending to 7 D or more in both the myopic and hyperopic directions. This distribution has been shown by Kemph et al.[8] to narrow considerably by 6 years of age; Mohindra and Held[9] have shown that the distribution begins to narrow even during the first year of life. The process of emmetropization therefore begins to operate at a very early age, and in fact the refractive error distribution is found to be more leptokurtic for children in the first year of school than at any other time during the life-span.

Received October 16, 1986; revision received January 13, 1987.

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Although relatively few children have clinically significant myopia or hyperopia when entering school, it has been shown by Hirsch[10] and by Blum et al.[11] that the prevalence of myopia increases steadily during the school years, reaching about 15% by the age of 14 or 15 years. For these children, the process of emmetropization has ceased to operate.

In his classic study of the refractive components of the eye, Stenstrom[12] found a correlation between refractive error and axial length of −0.76, indicating that axial length is the most important determiner of refractive state. Hirsch and Weymouth[2] reanalyzed Stenstrom's data, using partial correlations, and found that if the other components of refraction were held constant, the correlation between refractive error and axial length increased to −0.87. They proposed the existence of two emmetropizing mechanisms; one involving the deepening of the anterior chamber (leading to hyperopia) and the other involving the flattening o the cornea (also leading to hyperopia) as the eye grows in length (which would, of course, lead to myopia).

Sorsby et al.[3] concluded that the eye grows some 8 mm in length from birth to the adult years, and proposed that changes in the refracting power of the cornea and the lens could act as correlating mechanisms to maintain the eye at or near emmetropia as it grows. They suggested that the relation with the corneal refraction is simply a mechanical one, involving a tendency for the cornea to flatten as the eye grows; for the mechanism involving the lens they proposed the existence of a biological mechanism in which the retina acts as an "organizer," determining the curvature of the lens as the eye grows.

Van Alphen[4] also reanalyzed Stenstrom's data, submitting them to factor analysis, and proposed that emmetropization takes place by means of a closed loop mechanism in which the choroid and the ciliary body act as an elastic envelope, working in opposition to the intraocular pressure and controlling the amount of stretch of the eye. He proposed that information concerning focus is fed back from the macula to a cortical or subcortical center in the brain, which in turn sends information to the ciliary muscle by means of the Edinger-Westphal nucleus, bringing about the amount of stretch required for the eye to maintain emmetropia. However, if the choroid-ciliary body envelope loses its elasticity, the increase in the length of the eye would be uncontrolled, resulting in myopia.

Hofstetter[5] suggested that emmetropization may not be a biological process at all, but can be accounted for entirely on the basis of mathematics. Calling attention to the fact that radial dimensions are used both for the dioptric and the structural appraisal of the eye, he argued that by using a "universal" schematic eye it would be possible to construct an assortment of emmetropic eyes. A frequency distribution of such eyes would produce an infinitesimally slender vertical line, the absolute limit of leptokurtosis. However, small variances in the values of the refractive components would be expected to cause a slight dispersion away from perfect leptokurtosis.

These and other theories of emmetropization have been applied only to the developing eye. Their use has been confined almost entirely in attempting to explain (1) how the eye maintains emmetropia, and (2) how myopia develops when emmetropizing mechanisms fail to operate.

POSSIBLE EMMETROPIZING MECHANISMS IN THE ADULT EYE

Vision care practitioners have long been aware that ocular refraction tends to change little during the early adult years. Morgan[13] reported on a study of 95 nonvisually selected subjects who were refracted at age 13 years and again at age 33 years. During the 20-year period, mean spherical equivalent refraction changed (in the direction of less hyperopia or more myopia) only 0.22 D for females and 0.04 D for males. On the basis of a retrospective study of the refractive data of a group of 111 optometrists, Grosvenor[14] found that those subjects who were emmetropic at age 20 tended to be still emmetropic at age 40, whereas those who were myopic at age 20 were still myopic at age 40, and those who were hyperopic at age 20 were still hyperopic at age 40.

Changes in the refraction of older adults were studied by Hirsch,[15] who analyzed refractive data for patients seen in his optometric practice and found that although many patients changed little in refraction beyond the age of 55 years, some patients who had been emmetropic changed in the direction of increasing hyperopia, whereas others changed in the myopic direction due to nuclear sclerosis of the lens.

The fact that many people remain emmetropic or near-emmetropic throughout the adult years points to the existence of one or more emmetropizing mechanisms in the adult eye. In looking for such a mechanism, our attention is called to the relation between anterior chamber depth and lens power: the crystalline lens grows throughout life, bringing about a gradual decrease in anterior chamber depth. Because the decrease in anterior chamber depth would tend to drive the refraction of the eye in the direction of myopia, one might imagine that there exists an emmetropizing mechanism involving the lens

that drives the refraction in the direction of hyperopia. Such an emmetropizing mechanism was at one time very attractive, inasmuch as Donders[16] had suggested that the lens flattened as it became thicker with age. However, it has been reported by Brown[17] that the lens actually steepens in the process of becoming thicker with age, and the combination of a shallowing anterior chamber and a steepening lens would not be expected to act as an emmetropizing mechanism, because both would drive the refraction in the direction of myopia.

Although the literature on refraction includes many studies of the components of ocular refraction, none of these studies have considered the components of refraction of the adult eye in the light of possible variations with age. Fortunately, Sorsby and his co-workers have published complete refractive component data for each of their subjects in two large-scale studies: *Emmetropia and its Aberrations* (already referred to)[3] and *Refraction and its Components in Twins*, by Sorsby et al.[18]

Procedures used in the studies by Sorsby et al. were the following: ocular refraction was performed by retinoscopy under cycloplegia, and recorded as the refraction in the vertical meridian; corneal refraction in the vertical meridian was measured by means of a Haag-Streit keratometer; anterior chamber depth and lens thickness were obtained by the use of slitlamp photography; lens radii of curvature in the vertical meridian were obtained by photographing the 3rd and 4th Purkinje images; and the axial length of the eye was calculated on the basis of the measured values of the refracting components, using assumed indices of refraction for each of the ocular media. The two studies included data for a total of 1091 eyes of subjects ranging in age from 4 to 70 years, with refractive errors ranging from +12 to −21 D.

ANALYSIS OF THE SORSBY DATA

Because the object of the analysis was to consider only subjects who were emmetropic or near-emmetropic, my analysis of the Sorsby data included only those subjects whose vertical ocular refraction was between plano and +2.00 D. A range of plano to +1.00 D would have been more desirable, but unfortunately the numbers of subjects in each age group would have been too small to make it possible to apply tests of statistical significance. Data were used for only one eye of each subject (the right eye, unless the right eye was outside the plano to +2.00 D range, in which case the left eye data were used if within this range). In the case of unovular twins, data for the right eye of only one of a pair of twins were used. Data for a total of 271 subjects (135 males and 136 females) were included in the analysis.

RESULTS

As shown in the third and fourth columns of Table 1, the selection of subjects whose refractive error was between plano and +2.00 D resulted in a reasonably large number of subjects in each decade of life, with roughly equal numbers of males and females in each age group. The final column in this table shows that mean refractive error of the subjects varied little with age, ranging only from 0.6 to 1.1 D.

Refractive component data will be considered in terms of two age ranges: (1) ages 4 to 19 years, during which much of the growth of the eye takes place, and (2) ages 20 years and beyond. Mean values of the refractive components of subjects age 4 to 19 years are shown in Table 2. As shown in this table, each of the refractive components varies rather markedly during this period of life: mean corneal refracting power decreases by 0.5 D from age 4 to 9 years to age 10 to 19 years, whereas mean anterior chamber depth increases by 0.2 mm, mean lens power decreases by 1.6 D, mean refractive power of the eye decreases by 1.9 D, and mean axial length of the eye increases by 0.7 mm. These results are not unexpected. They demonstrate the effects of the process of emmetropization, the refractive power of the eye decreasing to "fit"

TABLE 1. Categories of subjects on the basis of age, sex, and refractive error, for subjects whose ocular refraction is between plano and +2.00 D.[a]

Age Group (yr)	Mean Age (yr)	No. of Subjects			Mean Refractive Error (D)
		Male	Female	Total	
4–9	7	24	25	49	+1.1
10–19	13	24	24	48	+0.6
20–29	23	27	19	46	+0.6
30–39	36	15	20	35	+0.7
40–49	44	32	33	65	+0.9
50+	55	13	15	28	+0.9

[a] Data of Sorsby et al.[3] and Sorsby et al.[18]

the increasing axial length of the eye during the period of ocular growth.

Mean values for the refractive components of subjects age 20 years and beyond are shown in Table 3. This table shows the following differences between mean values for eyes of subjects age 50 years or older, compared to those between 20 and 29 years of age: at age 50 and beyond (1) mean anterior chamber depth is 0.3 mm shorter; (2) mean lens power is 1.0 D greater (confirming the finding of Brown[17] that the lens increases in convexity with age); (3) mean refracting power of the eye is 1.9 D greater; and (4) mean axial length is 0.6 mm shorter. The results shown in this table support the hypothesis that concurrently with an increase in the refracting power of the eye during this period of life (due to an increase in the refracting power of the cornea and lens and a decrease in anterior chamber depth) the axial length of the eye decreases, thereby "fitting" the stronger optical system.

The refractive component variations with age can be visualized more easily with the help of the graph shown in Fig. 1, which displays mean values for each of the refractive components for each of the 10-year age groups. It is of interest that the apparent increase in mean refracting power of the eye ("apparent," because we are dealing with cross-sectional data rather than longitudinal data) from age 20 to 29 to age 50 years and beyond, amounting to almost 2 D, is compensated by an apparent decrease in mean axial length of 0.6 mm. This corresponds very closely to the time-honored ratio of a 3 D change of refracting power for each millimeter change in axial length.

Statistically Significant Relations

In order to determine whether the age differences found in refractive components were statistically significant or could have occurred by chance, mean values for total refractive power of the eye and for axial length were tested for age 4 to 9 years compared to age 10 to 19 years and for age 10 to 19 years compared to age 40 to 49 years (the latter age group was chosen rather than the "age 50 and over" group because it included a much larger number of subjects, almost equally divided between males and females).

As shown in Table 4, when means were compared, using the t-test, critical ratios were sufficiently large to establish significance at the 0.01 level for each of the tested relations.

DISCUSSION

On the basis of what is known about age changes in general, it should not come as a surprise that the eye appears to become shorter with age. A review of the literature indicates that a number of investigators have found evidence of a reduction of axial length with age, but (to this writer's knowledge) none have made the suggestion that a reduction in axial length serves as a mechanism for emmetropization. Leighton and Tomlinson[19] used ultrasonography to measure the axial lengths of the eyes of 36 "young" subjects (19 through 51 years of age) and 36 "old" subjects (44 through 92 years of age), and plotted a regression line showing mean axial lengths of 23.56 mm at the age of 20 and 22.97 mm at the age of 70. They suggested that

TABLE 2. Mean values for refractive components for subjects whose ocular refraction is between plano and +2.00 D, ages 4 to 19 years.[a]

Age Group (yr)	Corneal Power (D)	Anterior Chamber Depth (mm)	Lens Power (D)	Power of Eye (D)	Axial Length (mm)
4–9	43.3	3.4	21.4	60.7	23.4
10–19	42.8	3.6	19.8	58.8	24.1

[a] Data of Sorsby et al.[3] and Sorsby et al.[18]

TABLE 3. Mean values for refractive components for subjects whose ocular refraction is between plano and +2.00 D, ages 20 years and older.[a]

Age Group (yr)	Corneal Power (D)	Anterior Chamber Depth (mm)	Lens Power (D)	Power of Eye (D)	Axial Length (mm)
20–29	42.8	3.5	19.9	58.9	24.1
30–39	43.6	3.2	20.2	59.8	23.8
40–49	43.4	3.2	20.9	60.8	23.6
50+	43.3	3.2	20.9	60.8	23.5

[a] Data of Sorsby et al.[3] and Sorsby et al.[18]

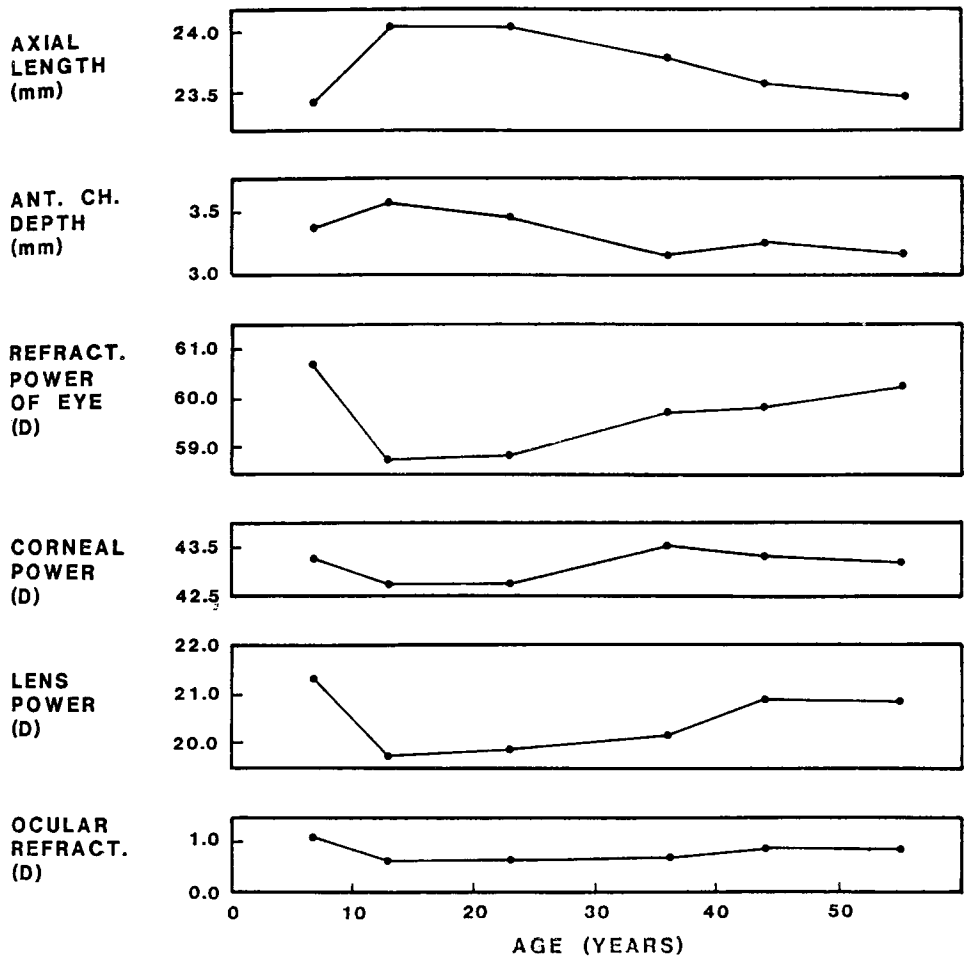

FIG. 1. Mean values of ocular refraction and its components, by age groups (data from Sorsby et al.[3] and Sorsby et al.[18]).

TABLE 4. Statistically significant relations

Component	Ages Compared (yrs)	Significance
Mean axial length	4 to 9 and 10 to 19	0.01
	10 to 19 and 40 to 49	0.01
Mean power of eye	4 to 9 and 10 to 19	0.01
	10 to 19 and 40 to 49	0.01

a decrease in the size of the corneo-scleral envelope may occur due to a reduction in the length of the constituent collagen fibers. Francois and Goes[20] reviewed ultrasound axial length data for emmetropic eyes of young and old subjects, which had been reported in two separate investigations, showing the mean axial length for older subjects to be 0.4 and 0.9 mm shorter than that for younger subjects. Axial length data on the basis of age, as reported by various investigators, are summarized in Table 5.

TABLE 5. Comparison of age differences in axial length for various investigators.

Investigators	Age (yr)	Axial Length (mm)	Difference (mm)
Leighton and Tomlinson[18]	20	23.56	0.59
	70	22.97	
Gernet[a]	20 to 25	23.6	0.9
	25 to 68	22.7	
Francois and Goes[19]	>50	23.87	0.4
	>50	23.47	
Present study	20 to 29	24.1	0.6
	50+	23.5	

[a] Reported by Francois and Goes.[20]

For the present, the concept that emmetropization of the adult eye occurs as a result of a decrease in the length of the eye must be considered only as a hypothesis. Before this hypothesis

can be confirmed, solutions must be found to the following problems:

1. As already noted, the Sorsby et al. studies were cross-sectional studies, and even though statistically significant relations were found, it cannot be assumed that these relations necessarily occur longitudinally. In this regard it has been suggested by Borish[21] that at the time the Sorsby studies were conducted, the older subjects may have been smaller in stature than the younger subjects and therefore might have been expected to have smaller eyes.

2. The data concerning axial length, as already noted, were obtained by a process of calculation, making use of corneal radii, lens radii, anterior chamber depth and lens thickness data obtained by keratometry, Purkinje image photography, and slitlamp photography. These calculations made use of "standard" indices of refraction of the ocular media, which were assumed to remain constant with age. The validity of this assumption should be a subject of investigation.

On the other hand, the hypothesis that a reduction in axial length serves as an emmetropizing mechanism is supported by the following findings:

1. The results reported by Leighton and Tomlinson and by Francois and Goes were all obtained by ultrasonic measurement, which, although being also dependent on assumed indices of refraction, has the advantage that errors due to possible index changes in the lens and vitreous would tend to cancel out each other. As noted by Leighton and Tomlinson,[19] an increase in the density of the lens would increase the ultrasound speed, under-estimating the lens thickness and also the axial length, whereas liquification of the vitreous would decrease the ultrasound speed, over-estimating the vitreous depth and also the axial length of the eye.

2. Because there is a tendency for some eyes that are emmetropic in youth or the early adult years to become hyperopic with increasing age, the decrease in axial length for a given eye would be likely to be greater than the decrease found by the use of cross-sectional data of eyes having about the same refraction for all age groups. This applies both to the Sorsby data and, as commented by Leighton and Tomlinson, to ultrasound data.

CONCLUSION

It is hypothesized that the adult eye decreases in axial length with increasing age, and that this decrease in axial length acts as an emmetropizing mechanism in harmony with an increase in the refracting power of the eye, the latter being the result of an increase in corneal and lens refracting power and a decrease in anterior chamber depth.

The crucial test of this hypothesis would be to conduct a longitudinal study of refraction and its components (including ultrasound measurements of the axial distances within the eye) on a group of subjects throughout the entire span of the adult years. Inasmuch as the Sorsby et al. data shown in Table 3 and Fig. 1 indicate that the greatest apparent reduction in mean axial length occurs between the 3rd and 6th decade, a study undertaken with subjects in their early 20s and continuing until their early 50s should prove to be adequate. For the researcher who embarks upon this task, the encouragement given by Donders[22] to von Jaeger, who had announced his intention of the following the refraction of the same persons throughout their whole lives, is appropriate: "we wish him for that purpose a long life and faithful patients."

Until a longitudinal study is done, the most appropriate test of the hypothesis will be to obtain ultrasound measurements of the axial lengths of the eyes of a very large number of adults of various ages. Because A-scan ocular ultrasound equipment is currently possessed by a number of optometry schools, a multicenter study would seem to be an efficient method of collecting the necessary amount of data.

As a final comment, it is too early to foresee what clinical implications may arise from the hypothesis that a reduction in axial length serves as an emmetropization mechanism for the adult eye. But, clinical implications or not, if and when the hypothesis is confirmed it will add an important dimension to our understanding of the refraction of the human eye.

ACKNOWLEDGMENT

The analysis of the Sorsby et al. data, upon which this paper was based, was first presented at the 3rd International Symposium on Presbyopia, March 16 to 23, 1985, sponsored by Essilor International, and published in Volume 2, pp. 85–94, of the Work Reports on the Symposium under the title *A Biometric Study of the Presbyopic Eye*.

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