The development and maintenance of emmetropia

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REVIEW

Abstract

The human eye is programmed to achieve emmetropia in youth and to maintain emmetropia with advancing years. This is despite the changes in all eye dimensions during the period of growth and the continuing growth of the lens throughout life. The process of emmetropisation in the child's eye is indicated by a shift from the Gaussian distribution of refractive errors around a hypermetropic mean value at birth to the non-Gaussian leptokurtosis around an emmetropic mean value in the adult. Emmetropisation is the result of both passive and active processes. The passive process is that of proportional enlargement of the eye in the child. The proportional enlargement of the eye reduces the power of the dioptric system in proportion to the increasing axial length. The power of the cornea is reduced by lengthening of the radius of curvature. The power of the lens is reduced by lengthening radii of curvature and the effectivity of the lens is reduced by deepening of the anterior chamber. Ametropia results when these changes are not proportional. The active mechanism involves the feedback of image focus information from the retina and consequent adjustment of the axial length. Defective image formation interferes with this feedback and ametropia then results. Heredity determines the tendency to certain globe proportions and environment plays a part in influencing the action of active emmetropisation. The maintenance of emmetropia in the adult in spite of continuing lens growth with increasing lens thickness and increasing lens curvature, which is known as the lens paradox, is due to the refractive index changes balancing the effect of the increased curvature. These changes may be due to the differences between nucleus and cortex or to gradient changes within the cortex.

Key words Emmetropia, Emmetropisation, Eye growth, Lens growth, Lens paradox, Refractive components

The human eye is genetically programmed to achieve emmetropia in youth and to maintain emmetropia with advancing years. This is despite the changes in all eye dimensions during the period of growth and the continuing growth of the lens of the eye throughout life.

Studies have been made of the ocular components of refraction and how these change with age and with refractive state. Developmental biologists have identified and studied the physiological mechanism of emmetropisation, by which the mainly ametropic neonatal eye is directed to develop emmetropia. Sufficient information is now to hand to give an overall view of the process of achieving and maintaining emmetropia.

Refraction and its components

Steiger,[1] whose monograph was published in 1913, was the first to show that the emmetropic eye might be constructed with a combination of various corneal curvatures and axial lengths. He also found that the distribution of refractive power of the cornea in the population studied followed the usual Gaussian curve with a range extending from 39 to 48 D. From this Steiger calculated the range of axial lengths that would be needed to produce emmetropia as extending from 21.5 to 25.5 mm. Steiger's calculations necessarily ignored the lens power, for which he lacked measurements.

Steiger considered that refractive errors were variants arising from the free association of the different components of refraction, from which it would be expected that the distribution of refractive errors would follow the normal Gaussian curve.

The Gaussian distribution of axial lengths that Steiger had postulated was confirmed by the measurements made by Tron[2] and Stenström.[3] Tron, Stenström and Sorsby[4–6] were later to present data from the measurement of the three main components of refraction – corneal power, lens power and axial length – that confirm the Gaussian distribution of the measurements of these components.

Since the work of Sorsby[4–6] it has been accepted that the distribution of refractive errors in the adult Western population does not follow the usual Gaussian curve found for other human parameters such as height. Sorsby showed that the graph for the distribution of

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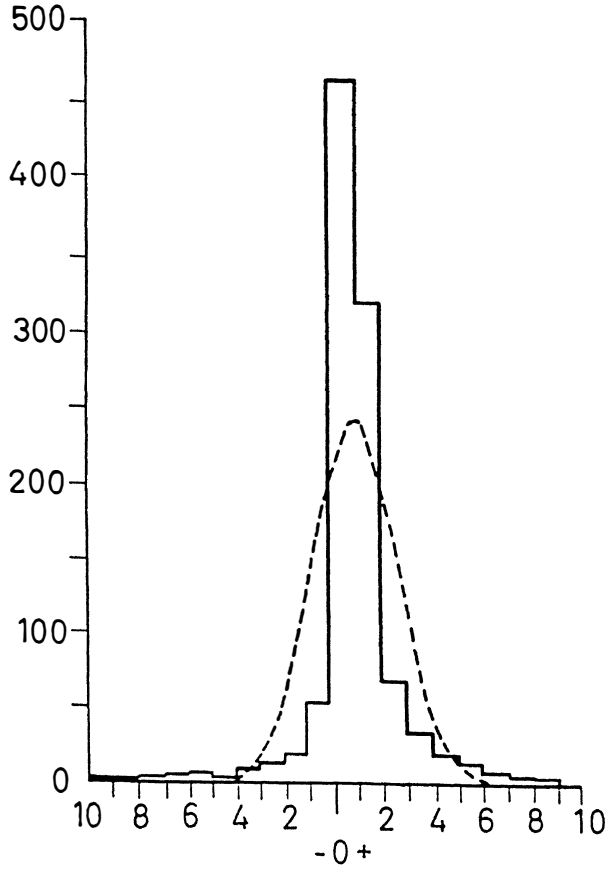

Fig. 1. *The distribution of refractive errors. x-axis, refraction in dioptres; y-axis, numbers of individuals. The dashed line represents a normal Gaussian distribution. After Sorsby.*[4]

refractive errors has a leptokurtic distribution, having an excess of refractions near emmetropia (Fig. 1), although he also confirmed that the distribution of other parameters, namely the power of the cornea, the depth of the anterior chamber, the power of the lens and the axial length of the eye, followed the common Gaussian distribution. Emmetropia was shown to be due to a variety of combinations of corneal power, lens power and axial length. Sorsby confirmed the previously suggested relationship between globe size and the power of the cornea and of the lens in emmetropia.

A problem with many of the early studies is in the methodology by which the results were obtained, as critically evaluated by van Alphen[7] in his monumental analysis of emmetropia and ametropia in 1961. While the data for corneal refractive power and axial length have stood the test of time, anterior chamber depth, lens thickness and lens curvature have been shown to be age-dependent.

The knowledge of ocular biometry in 1976 was reviewed in the monograph by Delmarcelle *et al.*[8] It had then been established by ultrasound measurement that the lens grew steadily in width through life and that this was associated with the reduction in depth of the anterior chamber.

The inverse relationship between the power of the cornea and the axial length was confirmed in emmetropia by Francois and Goes[9] and by Seiler *et al.*[10] The relationship between the power of the cornea and the axial length gives rise to a concept that has been called the 'inflatable globe' as discussed by Koretz *et al.*[11] As the globe becomes bigger, so the cornea becomes proportionately flatter. This would offer some automatic compensation for globe length in the healthy eye and ametropia is then explained by a non-proportionality of the globe. These concepts can be upheld today, but do not explain the leptokurtic distribution of refractive errors around emmetropia.

The excess of refractions near emmetropia (Fig. 1) was considered to be possible by Sorsby because the multifactorial nature of the inheritance of the eye parameters could allow a number of combinations of these parameters to produce emmetropia – a concept initiated by Steiger.[12] The studies of monozygotic twins by Sorsby and Leary,[13] Kimura[14] and Minkovitz *et al.*,[15] showed that there is a high correlation for refraction and for its components (corneal power, lens power and axial length) in twins, which clearly indicates a hereditary component to the refractive state. But since the time of Sorbsy, the existence of the excess refractions near emmetropia has led to the intriguing idea that the growth of one or more of the components influencing refraction may be regulated to achieve emmetropia. The hereditary basis for refraction may thus act via the control of the emmetropisation process, which has been suggested by Norton and Siegwart.[16]

Recent work by Koretz *et al.*[11] (Table 1) supports the concepts of Sorsby and has given us a detailed insight into the relationship between the various eye parameters that may be assembled together to achieve emmetropia. Koretz *et al.* found that the globe length correlates with corneal refractive power in emmetropes, but only weakly in ametropes. The anterior segment length correlates with the corneal refractive power in emmetropes, but not in ametropes. The anterior segment length correlates with globe length in emmetropes, but again not in ametropes. These findings, as Koretz *et al.* have commented, confirm Sorsby's concept of the ametropic eye as one that has failed to achieve proportionality. Conversely, the vitreous cavity length corresponds with the globe length in ametropes, but only weakly in emmetropes. This too is explained on the basis of lack of

Table 1. *Eye correlates in emmetropia and ametropia*

	Emmetropia	Ametropia
Refraction/globe length	Correlated	Correlated
Refraction/anterior segment length	Correlated	Not correlated
Refraction/corneal power	Not correlated	Not correlated
Globe length/corneal power	Strongly correlated	Weakly correlated
Globe length/vitreous cavity length	Weakly correlated	Correlated
Anterior segment length/corneal power	Not correlated	Correlated
Anterior segment length/globe power	Correlated	Not correlated
Anterior segment length/vitreous cavity length	Not correlated	Correlated

After Koretz *et al.*[11]

proportionality of the globe in the ametrope. This lack of proportionality is seen in the myope as a disproportionately long vitreous cavity.[17]

The power of the lens may have some relationship to the axial length of the eye. The lens power is less in the longer eye, and it had long been considered that the power of the lens might be important in emmetropisation.[18] Sorsby[5] demonstrated a relationship between lens power and globe length, which is confirmed by Garner *et al.*[19] McBrien and Millodot[20] in their study of late-onset myopia showed that late-onset myopes had deeper anterior chambers and thinner lenses than the controls. Zadnik *et al.*[21] in their study of children between the ages of 6 and 14 years showed that the lenses were thinner in myopic children. However, other ultrasound studies have not shown differences in lens width between myopes and emmetropes.[22] Francois and Goes[9] showed a lack of correlation between lens thickness and axial length in emmetropes. These findings do not negate the work of Sorsby. His estimate of lens power was based on observation of the Purkinje images. The power of the lens is not directly related to the lens thickness, since lesions of the same thickness can differ in radius of curvature. It therefore remains acceptable that the longer eye tends to carry less lens power. The longer eye also has the deeper anterior chamber,[11] which reduces the lens effectivity.

Growth of the eye in the child

Sorsby *et al.*,[23] Larsen[24] and Fledelius and Christensen[25] have shown that the eye grows rapidly in early childhood and then slowly. Sorsby estimated the axial length of the eye at birth to be 18 mm, and 23 mm by the age of 3 years. This change of 5 mm would produce a myopic shift of 15 D, requiring compensation by this amount in the dioptric strength of the eye to maintain emmetropia. After the age of 3 years the rate of growth in axial length is slowed. Sorsby estimated that between the ages of 3 and 14 years the rate of growth of the globe is 0.1 mm per year in the sagittal plane and the growth in the normal eye is then complete. Fledelius[26] and Zadnik[27] have confirmed continuing growth of the globe into adolescence. Fledelius showed continuing growth up to the age of 18 years, with an increase of 0.4 mm between 10 and 18 years (0.05 mm per year). Zadnik showed a gradual slowing of growth during the 6–14 year age period. It would seem likely that any physiological control that the body may exert in driving the eye to emmetropia is likely to be most active during the early years in which dimensions are changing most rapidly. Emmetropisation then occurs particularly in the very young, and it has recently been confirmed that there is rapid emmetropisation from 9 months of age and during the following year.[28]

The refractive status of the infant has been shown to centre around hypermetropia, estimated by cycloplegic refraction to have a mean value of 2–2.4 D at birth,[29–31] and the distribution of refractive errors around the newborn mean has a normal Gaussian distribution.[30,31] However, in more recent studies in which non-cycloplegic near retinoscopy has been used, the results show less hypermetropia. The study by Gwiazda *et al.*[31] indicates approximate emmetropia at birth, reaching a hypermetropic maximum of about +0.75 D at the age of 5 years and thereafter declining to emmetropia by the age of 12 years. Cycloplegic studies have shown that hypermetropia declines steadily to a mean of about 1 D by the age of 14 years,[23,27,29] after which the change is slow. The nearest approach to emmetropia is reached in the late twenties.[29] The difference between the cycloplegic and non-cycloplegic studies is probably that the non-cycloplegic studies include an accommodative component. The decline in hypermetropia would appear to be brought about by the increasing axial length of the eye. The increase in axial length alone would produce an excessive shift towards myopia, but this is offset by the changes that the lens is undergoing at the same time, which reduce its power.

Growth of the lens in the child

The few studies that have addressed the growth of the lens in childhood (up to 20 years) show, by ultrasound[32,33] and by slit-imaging[34] (Fig. 2), that the width of the lens does not increase during this period. The study of Zadnik *et al.*[21] has shown that the width is actually thinning in childhood. Their period of observation was from 6 to 14 years of age, during which the lens was shown to thin between the ages of 6 and 10 years and then to remain unchanged to 14 years. The surprising finding of lack of growth in width or of actual thinning in the lens of the child is explicable by the processes of equatorial expansion and central compaction within the lens (see below). During the period of rapid globe growth the increase in equatorial diameter of the globe is associated with an increase in equatorial diameter of the lens.[35] This can be considered to generate a force which stretches the lens in the

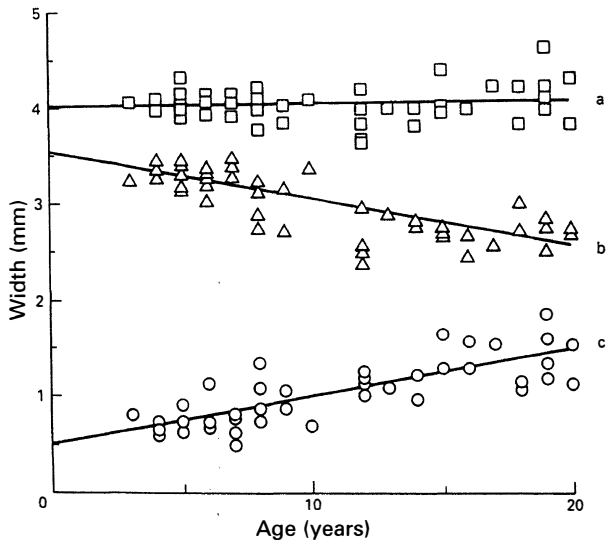

Fig. 2. *Growth of the lens in width up to the age of 20 years: (a) total lens width, (b) nuclear width, (c) cortical width (anterior and posterior combined).*[3,4]

equatorial plane. The increase in diameter of the globe in the region of the ciliary body is considered to increase the diameter of the ciliary ring, which in turn tensions the zonule and flattens the lens. This process is likely to be effectively complete by the age of 6 years in the emmetropic eye. The lens in the young child is evidently changing shape from a more spherical contour at birth to a more flattened shape, and does not actually cease to grow since a steady rate of growth is confirmed by lens weight measurements. Harding and Crabbe[36] showed a rapid rate of increase in weight of the lens up to the age of 2 years, followed by a slower and steady rate of growth from 2 to 20 years.

Central compaction was conceived to be a component in the process of lens growth because of the apparently fast rate at which superficial lens opacities sink into the lens with time (about twice the rate of lens growth[37]) and by the reduction in size of congenital cataracts with time.[38] This process appears to be most active in youth. In the young child[34] there is rapid growth of the cortex during a period when the lens is not increasing in width, or is actually thinning.[21] This is made possible by compression of the nucleus.

The dioptric power of the lens decreases relatively rapidly in childhood from about 23 D at the age of 3 years to 20 D at the age of 14 years.[23] During this same period the corneal power declines only minimally from about 43 D to 42.7 D.[23] Thus it appears that the reduction in the power of the lens in early childhood is the main factor that permits the eye to achieve emmetropia, in spite of the rapidly increasing axial length. The reduced lens power is accounted for by flattening of the lens and to a lesser extent by deepening of the anterior chamber, although refractive index changes in the lens may play a part, as indeed they do in adult life.

It is seen in children developing myopia[21] and in young adults developing myopia[20] that the lens is thinner in these persons than in emmetropes. This can be regarded as confirming the concept that the enlargement of the globe is a factor responsible for the thinning of the lens in the growing eye of the young child. This consideration does not exclude central compaction as a factor, which would occur independently of globe enlargement.

Emmetropisation in the growing eye

The Gaussian distribution of newborn refractive errors[30,31] and the non-Gaussian distribution of adult refractive errors[4–6] (Fig. 1) strongly suggests the possibility of an active mechanism of emmetropisation in the human eye involving a feedback to adjust one or more of the refractive components in response to the state of the retinal image. The concept lends itself to mechanical modelling, which gives theoretical support to the possibility of such a mechanism involving a possible simple first-order feedback.[39] Evidence exists for an active mechanism of emmetropisation in animals including tree shrews,[40] chickens[41,42] and non-human primates.[43–45] The evidence for an active emmetropisation mechanism in man has been reviewed by Troilo[46] and its existence in Western populations seems beyond dispute, although it can not be supported by controlled experiments, such as are possible in animals. Neither can the mechanism of emmetropisation yet be defined, although humoral transmitters are implicated in animals.[47,48]

Early theories of emmetropisation in man had considered that an anterior segment change was responsible. Gernet and Olbrich[49] had considered that the power of the lens of the eye might be the component of refraction that was adjusted in the process of emmetropisation, which seemed reasonable in view of the large change which takes place in lens power in infancy. In addition to lens changes, Mark[50] theorised a more general change in the anterior segment with reduction in power of the cornea and deepening of the anterior chamber as additional to the change in power of the lens. van Alphen[51] has considered ciliary muscle tone and choroidal tension to be significant in globe elongation.

The axial length in several animal species can be affected by intervention of various kinds. This has been reviewed by Norton and Siegwart.[16] These interventions include the deprivation of form vision and the shifting of the focal plane by positive or by negative lenses.[52] The results show that a shift in focal plane is followed by an appropriate shift in axial length to meet the focal plane. The relationship is then maintained. It is then conceivable that human axial length also may be affected by shifting the plane of focus. The effect of near work would be expected to produce an increased axial length appropriate to the conjugates involved, and this has been implicated as a cause of myopia in Inuit children by Johnson *et al*.[53] Giving plus lenses to young human hypermetropes should interfere with the process of emmetropisation and this has been found to be the case.[54]

Near work in man might be expected to produce 'emmetropisation' for near objects and so induce myopia. Several studies confirm the relationship between near work and myopia.[27,55–60] Axial length studies show that the increase in myopia in these eyes is due to increased axial length.[61] Plus lenses given to myopes doing close work might be expected to reduce the rate of progression, but bifocal glasses given to young adults with myopia did not affect the rate of progression.[62] A positive relationship has been found between esophoria and increasing myopia in young adults,[63] and bifocal glasses given to myopic subjects with esophoria did reduce the rate of progression in these subjects. This suggests a possible relationship between myopic progression and the accommodation/convergence synkinesis.

When the young eye is occluded or otherwise deprived of a formed image, axial elongation continues uncontrolled. In animals, visual deprivation generally causes the development of myopia associated with increasing length of the vitreous cavity. This change was first shown in monkeys by Wiesel and Raviola[64] and

confirmed by von Noorden and Crawford.[65] Chicks also respond to deprivation with globe elongation.[41] In human children the interference with image formation causes ametropia. The result is usually myopia, but hypermetropia also occurs in certain conditions. Children who have been affected by the closure of the lid of one eye tend to become myopic in that eye[66–68] and increasing axial length is reported in the human with corneal opacity,[69] in association with infantile traumatic cataract[70] and in optic nerve hypoplasia.[71] These observations suggest that it is the length of the eye and probably specifically that of the vitreous cavity that may be influenced by image formation in the vertebrate eye including the human eye. The failure of emmetropisation with hypermetropia in some children with defective vision was considered by Nathan *et al.*[72] to occur when foveal vision was primarily affected and that myopia occurred with peripheral, or peripheral plus central impairment. Stark[73] also reported cases of hypermetropia.

In the chicken the myopia induced by visual deprivation is reversible. Visual deprivation induces myopia by elongation of the vitreous cavity and this is followed by recovery when the visual restriction is removed in the first 6 weeks of life. The recovery occurs by cessation of vitreous cavity elongation and the rate of recovery is proportional to the degree of induced myopia.[42] In normal human infants the rate of progress from hypermetropia towards emmetropia is also dependent on the initial degree of hypermetropia.[74] Infant myopes are seen to progress towards a normal hypermetropia by the age of 3 years.[75]

It appears most likely that emmetropisation is determined by the response of the eye to the retinal image and that the eye is able to react appropriately to correct the axial length for either a hypermetropic or a myopic refractive error. This mechanism in vertebrates appears to rest largely within the eye itself since emmetropisation still occurs in the presence of optic nerve section in the chick eye.[52] However, in the chick the emmetropisation overshoots in the presence of optic nerve section, which may be due to loss of the retinal ganglion cells or to loss of some central control. In the human infant it has been noted that optic nerve hypoplasia is associated with myopia,[71] so that in the human it seems likely that an intact optic nerve may be of more importance than in the chick, as noted above. Within the eye humoral transmitters are implicated in animals.[47,48]

It appears likely that the process of emmetropisation in man is most active during the very early years of life, since the non-Gaussian leptokurtic distribution of refractive errors, as observed by Sorsby in the adult, is established in 6- to 8-year-olds.[31,76] The influence of near work on the eye has been shown to affect young persons older than this, which implies that the parameters of the eye remain malleable after the age of 8 years.

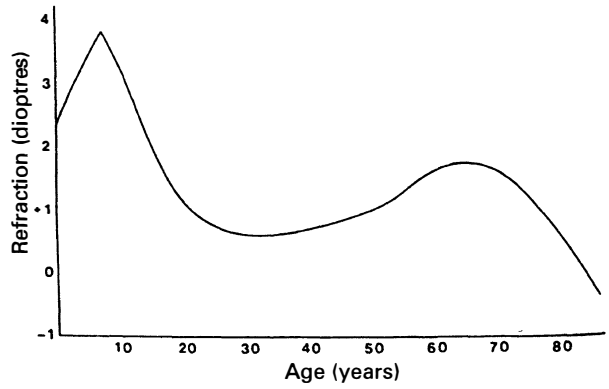

Fig. 3. *Change in cycloplegic refraction of the eye with age. After Slataper.*[29]

The maintenance of emmetropia in adult life

Slataper's study,[29] which was based on clinical material, showed that the hypermetropia of the child declines to near emmetropia in the later twenties (Fig. 3). Thereafter Slataper's figures show a gradually increasing hypermetropia, which becomes more rapid after the age of 40 years and peaks at about 65 years, after which, on average, there is a shift towards myopia with the recovery of emmetropia at about the age of 80 years. During adult life the corneal curvature and the axial length remain constant,[77,78] whilst major changes take place in the lens.

Growth of the adult lens: width

During adult life the lens is growing steadily in width, as confirmed both by ultrasound[79,80] and by slit-image photography.[37,80] The rate of growth is approximately 0.02 mm per year and has been estimated at rather widely varying rates by different authors.[81] There may be some slowing in the rate in old age.[82] The growth is essentially cortical with little change in nuclear width. A steady rate of growth in the adult is confirmed by lens weight measurements.[36]

The lens grows by the surface accretion of new lens fibres and at the same time central compaction is occurring. The evidence for this has been presented above. The rate of growth of the lens is thus the algebraic sum of the rates of surface accretion and of central compaction. It is probable that the rates of each of these two functions decline with age, but the sum remains constant.

Lens growth in the adult is associated with a steady rate of shallowing of the anterior chamber, but not with a reduction in the length of the posterior segment.[77] This is probably made possible by the anterior zonular shift with age[83] and by a redistribution of the zonular fibres in relation to the lens equator. The differential growth between the posterior and anterior parts of the capsule appears to be an integral part of this asymmetrical lens growth (see below). The forward movement of the lens anterior surface increases its effectivity, but this appears to be a minor factor influencing the lens power compared with the change in curvature.

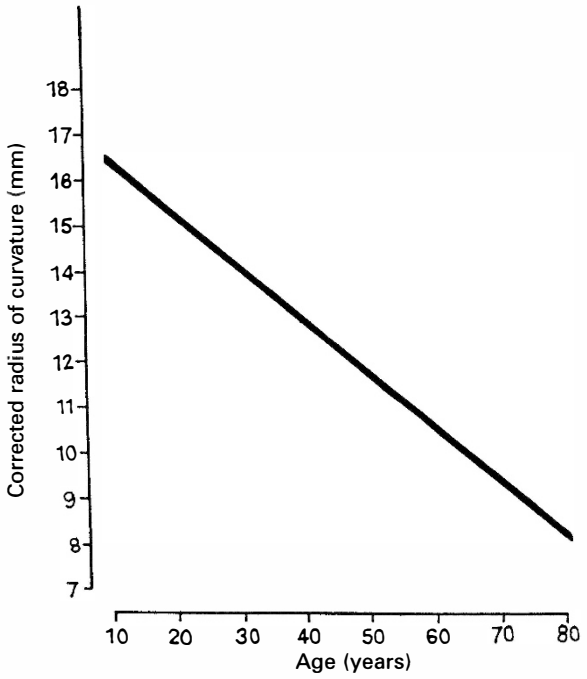

Fig. 4. *Change in curvature of the anterior surface of the lens with age. After Brown.*[89]

Growth of the adult lens: equatorial diameter

In the young child the lens grows rapidly in equatorial diameter[35] during the period of rapid growth of the globe, but in the adult the lens grows more in width than in equatorial diameter.

Early studies of lens growth[84,85] showed an increasing equatorial diameter in adult life, but more recent studies have shown there to be no increase. Willekens *et al.*[86] showed no change after the age of 30 years and Pierscionek[87] found no change between 16 and 84 years.

Change in lens curvature with age

The surfaces of the lens have been shown to increase in curvature with age[88,89] (Fig. 4). These observations were made at a time when the relatively static nature of the equatorial diameter was not known, and it can now be seen that the observations of increasing width and increasing curvature are compatible with a static equatorial diameter.

The increase in curvature would be expected to produce an increase in effective lens power and thus a shift in vision towards myopia. In fact, the opposite occurs and the healthy ageing eye progresses in the direction of hypermetropia.[90] Slataper's[29] refractive figures included subjects developing cataract and it

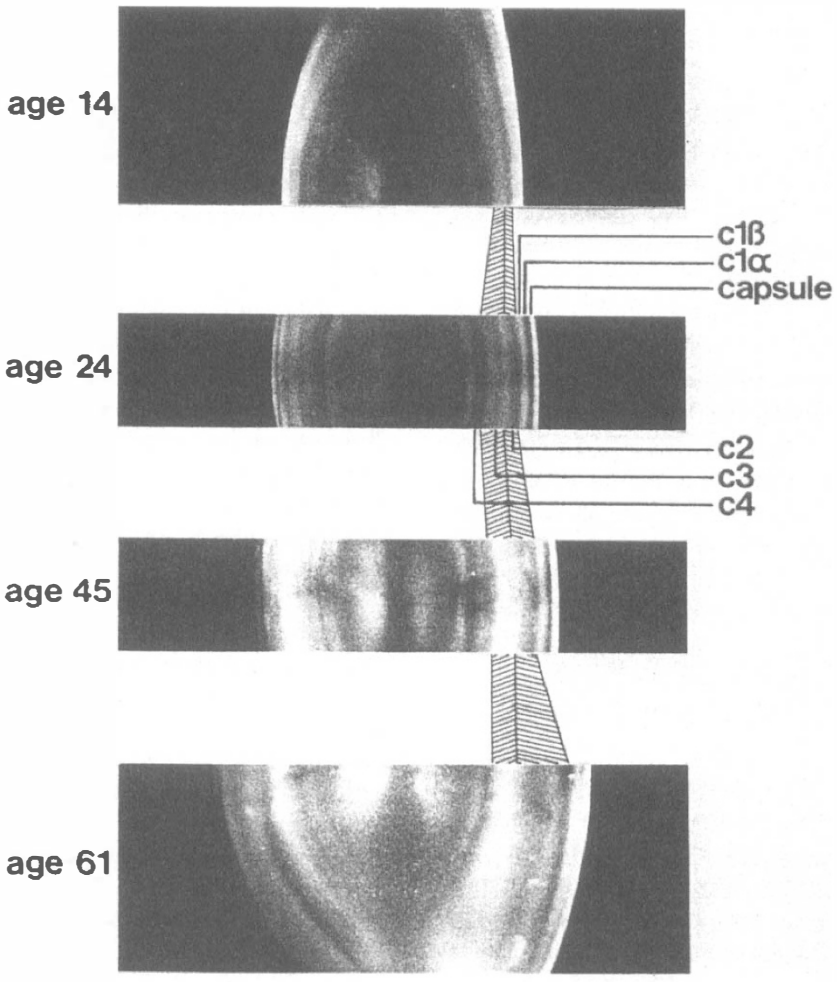

Fig. 5. *The development of the zones of discontinuity of the lens with age. From Smith et al.*[95]

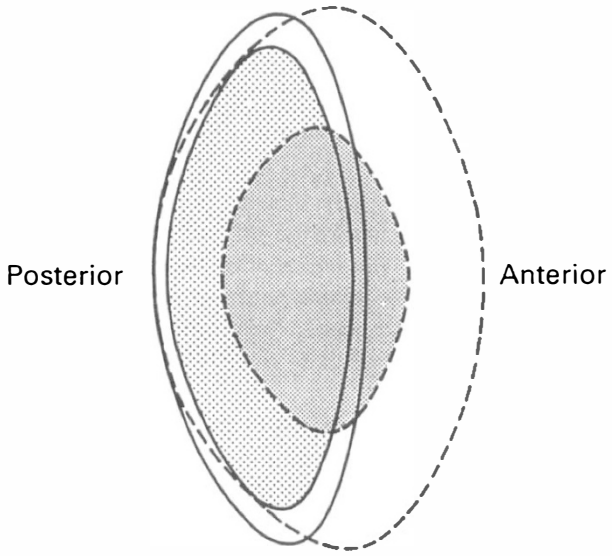

Fig. 6. *Diagrammatic cross-section of the lens in childhood (8 years) and in old age (80 years). Outline: 8 years, continuous line; 80 years, dashed line. Nucleus: 8 years, coarsely stippled; 80 years, finely stippled.*

seems likely that the overall shift in the aged population towards myopia, as reported by Slataper, is accounted for by a proportion of individuals developing nuclear cataract. In the healthy ageing eye the hypermetropic shift is continued.

Capsular remodelling

The lens is invested by a thick lens capsule which is the basal lamina of the lens epithelium. In the rat, in fetal life, the posterior capsule initially grows more vigorously than the anterior.[91] Postnatally, there is negligible growth of the posterior capsule and continued increase in capsular thickness only occurs anteriorly in relation to the lens epithelium.[6,92] A similar process is thought to occur in the human lens since there is little evidence of thickening of the posterior capsule postnatally. With the continued increase in volume of the lens with age, there is not only an increase in thickness of the anterior capsule, but an increase in surface area of the capsule as a whole. This must imply considerable remodelling of the capsule and includes an increase in surface area of the lens epithelium and either some synthetic capacity for the post-equatorial segments of the subcapsular cortical fibres or, as seems more likely, a spinning off of the posterior capsule by the equatorial epithelial cells. Such a remodelling process must have implications for the insertion of the suspensory ligament of the lens. It is known, for instance, that with age the boundaries of the zonular attachments become more diffuse and the anterior and posterior attachments diverge, while equatorial attachment is unchanged.[83]

Internal change in the lens

The internal architecture of the lens is clearly changing as the lens ages. The lens zones of discontinuity develop from childhood to reach the fully developed pattern in middle age.[93,94] The scattering properties of the brighter zones of discontinuity increase with age, and in particular that of the deeper cortical zone designated C3, which is absent at birth and during early childhood. C3 appears as a distinct layer during the second decade and shows the most marked increase to become the most highly scattering zone[95,96] (Fig. 5). The growth in width of the cortex with age is mainly accomplished by the expansion of C2, with C3 showing little increase in width once it has become established. The changes with age in the zone system are likely to be important to the power of the lens since it has been considered that the degree of scatter is related to the refractive index[97] and in turn to the protein concentration.[98] The basic differences between the lens in childhood and in old age are shown in Fig. 6.

Change in optical power of the lens with age

The maintenance of emmetropia, or increasing hypermetropia with age, in spite of the increase in curvature of the lens has been described by Brown as the lens paradox.[99] For the hypermetropic shift to occur, there must be changes in refractive index in the lens, or alternatively a reduction in the axial length of the eye.[100] The refractive index changes in the lens may be in the differences between the nucleus and the cortex or the change may be a gradient change confined to the cortex.

The effect of the change in the difference between the nucleus and the cortex may be explained by regarding the cortex as a concavo-convex negative lens element and the nucleus as a biconvex positive element (Fig. 7). In youth it appears that the nucleus has the higher

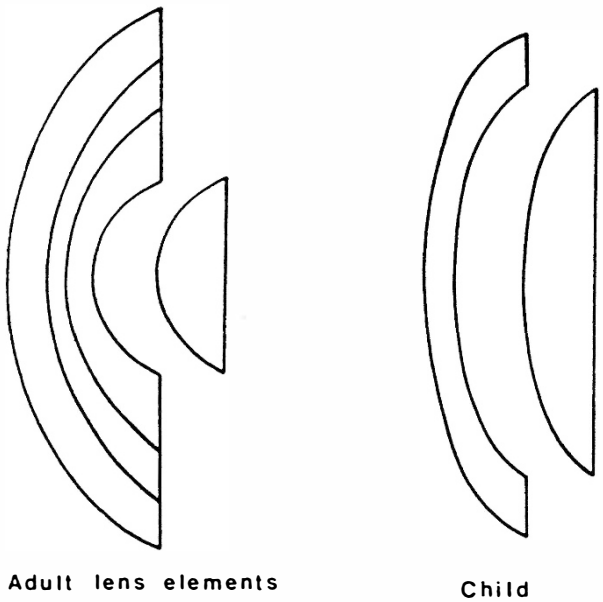

Fig. 7. *The lens cortical and nuclear shells of an old adult and of a child. The cortex may be regarded as a concavo-convex element.*

refractive index and that the situation may become reversed in the aged with the cortex assuming the higher refractive index of the two (except in age-related nuclear cataract). An increase in refractive index of the cortex with age could be explained on the basis of increased light scatter.[97] A reduction in refractive index of the ageing nucleus could be explained on the basis of a reduction in the protein concentration in the nucleus, which has been demonstrated by Raman spectroscopy.[101] Additionally the ageing of lens protein leads to the formation of an increasing proportion of insoluble protein. It has been suggested that the insoluble protein would no longer contribute to the refractive power of the cytoplasm.[80]

An alternative or additional explanation for the lens paradox concerns the change in refractive index gradient confined to the cortex. The refractive profile from the surface to the centre of the lens[87] shows a low refractive index at the surface, rising rapidly to a plateau through the lens centre. The refractive index profile is paralleled by the protein concentration profile, which has been measured by a number of techniques.[98,102,103] Pierscionek's study[98] showed that with ageing there is a rise in the surface refractive index which reduces the peripheral refractive index gradient. A reduction in gradient will reduce the refractive power of the lens surface and is thus likely to be a factor in maintaining the steady refractive power of the lens in the presence of increasing surface curvature.

A number of mathematical models can account for the lens paradox. Cook and Koretz[104] considered that the most probable model is the reduction in the refractive index of the nucleus and associated deep cortex leading to a shallower gradient between the lens centre and the surface. Ooi and Grosvenor[78] considered that it is the decrease in refractive index gradient from the surface that is responsible.

The possibility that a reduction in axial length with age is responsible for the hypermetropic shift was suggested by Grosvenor.[100] He had examined the data of Sorsby *et al*.[105] and this indicated a reduction in axial length with age, as do the data of Francois and Goes.[106] However, these sets of data were not from longitudinal studies, but compared the measurements derived from subjects of various ages living in Europe at that time. During the first half of the twentieth century there had been an improvement in nutrition in Europe, which had produced increases in other body parameters such as height. Thus the findings of a large globe size in the younger subjects in 1957 and in 1971 has to be considered in this context. Other ultrasound studies[77,78] have not shown increasing axial length with age and it is the first author's personal experience of lens implant subjects over the past 20 years that these individuals maintain better refractive stability than their phakic counterparts. With the evidence presently available, it seems that any reduction in axial length that may occur with age is at the most only minimal and that the hypermetropic shift with age is essentially a human lens-based phenomenon.

Conclusion

Emmetropisation is the result of both passive and active processes. The passive process is that of proportional enlargement of the eye in the child. The proportional enlargement of the eye reduces the power of the dioptric system in proportion to the increasing axial length. The power of the cornea is reduced by lengthening of the radius of curvature. The power of the lens is reduced by lengthening radii of curvature and the effectivity of the lens is reduced by deepening of the anterior chamber. Ametropia results when these changes are not proportional.

The active mechanism involves the feedback of image focus information from the retina and consequent adjustment of the axial length. This can be seen as a fine-tuning process that complements the passive processes. Active emmetropisation occurs in the very young child, but the eye remains malleable to environmental influences into young adult years.

Heredity determines the tendency to certain globe proportions, and environment appears to play a part in influencing the action of the active mechanism of emmetropisation.

The maintenance of emmetropia in the adult in spite of continuing lens growth and increasing lens curvature is due to the refractive index changes balancing the effect of the increased curvature. These changes may be due to the differences between nucleus and cortex or to gradient changes within the cortex. The exact mechanism is as yet obscure.

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